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ORIGINAL ARTICLE

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Frequency and Patterns of Retinal Eye Diseases in Outpatient Department of a District Hospital in Bangladesh

Md. Mahmud ul Huda*[1], Sajed Abdul Khaleque[2], Mohammad Habibullah[3], Zakia Farhana[4]

Abstract

Introduction: *Retinal diseases especially those due to diabetes and AMD are coming up as important causes of blindness and visual impairment. Our study is to evaluate the frequency and pattern of retinal diseases in ophthalmic outpatient department of a district hospital in Bangladesh.* ***Materials and Methods:*** *A total number of 173 referred new retina cases were reviewed at outpatient department of the 250 beded General Hospital, Jamalpur, Bangladesh, between February 2017 and November 2017. Data was analyzed according to age, sex and clinical diagnoses and detailed fundus evaluation done with binocular indirect ophthalmoscope and slit lamp using 20D and 90D lenses respectively.* ***Results:*** *Out of 7164 new patients,of whom 173 patients were (2.42%) presented with retinal diseases.139 patients were reviewed and followed up. Male: female ratio was 1.24:1. Out of 139 patients 77 (55.4%) were males and 62 (44.6%) females with a peak age group of 41-50 years. Diabeticrelated retinal conditions were 36 cases (25.9%), the most common cause.16 (44.44% of total DR) patients had CSME and 5(14.9%) had ADED. Diabetic retinopathy 36 cases (25.9%), Chorioretinitis 20 cases (14.4%), ARMD 11 cases (7.9%), and Optic atrophy 10 cases (7.2%).* ***Conclusion:*** *Retinal diseases remain an important cause of visual morbidity. There is increasing incidence of retinal blindness especially diabetic retinopathy in Bangladesh. The impression based on hospital practice is that the problem is increasing. This entails the necessity for accessible comprehensive eye care services, establishment of human resources, screening and awareness of the disease and affordable eye health policy.*

Keywords: *Retinal diseases, Retinal detachment, Age related macular degeneration, Diabetic retinopathy, Visual loss, Bangladesh.*

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Introduction:

Retinal diseases are the major causes of visual[1] impairment in developed countries. Retinal disease has had a low priority in prevention of blindness programmes in developing countries mainly because retinal diseases were considered an uncommon cause of blindness in the developing world[2]. In developing countries like Bangladesh, the leading causes of avoidable blindness are cataract, nutritional blindness, corneal scarring and glaucoma[3]. With various intervention programmes, the emphasis has been on the elimination of these conditions such as availability of low cost technology resulting in increasing cataract surgical rate, vitamin A distribution and food fortification for corneal scarring from[4,5] Vitamin A deficiency . All these have resulted in a gradual but definite reduction in the burden of blindness from these conditions. This has also resulted in less attention being paid to retinal diseases leading to late detection with equaling blinding and almost irreversible visual loss from preventable and treatable causes like diabetic retinopathy, retinal detachment and age related macular degeneration (ARMD). There are limited low vision and visual rehabilitation services available for cases with irreversible visual impairment. Recently there has been a significant increase in the burden of vitreo-retinal disorders globally. With increased longevity and increased uptake of cataract surgical services, retinal diseases especially those due to diabetes and AMD are coming up as important causes of blindness and visual impairment. In spite of the effort and expense involved in acquiring costly equipment and developing skilled human resource for retinal sub specialty, failure in justifying the treatment results of retinal disease has also contributed to the development and strengthening of this assumption[6]. Previous reports from hospital based studies and general population surveys of causes of low vision have implicated vitreo-retinal diseases as the major public eye health burden. Population-based surveys reported vitreo-retinal disorders to be responsible for 8.56% and 12.7% in Iran[7] and India[8] respectively. The age and sex-adjusted prevalence of vitreo-retinal diseases in Korean adults 50 years of age and older was 9.9%[9]. According to the Pakistan National Survey for blindness and visual

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impairment done in year 2002-03, posterior segment diseases accounted for 3.4% of total blindness and visual impairment. Retinal diseases vary widely ranging from common but easily treatable to rare and untreatable.The purpose of our study is to generate data on frequency and pattern of retinal disease in patients aged above 16 years presenting at 250 beded general hospital, Jamalpur.

Materials and Methods:

250 beded general hospital, Jamalpur, provides secondary level health care to Jamalpur district and its suburbs as well as rural population of far-flung areas of Brahmaputra and Jamuna river. All services are provided free of charge.

A total number of 7164 referred newcases were reviewed at outpatient department of the 250 beded General Hospital Jamalpur, Bangladesh, between February 2017 and November 2017. Out of them 173 were new retina cases were reviewed. The data was obtained from the ophthalmic outpatient department. Data was analyzed according to age, sex and clinical diagnoses and detailed fundus evaluation done with binocular indirect ophthalmoscope and slit lamp using 20 D and 90 D lenses respectively.

The study was conducted with adherence to institutional policy. Patients' privacy was maintained by excluding identification names and hospital numbers of patients from data analysis and manuscript preparation.

Results:

Out of 7164 new patients attended in ophthalmic OPD, of which 173 patient were (2.42%) patients presented with retinal diseases. Some patients were lost from follow up.139 patients were reviewed and followed up. Demographic characteristics of patients (table I) shoes the age range was 1 to 90 years with the peak age group between 41-50 years.77 (55.4%) were males while 62 (44.6%) were females. The minimum age of the patient registered in retina clinic was 8 years while the maximum age was 88 years. It appeared that the conditions are more common in 41 to 60 years age group. Disease pattern (table-II) shows out of these 139 patients 75(54%) were from rural areas while the rest of the patients 64 (46%) belonged to rural areas.76 (54.7%) patients had monocular involvement while rest of the 63 (45.3%) patients had bilateral involvement. The common retinal vascular diseases (presented in table II) were diabetic retinopathy/ maculopathy in 36(25.9%), hypertensive retinopathy, 2(1.4%) and retinal vascular occlusion in 11(7.9%) eyes respectively.

Table –I: Demographic characteristics of patients.

Age	Male	Female	Total (%)
≤ 10	1	1	2
11 – 20	4	5	9
21 – 30	7	3	10
31 – 40	12	10	22
41 – 50	20	20	40
51 – 60	14	16	30
61 – 70	13	5	18
71 – 80	5	2	7
≥ 81	1	0	1
Total	77	62	139

Age related macular degeneration and macula hole were the common macula pathologies documented in 37(15.0%) and 10(4.0%) eyes respectively while retinal detachment was the most common condition that required emergency vitreo-retinal surgical intervention in 11(4.5%) eyes. Bilateral visual impairment (low vision and blindness) from retinal diseases was present in 42(30.2%) while uniocular visual impairment was documented in 51(36.7%) persons. The causes of bilateral blindness were age related macular degeneration 8(38.1%), diabetic retinopathy 5(23.8%), chorioretinitis, mostly congenital toxoplasmosis 4(19.0%), retinal detachment 2(9.5%) and retinitis pigmentosa 2(9.5%). Chorioretinitis 20(14.4%) and age related macular degeneration 11(7.9%) were the common macula pathologies documented.

Table – II: Retinal Disease pattern.

Retinal Disease	No. of Patients n (%)	Unilteral n (%)	Bilateral n (%)
Amblyopia	5 (3.6%)	3	2
ARMD	11(7.9%)	6	5
BRVO	5(3.6%)	2	3
Chorioretinitis	20(14.4%)	13	7
Coloboma	4(2.9%)	3	1
CRVO	6(4.3%)	5	1
CSCR	4(2.9%)	3	1
Diabetic Retionpathy	36(25.9%)	13	23
Drusen	2(1.4%)	0	2
Hypertensive rettnopathy	2(1.4%)	1	1
Macular dystrophy	4(2.9%)	2	2
Macular Edema	4(2.9%)	3	1
Macular Hole	2(1.4%)	2	0
Myopic degeneration	4(2.9%)	2	2
Optic Atrophy	10(7.2%)	6	4
Optic neuritis	4(2.9%)	1	3
Retinal Detachment	1(0.7%)	9	1
RP	5(3.6%)	2	3
Vasculitis	1(0.7%)	0	1
Total	**139(100%)**	**76(54.7 %)**	**63(45.3 %)**

Discussion:

The retinal disease pattern noted at 250 beded general hospital, Jamalpur is comparable to those noted at other institutions of the developing world. Vitreo-retinal disorders constituted a significant reason for presentation to eye clinics and eye department of district hospital, ranging from 3.9% in South-Eastern Nigeria[10] to 12.5% in Ethiopia [11]. In Nigeria vitreo-retinal disorders constituted a significant cause of ocular morbidity and vision loss with reported hospital prevalence rate of 13.0%[12]. A study from Malaysia has also reported retinal diseases to be responsible for 12% of patients presenting to outpatient department of eye units[13]. In our study we found 2.42% patients had vitreo-retinal disorders. This may be due to lack of awareness and socio economic condition of this area. The male to female

ratio was 1.24:1. This is again similar to the study done in Ethiopia[11]. The higher male attendance of hospitals for healthcare in developing countries contributes to the male preponderance. However greater uptake of cataract surgical service by males may be another reason for increased number of males with retinal diseases. Since the study was aimed to find out the age, sex and diagnostic varieties in order to assess pattern of posterior segment disease in patients presenting at the 250 beded general hospital, Jamalpur, other demographic and therapeutic details were not included. It appeared that the conditions are more common in 41 to 60 years age group (70%). This is similar to the findings from Nigeria[12] and can be compared to the study done in Malaysia where majority (61.9%) patients were above the age of 50 years. Diabetic retinopathy(25.9%) was the most common cause for attendance in the OPD showing that diabetic eye disease is emerging as a challenge. This is similar to the results from Nepal eye hospital where diabetic related conditions were most common cause for visiting the retina OPD[14]. In Malaysia[13] and Nigeria[12] diabetic retinopathy accounted for 9.7% and 9.6% retinal diseases respectively. This warrants timely screening, evaluation, treatment, follow up and education for diabetic related conditions.Retinal detachment represented 1% of retinal diseases in this study as opposed to only 7% in Nepal, 12% in Malaysia[13] and Ethiopia 24.5% of diseases.

In our study, ARMD accounted for 7.9% of retinal diseases, which is similar to a hospital based study in Pakistan shows 9.3%[15].This is in contrast to the prevalence of 2.7% AMD from Ethiopia. The age adjusted prevalence of ARMD was 4.72 % in Sri Lanka[16]. In the second national blindness survey of Pakistan (2002-2004) macular degeneration accounted for 2.8%[2]. This difference may be due to the fact that the current study was a hospital based study where patients have manifest retinal conditions. It appears that inspite of proliferation of various levels of posterior segment service facilities within the country and even the city the number of attendance in OPD at 250 beded general hospital, Jamalpur, is on rise. This on one hand stresses and justifies additional investments needed to tackle all kinds of posterior segment eye problems including the ones needing complex vitreo-retinal surgical procedures while on the other recommends general community awareness in order to reduce undue blindness and visual impairment due to avoidable causes.

The results of this study gave an insight into thepattern of retinal eye diseases seen in a district hospital of Bangladesh. However in order to generalize the results it is necessary to conduct a larger multi center study or a population based study.

Conclusion:

There is a tremendous impact of increasing retinal blindness secondary to retinal diseases especially DR in Bangladesh. The impression based on hospital practice is that the problem is on rise. The set up for their evaluation and management especially surgical is expensive and for average Bangladeshi population the treatment is not affordable unless subsidized by the hospital. This entails the necessity for accessible comprehensive eye care services, establishment of human resources, screening and awareness of the disease and affordable eye health policy.

Conflict of Interest: None.

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References:

1. Kumaresan J. What's new in trachoma control? Comm Eye Health. 2004; 17: 49 - 51.

2. Yorston D and Jalali S. Retinal detachment in developing countries. Eye. 2002; 16: 353-8.

https://doi.org/10.1038/sj.eye.6700188

PMid:12101440

3. World Health Organization. World Health Assembly Resolution WHA 51.11 Geneva: WHO;1998.

4. Yorston D. Retinal diseases and vision 2020. J Comm Eye Health. 2003; 46: 19-20.

5. Lewallen S, Courtright P.Blindness in Africa:present situation and future needs. Br J Ophthalmlol. 2001; 85: 897-903.

https://doi.org/10.1136/bjo.85.8.897

PMid:11466240 PMCid:PMC1724094

6. Yorston D. Retinal Diseases and VISION 2020.Community Eye Health.2003; 16: 19-20.

7. Hatef E, Fotouhi A, Hashemi H. Prevalence of retinal diseases and their pattern in Tehran: the Tehran eye study. Retina.2008; 28: 755-62.

https://doi.org/10.1097/IAE.0b013e3181613463

PMid:18463522

8. Dandona L, Dandona R, Srinivas M. Blindness in the India State of Andrapradesh. Invest Ophthalmol Vis Sci. 2001; 42: 908-16.

9. Youm DJ, Oh HS, Yu HG. The Prevalence of Vitreoretinal Diseases in a Screened Korean Population 50 Years and Older. J Korean Ophthalmol Soc. 2009; 50: 1645-51.

https://doi.org/10.3341/jkos.2009.50.11.1645

10. Eze BI, Uche JN, Shiweobi JO. The burden and spectrum of vitreo-retinal diseases among ophthalmic outpatients in a resource-deficient tertiary eye care setting in South-Eastern Nigeria. Middle East Afr J Ophthalmol. 2010; 17: 246-9.

https://doi.org/10.4103/0974-9233.65491

PMid:20844680 PMCid:PMC2934716

11.Teshome T, Melaku S, Bayu S. Pattern of retinal diseases at a teaching eye department, Addis Ababa, Ethiopia. Ethiop Med J. 2004; 42: 185-93.

12. Onakpoya OH, Olateju SO, Ajayi IA. Retinal Diseases in a Tertiary Hospital: The Need for Establishment of a VitreoRetinal Care Unit. Journal of the National Medical Association. 2008; 100: 1286-9.

https://doi.org/10.1016/S0027-9684(15)31506-6

13. Reddy SC, Tajunisah I, Low KP. Prevalence of eye diseases and visual impairment in urban population. A study from University of Malaya Medical Centre. Malaysian Family Physician.2008; 3: 25-8.

14. Karki DB, Malla OK, Byanju RN. Analysis of 400 cases of posterior segment diseases visiting retina clinic of Nepal eye hospital. Kathmandu University Medical Journal. 2003; 1: 1615.

15. Aimal Khan, Qamar Riaz, Fayaz Soomro, Umair Qidwai, UmerQazi. Frequency and Patterns of Eye Diseases in Retina Clinic of a Tertiary Care Hospital in Karachi. Pak J Ophthalmol. 2011; 27 : 155-9.

16. Goold LA, Edussuriya K, Selva D. Prevalence and determinats of age-related macular degeneration in central Sri Lanka: The Kandy Eye Stury. Br J Ophthalmol. 2010; 94: 150-3.

https://doi.org/10.1136/bjo.2009.163808

PMid:19713196

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ORIGINAL ARTICLE OPEN ACCSESS

Clinical Evaluation of Corticosteroid on Post Operative Morbidity in Impacted Lower Third Molar Surgery

Mohammad Iqbal Kabir [*1], Muhammad Mizanur Rahaman [2], Md. Atiqul Islam Rabby [3], Samira Taufique Reshma [4], Md. Abdur Rab [5], Golam Mohiuddin Chowdhury [6], Ismat Ara Haider [7]

Abstract

Introduction: *Mandibular third molar is most commonly dental impacted and it's removal is not without morbidity. Post operative pain, swelling and trismus are universal. This study was carried out to compare the effect of co-administered corticosteroid and diclofenac Na^+/K^+ with diclofenac Na^+/K^+ alone on the post operative morbidity like pain, swelling and trismus after surgical removal of impacted lower third molars.* ***Materials and Methods:*** *The study was a prospective study done over a period of 01.01.13 to 30.06.14 in Department of Oral & Maxillofacial Surgery (OMS), Military Dental Center, Combined Military Hospital, Dhaka and the department of OMS Dhaka Dental College and Hospital, Dhaka. Sixty patients were included in the study and were randomly divided into two study groups: Control group received only diclofenac Na^+/K^+, Experimental group received corticosteroid and diclofenac Na^+/K^+. Pain, swelling and trismus were evaluated before, 1st, 2nd and 7th POD after surgery.* ***Results:*** *Among the 60 cases common type of impaction was horizontal and vertical type (30%) followed by Mesioangular (23%) and Distoangular (17%). All corticosteroid groups showed statistically significant ($p < 0.01$) improvement in pain and trismus at 1st and 2nd pod, but statistically significant ($p < 0.01$) improvement of swelling occur only on 2nd pod as compared to control group.* ***Conclusion:*** *Corticosteroid and diclofenac Na^+/K^+ combination was found effective for early recovery and the patients who used this combination suffered less pain, swelling and trismus after impacted mandibular third molar surgery.*

Keywords: *Impaction, Corticosteroid, Diclofenac.*

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Introduction:

Surgical removal of third molars is one of the most frequently performed surgical procedures to treat pathose originated by impacted teeth[1]. An impacted tooth causes infection in the gum surrounding the tooth leading to pain and swelling. Sometimes cyst also forms due to impacted wisdom tooth. To overcome this problem offended tooth should be extracted. But surgical extraction of impacted third molar almost always cause some post operative sequelae like pain and swelling. The management of pain and other problems consequent to tooth extraction is always a major concern for the individual[2,3].

Pain is the most common complain of human being. Postoperative pain especially after third molar surgery is said to be one of the most acute post surgical painful condition. Proper management of post extraction pain relieves patient suffering and leads to early mobilization, shortened hospital stay, reduce hospital costs and increased patient satisfaction[5,6,7].

Preliminary observations of various types of post operative pain indicate that the biological and psychological foundation for long-term persistent pain is in place within hours of injury[8]. Even brief painful stimulus can produce lasting changes in cells within the spinal cord[9]. Tissue injury causes a cascade of events (including peripheral inflammation) that release various mediators into the local environment[10]. These mediators activate the primary afferent nerves that sensitize local nerve receptors, which, in turn, can evoke changes at the level of the spinal cord, process referred to as "peripheral sensitization"[11]. This process is responsible for the development of hyperalgesia beyond the damaged site. If acute pain is not properly treated, prolonged activation of the pain pathways can lead to further neurophysiologic changes collectively called "central sensitization," which may prolong recovery and convert acute pain to a chronic condition[12].

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Proper analgesic treatment can reduce this risk[8]. The primary obligation and ultimate responsibility of oral health care providers is not only to restore function, but also to relieve pain. The NSAID diclofenac sodium claimed to be among the most successful aids in post operative pain control[13].

Many surgical procedures result in certain amount of oedema or swelling after surgery. Routine extraction of a single tooth will probably not result in that the patient can see, whereas the extraction of multiple impacted teeth with reflection of soft tissue and removal of bone may result in moderately large amounts of swelling. Swelling usually reaches its maximum 24 to 48 hours after surgical procedure. Swelling begins to subside on the third or fourth day and usually resolves by the end of first week. Increased swelling after the third day may be an indication of infection rather than continued post surgical oedema[14].

Surgical extraction of impacted third molars usually results in some degree of trismus because the inflammatory response to the surgical procedure is sufficiently widespread to involve several muscles of mastication. The trismus may also results from multiple injection of local anaesthetics specially if the injections have penetrated muscles[14].

The introduction of non steroidal anti-inflammatory drug (NSAIDs- Diclofenac Potassium, Sodium) has significantly improve the management of post operative pain in dentistry and surgery. There are two possible mechanisms form the efficacy of NSAIDs when administered prior to surgical trauma. The first may simply be a pharmacokinetic advantage. By administering the NSAIDs prior to pain onset, drug absorption would have begun and therapeutic blood level will be present at the time of pain onset. Second, the presence of a cyclo-oxygenase inhibitor at the surgical site may limit the prostaglandins and prostacyclins associated with hyperalgesia and oedema. The use of corticosteroids has another preventive strategy for limiting post operative sequelae following impacted lower wisdom teeth extraction. Post operative swelling and oedema may be due in part to the conversion of phospholipids to the arachidonic acid by phospholipase A_2 and the resultant production of leukotrienes, prostacyclins, prostaglandins and thromboxane A_2 acting as mediators of the inflammatory response. The use of steroids may inhibit the initial step in this process[4].

The many factors that contribute to postoperative complications are complex. But they originate from an inflammatory process initiated by surgical trauma. The adverse effects of the wisdom tooth surgery on the quality of life has been reported to show a three fold increase in patients who experienced pain, swelling and trismus alone or in combinations compared to those who are asymptomatic. Many clinicians have thus emphasized the necessity for better management of pain, swelling and trismus in patient who undergo impacted third molar surgery[4].

The use of synthetic glucocorticoids in reducing such postoperative sequelae has been investigated extensively, where studies demonstrated statistically significant improvement in postoperative sequelae when corticosteroids are administered. Currently, various forms of corticosteroids with different potencies and effects have been made available to choose form[15].

Clinical trials in oral surgery have also supported the hypothesis that preemptive NSAIDs and corticosteroids are effective in delaying and preventing many post operative sequelae. The apparent interactions between the mechanisms of action of NSAIDs and steroid suggests that co-therapy may provide beneficial imflammatory and pain relief in absence of side effects[4].

The purpose of this study was to compare the effects of coadministered Dexamethasone and diclofenac potassium/-sodium with diclofenac potassium/sodium alone on the post operative management of pain, swelling and trismus following surgical removal of impacted lower third molar.

The apparent interactions between the mechanism of action of Non Steroidal Anti Inflammatory Drugs (NSAIDs) and steroid suggests that co therapy may provide beneficial inflammatory and pain relief in absence of side effects. The study was conducted keeping in view to compare the effect of co administered steroid-diclofenac potassium with diclofenac potassium alone, on the post operative management of morbidity specifically pain, swelling and trismus following removal of impacted lower third molars. The present study was contemplated to compare the effect of co-administered steroid and diclofenac Na^+/K^+ with diclofenac Na^+/K^+ alone on the postoperative morbidity like pain, swelling and trismus after surgical removal of impacted lower third molars.

Materials and Methods:

This study was a Prospective study. Conducted at Oral & Maxillofacial department, Military Dental Center, Combined Military Hospital, Dhaka cantonment, Dhaka, Bangladesh & Oral & Maxillofacial department, Dhaka Dental College and Hospital, Dhaka. The duration of the study was 01/01/2013 to 30/06/2014. Study population were the patients reported to Military Dental Center, Combined Military Hospital Dhaka and Oral & Maxillofacial department, Dhaka Dental College and Hospital, Dhaka and with impacted lower third molar teeth. Total 60 patients were randomly selected into two groups - Group A (Control group) -- NSAID alone post operatively – 30, Group B (Experimental group) --Taking Dexamethasone along with NSAID post operatively -30.

Pain measurement

In the evaluation of postoperative pain, visual analogue scale (VAS) were given to patients. To indicate the intensity of pain, the following categorization was used: 0 = no pain, 2 = mild pain, 4 = moderate pain, 6 = severe pain, 8 = very severe pain and 10 =unbearable pain. The patients were asked to mark pain level hourly during waking hours starting from the first hours of the operation. By use of these forms, the severities of pain were evaluated for the first 48 hours.

Measurement of facial width

As no published method satisfies all criteria for assessing facial swelling, It was decided to use a measuring tape in this study to measure facial width and swelling in one-dimension only. Facial width (swelling) was measured using a measuring tape. The reference points used were the tip of tragus of left and right ears, with the gonion in between. A single operator, repeating the procedure three times on each patient, made the measurements. The average of measurements was then taken (in cm) and recorded. The measurements were carried out just before the surgery and at post-operative days 1, 2 and 7. Postoperative swelling was expressed as a percentage increase in facial width.

Measurement of mouth-opening ability

A vernier-calibrated sliding calipers was used to measure maximum interincisal mouth-opening ability of the patient at the commencement of the procedure. The reference point used was incisal edge of the maxillary central incisor and incisal edge of mandibular central incisor at maximum opening available.

The measurements were made in triplicate and the average was recorded in millimetres (mm). The measurement was carried out just before the surgery and at post-operative days 1, 2 and 7. Postoperative trismus was measured as a percentage decrease in mouth opening.

Ethical consideration

Ethical clearance was obtained from the Research Committee of DGMS office. Permission to use the records will be obtained from Oral & Maxillofacial department, Military Dental Center, Combined Military Hospital, Dhaka cantonment, Dhaka, Bangladesh; Oral & Maxillofacial department, Dhaka Dental College and Hospital, Dhaka. Written informed, consent will be obtained from the patient's or legal guardian for the use of any photographs. Patient confidentiality will be strictly maintained .No names, addresses or contact details of the patients will be divulged.

Method of data processing & statistical analysis

Data was analyzed using SPSS for windows (v11.5, SPSS Inc, Chicago, IL) statistical software package. One-way analysis of variance, student's t-test and χ^2 were used for repeated measures for category rating scale, interincisal opening and facial swelling. The level of significance was set at $P < 0.01$.

Results:

Out of 60 patients maximum patients fall in the age group of 26-35years (46.66%) followed by age group15-25 years (38.33%). Horizontal and Vertical type of impaction (30%) were more common than other (figure 1). The measurement of pain status in 1st POD in Group-A was 5.467±1.2243 and in Group-B was 3.333±1.2130, In 2nd POD in Group-A was 2.500±0.9377 and in Group-B was 1.467±0.7303 which was statistically significant (table I). Regarding the measurement of swelling before operation the mean value in Group-A was 15.237 ± 0.7863 cm and in Group-B was 15.627 ± 0.9468 cm, In 1st POD in Group-A was 15.787±0.7687 cm and in Group-B was 15.789±0.9193 cm, In 2nd POD in Group-A was 16.263±0.7069 cm and in Group-B was 15.910±0.8934 cm which was statistically significant (table II). The measurement of inter incisal opening before operation the mean value in Group-A was 46.267±2.572 cm and in Group-B was 46.033 ±3.872 cm, In 1st POD in Group-A was 40.267±2.625 cm and in Group-B was 43.833 ±3.992 cm, In 2nd POD in Group-A was 34.967±2.809 cm and in Group-B was 42.567 ±4.040 cm. In 1st and 2nd POD the change in inter incisal opening was statistically significant (table III).

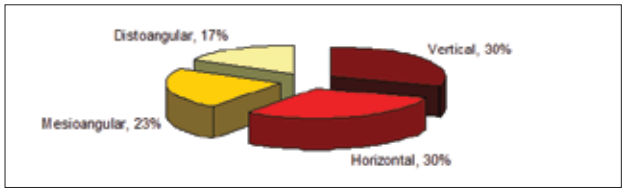

Figure- 1: Type of impaction.

Table-I: Status of pain after impacted lower third molar surgery (n=60).

Time	Group-A (Control group) Mean ± SD	Group-B (Experimental group) Mean ± SD	p value
1st POD	5.467±1.2243	3.333±1.2130	0.000#
2nd POD	2.500±0.9377	1.467±0.7303	0.000#
7th POD	0.2000±0.40684	0.067±0.2537	0.133

Table-II: Status of swelling after impacted lower third molar surgery (n=60).

Time	Group-A (Control group) Mean ± SD	Group-B (Experimental group) Mean ± SD	p value
Before	15.237±0.7863	15.627±0.9468	0.880
1st POD	15.787±0.7687	15.789±0.9193	0.992
2nd POD	16.263±0.7069	15.910±0.8934	0.095#
7th POD	15.2533±0.8080	15.6267±0.9468	0.106

Table-III: Status of trismus (inter incisal distance) after impacted lower third molar surgery (n=60).

Time	Group-A (Control group) Mean ± SD	Group-B (Experimental group) Mean ± SD	p value
Before	46.267±2.572	46.033 ±3.872	0.784
1st POD	40.267±2.625	43.833 ±3.992	0.000#
2nd POD	34.967±2.809	42.567 ±4.040	0.000#
7th POD	45.800±3.305	45.966±3.890	0.859

Discussion:

Surgical removal of impacted lower third molar tooth is usually Associated with post operative complications like pain, swelling and trismus as direct and immediate consequences of the surgical procedure. By pharmacologically controlling the extent of the inflammatory process, the intensity or severity of postoperative sequelae may be reduced[4].

One technique that has been proposed for reduction of postoperative inflammation is the administration of corticosteroids. Cortisol and the synthetic analogue of cortisol like dexamethasone have the capacity to interfere with the physiologic processes of inflammation and thus, suppress the development of local fever, redness, swelling and

tenderness by which inflammation is recognized[4].

Another technique is to control the synthesis of prostaglandins. Prostaglandins play a major role in the induction of pain, inflammation and fever. The reduction of biosynthesis of prostaglandins by inhibition of the cyclo-oxygenase enzyme system is considered an important mechanism of action of NSAIDs. When administered preoperatively, NSAIDs have been shown to be particularly effective in combating postoperative pain[4].

Preventive strategies for postoperative management of pain and inflammation are based on the known ability of NSAIDs to block the arachidonic acid cascade. When NSAIDs are administered preoperatively, absorption and distribution of the medication may occur before the initiation of tissue trauma, the ensuing synthesis of prostaglandins and the subsequent inflammatory response. Prevention of the inflammatory response may decrease the sequelae of tissue trauma; especially the accompanying pain[4].

Diclofenac Na^+/K^+ has been shown to be useful in controlling postoperative pain after removal of third molars. Diclofenac Na^+/K^+ is known to possess both analgesic and anti-inflammatory effect. Due to its anti-inflammatory effects, the administration of dexamethasone may synergize the anti-inflammatory effect of Diclofenac Na+/K+ and contribute to the reduction of inflammatory exudates as well as oedema and pain. Therefore the co-administration of diclofenac Na^+/K^+ and dexamethasone may be expected to reduce post-operative pain more than that achieved with diclofenac Na^+/K^+ alone[4].

The present study assessed the clinical effect of corticosteroid that is dexamethasone-diclofenac Na^+/K^+ combination and diclofenac Na^+/K^+ alone on pain, facial swelling and trismus. A study by Chiapasco M, Crescentin Metal on the complications after removal of impacted third molars that compared adults with patients aged 16-19 years showed that such complications were more frequent in patient 24 years of age and older[16]. The patients in this study had a mean age of 28 years and older which is very much close to that study.

The radiographic analysis of the type of impaction of lower third molar showed that maximum patient fall in horizontal and vertical type of impaction (30%) followed by Mesioangular (23%) and Distoangular (17%). In a study held in CMH Dhaka by Lt Col Golam Mohiuddin Chowdhury et al[17]. It was found that Mesioangular type of impaction was higher than other variety.

Regardless of the drug combination used, the pattern of postoperative pain has been reported to increase between the post-operative days 1 and 2, after which the symptoms subside gradually within one week[4].

Corticosteroids are employed particularly after surgery to reduce the presence of inflammatory mediators and thus lessen fluid transudation and edema. Although many studies have reported statistically significant analgesic action with corticosteroid use others have found the analgesic efficacy of corticosteroid to lack statistical significance ($p>0.05$), despite a lessening of postoperative pain. One study surprisingly suggests the possibility that corticosteroid treatment can increase patient reaction to pain secondary to a suppression of endorphin β levels[18].

In present study shows the comparison of pain intensity between Group-A and Group-B in different ages of patients after impacted lower third molar surgery. In Group-A and Group-B on 1st POD the Mean ± SD of pain severity on VAS is 5.467±1.224 and 3.333±1.213 respectively .The difference is statistically significant. In Group-A and Group-B on 2nd POD the Mean ± SD of pain severity on VAS is 2.500 ± 0.9377 and 1.467 ± 0.7303 respectively. The difference is also statistically significant. Among the age group of 15-25 yrs in control group the intensity of pain is higher than experimental group on 1st and 2nd POD and after 7th POD it not come to basal state. Some amount of pain persist even after 7th POD need additional dosage of medication. But among the age group of 26-35 yrs in control group the intensity of pain is significantly higher than experimental group and both come to basal state after completion of treatment.

Post-surgical facial edema is difficult to quantify accurately, since it requires a three-dimensional measurement with an irregular, convex surface and can manifest itself internally as well as externally. In the present study, a single measurement from the tip of tragus to going on to the tip of contralateral tragus was taken. In a study by Schultze-Mosgau Setal, comparing the use of NSAID (Ibuprofen) and steroid (methylprednisolone) concluded that the combination of both medications was well suited for treating these effects and should be used when extensive postoperative swelling of soft tissue is expected[19].

In present study showed that before surgery there was no significant difference of swelling between 2 groups. On 1st POD there was also no significant difference two group as ($p > 0.10$). But on 2nd POD swelling reduced significantly in Group-B in comparison to Group-A as ($p < 0.01$). on 7th POD both group returned to basal state as ($p > 0.10$). This result shows that co-administration of dexamethasone and diclofenac Na^+/K^+ also enhances the control of postoperative facial swelling which supports study done by Babatunde Olamide Bamgbose et al .

Many studies have used corticosteroid in combination with other drugs to evaluate their effect upon pain, swelling and trismus. Statistically significant findings have been reported when corticosteroid are used in combination with drugs such as diclofenac in application to pain and swelling, but not in reference to trismus[18].

In present study the measurement of inter incisal distance before operation the mean value in Group-A was 46.267±2.572 cm and in Group-B was 46.033 ±3.872 cm, In 1st POD in Group-A was 40.267±2.625 cm and in Group-B was 43.833 ±3.992 cm, In 2nd POD in Group-A was 34.967±2.809 cm and in Group-B was 42.567 ±4.040 cm. In 1st and 2nd POD the change in inter incisal opening was statistically significant. These results indicate a positive clinical association between the adjunct use of dexamethasone and postoperative recovery of trismus in impacted lower third molar surgery. Similarly, the study

done by Bamgbose et al. [4] found that the combination therapy was more effective in controlling pain, swelling, and trismus following third molar surgery.

Conclusion:

The use of corticosteroid with NSAID appears to be a safe and effective method to reduce postoperative clinical symptoms like pain, trismus, and swelling after impacted lower third molar surgery and can be used when extensive postoperative swelling of the soft tissue is anticipated.

Conflict of Interest: None.

Acknowledgement:

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References:

1. Chiapasco M, De Cicco YL, Marrone G. Side effects and complications associated with third molar surgery. Oral SurgOral Med Oral Pathol. 1993; 76: 412-20.

https://doi.org/10.1016/0030-4220(93)90005-O

2. Akadiri OA, Obiechina AE. Assessment of difficulty inthird molar surgery- a systemic review. Oral and Maxillofacial Surgery. 2009; 67(4):771-4.

https://doi.org/10.1016/j.joms.2008.08.010

PMid:19304033

3. P Capuzzi, L Montebugnoli, MA Vaccaro. Extraction of impacted third Molars, A longitudinal prospective study on factors that affect post operative recovery. Oral surgery Oral Med Oral Path. 1994; 77(4): 341-3.

https://doi.org/10.1016/0030-4220(94)90194-5

4. Bamgbose BO, Akinwande JK. Effects of co-administered dexamethasone and diclofenac potassium on pain, swelling and trismus following third molar surgery. Head and Face Medicine. 2005; 1: 11.

https://doi.org/10.1186/1746-160X-1-11

PMid:16274480 PMCid:PMC1291385

5. Pavlin DJ, Chen C, Penaloza DA. Pain as a factor complicating recovery and discharge after ambulatory surgery. Anaesthesia analgesia. 2002; 95: 627-34.

https://doi.org/10.1097/00000539-200209000-00025

https://doi.org/10.1213/00000539-200209000-00025

6. Al-Khateeb TH, Alnahar A. Pain experience after simple tooth extraction. Journal of Oral and Maxillofacial Surgery. 2008; 66: 911-7.

https://doi.org/10.1016/j.joms.2007.12.008

PMid:18423280

7. De Beer Jde V, Winemaker MJ, Donnelly GA. Efficacy and safety of controlled release oxycodone and standard therapies for postoperative pain after knee or hip replacement. Canadian journal of surgery. 2OO5; 48: 277.

8. Niv D, Devor M. Transition from acute to chronic pain. In: Aronoff GM, ed. Evaluation and treatment of chronic pain. 3rd ed. Baltimore: Williams & Wilkins; 1998: 27-45.

9. U.S. Department of Health and Human Services, Agency for Health Care Policy an Research. Acute pain management: Operative or medical procedures and trauma. Rockville, Md.: DHHS, Public Health Service, AHCPR; 1992. AHCPR publication 92- 0032.

10. Urquhart E. Analgesic agents and strategies in the dental pain model. J Dent. 1994; 22: 336-41.

https://doi.org/10.1016/0300-5712(94)90084-1

11. Woolf CJ. Recent advances in the pathophysiology of acute pain. Br J Anaesth. 1989; 63(2): 139-46.

https://doi.org/10.1093/bja/63.2.139

PMid:2669905

12. Dworkin RH. Which individuals with acute pain are most likely to develop a chronic pain syndrome? Pain Forum. 1997; 6:127-36.

https://doi.org/10.1016/S1082-3174(97)70009-6

13. El Shenaway H, Aboelsoud NH, Zaki AA, El Zawahry M, Shaibeta A. Postoperative Pain Control in Patients after Lower Third Molar Extraction. Journal of American Science. 2010; 6(11): 1068-1072.

14. Hupp JR, Edward E III, Myron RT. CONTEMPORARY Oral and Maxillofacial Surgery. 5th ed. 2008; USA: Elsevier.

15. Tarek L, AI-Khateeb HA, Marouf MA. Clinical evaluation of Dexamethasone VS Methylprednisolone for reducing Postoperative Inflammatory Sequelae following Third Molar surgery. The Saudi Dental Journal. 1996; 3: 131-5.

16. Chiapasco M, Crescentin M, Romanoni G. Germectomy or delayed removal of

mandibular impacted third molars : the relationship between age and incidence of

complications. J Oral Maxillofac Surgery. 1995; 53: 418-22.

https://doi.org/10.1016/0278-2391(95)90715-7

17. Chowdhury GM. Radiographic evaluation of angulation, eruption space and depth of impacted mandibular third molar tooth. Bangladesh Armed Forces Medical Journal. 2005; 36:11-13.

18. Ata-Ali J, Ata-Ali F, Peñarrocha-Oltra D, Peñarrocha M. Corticosteroids use in controlling pain, swelling and trismus after lower third molar surgery. J Clin Exp Dent. 2011; 3(5): e469-75.

https://doi.org/10.4317/jced.3.e469

19. Schultze-Mosgau S, Schmelzeisen R. Use of ibuprofen and methylprednisolone for prevenyion of pain and swelling after removal of impacted third molars. J Oral Maxillofac Surg. 1995; 53:2-7.

https://doi.org/10.1016/0278-2391(95)90486-7

DOI: https://doi.org/10.3329/medtoday.v32i1.44821

ORIGINAL ARTICLE OPEN ACCSESS

Evaluation of Nutritional Status of Hospitalized Infants with Cholestatic Jaundice in a Tertiary Care Center in Bangladesh

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Abstract

Introduction: *Nutritional management becomes crucial for the infants of cholestatic jaundice as they suffer from impaired digestion and absorption of fatty acid and fat soluble vitamins. Thus evaluation of nutritional status was done to identify undergoing malnutrition.* ***Materials and Methods:*** *It was a cross-sectional, prospective study done in the indoor patients department of paediatric gastroenterology and nutrition, BSMMU; since April 2016 to September 2016 (6 months). Purposive sampling was done from the infants (2 weeks to 12 months age) with cholestatic jaundice or having neonatal jaundice persisting more than 2 weeks. Linear regression analysis was done for correlation between different anthropometric variables.* ***Results:*** *Total 47 patients were enrolled in this study although 93 patients were admitted with cholestatic jaundice in that time period in the department. Most of them were found biliary atresia (61.70%) Idiopathic Neonatal Hepatitis (31.91%). Among anthropometric indices, Weight for length Z(WLZ) Score mean was -1.6 (±1.6), Triceps skin fold thickness (TST) mean 5.48 (±2.75) mm, and Subscapular skin fold thickness(SST) mean was 4.13 (±2.39)mm. Linear regression analysis of each variables done with other and found positive correlation between TST with WLZ Score and SST with WLZ score and found highly significant (p=0.008 in ANOVA) and (p=0.011 in ANOVA) respectively.* ***Conclusion:*** *Cholestatic babies were found mild to moderately malnourished in our study. Skin fold thickness was found to be an important early clinical marker for diagnosis of malnutrition which was found positively correlated with the standard procedure of anthropometry in our study.*

Key words: *Cholestatic jaundice, Nutritional status, Biliary atresia, Neonatal hepatitis.*

Number of Figures: 02; Number of References: 13; Number of Correspondence: 06.

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Introduction:

Jaundice is a clinical sign characterized by yellowish discoloration of skin and mucous membrane caused by elevated serum bilirubin concentration. Elevated conjugated or direct bilirubin in serum >20% of total serum bilirubin is one of the important biochemical markers of cholestasis indicating perturbation of bile flow [1,2]. Conjugated hyperbilirubinemia is less common, affecting approximately 1 in 2,500 infants [3,4,5] and biliary atresia is one of the important causes of cholestasis occurring in between 1 in 10,000 to 1 in 20,000 infants[6]. In India it constitutes 19% to 33% of all chronic liver diseases in children reporting to tertiary care hospitals[7]. Early recognition of cholestasis is essential for effective treatment of medical condition or the surgical management of biliary anomalies, even when treatment is not yet available or effective, infants who have progressive liver disease can be benefited from optimal nutritional support and medical management of the complications of cholestasis and possibly cirrhosis [8].

The consequences of prolonged cholestasis are profound, resulting in malabsorption, failure to thrive, and deficiencies of fat-soluble vitamins like A, D, E, K and various minerals like Calcium, Phosphate and Zinc. Cholestatic babies are at special risk for life-threatening bleeding due to vitamin K deficiency. These babies need more calories (130% more than RDA) to maintain growth and also nutritional supplementation at diagnosis and thereafter. Very few studies found in Bangladesh as well as in other parts of the world regarding the nutritional status of infants with cholestatic jaundice. Routine measurements of anthropometry like stunting or wasting can be a good marker of chronic nutritional deprivation. In Bangladesh, 36 % among all children under 5 are considered to be

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short for their age or stunted, while 12 % are severely stunted (below -3 SD). The prevalence of stunting increases with age from 14% of children under age 6 months to 46 % of children 18-23 months and decreases to 38% among children 48-59 months[9]. Thus, study was conducted to indentify the nutritional status of the study subjects and quantify them among other parameters for correlation so that we could manage accordingly.

Materials and Methods:

It was a prospective cross-sectional study done in the indoor patients Department of Paediatric Gastroenterology and Nutrition, BSMMU since April 2016 to September 2016 (Six months after approval of protocol) among the children admitted between ages 2 weeks -12 months with cholestatic jaundice. There were 93 patients admitted with cholestatic jaundice during study period and among them 47 were enrolled in the study. Inclusion criteria were Infants (2 weeks to 12 months age) with cholestatic jaundice (S. conjugated bilirubin > 2 mg or more than 20% of total serum bilirubin) or Neonatal jaundice persisting more than 2 weeks. Patients with age less than 2 weeks or more than 12 months, jaundice due to other causes, seriously ill infants who need referral or whose parents refused to be enrolled in the study were excluded. Sample selection was done by purposive sampling; interview was taken after written informed consent. Data were collected and put in the structured questionnaire then tabulated for analysis. A detailed history was obtained from mother or informant of infants with cholestatic jaundice (scleral icterus, dark urine and pale stool) persisting beyond 2 weeks of postnatal age and recorded in a predesigned questionnaire. A thorough physical examination, including examination of the eyes, stool color was observed daily after admission to look for persistent or intermittent nature of icterus and pale stool and examination findings were recorded. Anthropometric measurement (Weight, Length, MUAC, Triceps skin fold thickness, Sub-scapular skin fold thickness) was taken by standard routine procedure. Weight was measured by electronic baby weighing scale in kilogram and gram, Length was measured by Infantometer in centimeter, Mid Upper Arm Circumference (MUAC) was measured by Shakir's Tape in millimeter, measurement of Triceps & Sub-scapular skin fold thickness was done by Harpenden calipers in millimeter. After measurement, each data was compared with 'WHO Child Growth Standards' published by Center for Disease Control and Prevention, November 1, 2009. Complete blood count and liver function tests (S. bilirubin - total & direct, ALT, ALP, and PT) was done. Urine was tested for non-glucose reducing substances. Ultrasonography of the hepatobiliary system, and Hepatobiliary scintigraphy was done. In scintigraphy, absence of radioactivity in the small bowel after 24 hours was considered as absent tracer excretion. Data were processed and analyzed using SPSS (Statistical Package for Social Sciences) software version 16. Continuous scale data were presented as mean, standard deviation, range and Categorical data were presented as number percentage. The summarized data were present in the table and chart. All the participants were volunteered. Informed written consent was taken from all patients' guardian. All documents were preserved confidentially. Questions were asked in Bangla and also in local easily understandable language.

Results:

Anthropometric measurements showed mean weight (n=47) was 4651.48 (±1456.36) grams, mean length (n=47) was 57.36 (±3.93) cm, mean MUAC(n=7) was 112.50(±7.50) mm, Triceps Skin fold Thickness(TST) (n= 23) 5.48 (±2.75) mm and TST Z score mean (n=23) was -2.83 (±2.21) , Sub-scapular Skin fold Thickness(SST) (n=23) 4.13(±2.39) mm and SST Z score mean was (n=47) -3.26 (±2.62). Weight for age Z score (WAZ) mean was -2.51 (±1.59) (n=47) , length for age Z score (LAZ) mean was-1.92 (±1.49) (n=47) and weight for length Z score mean was -1.61 (±1.60) (n=47). Linear regression analysis of each variables done with other and found positive correlation between Triceps skin fold thickness with Weight for length Z Score and it was highly significant (p=0.008 in ANOVA). Same positive correlation was found between Subscapular skin fold thickness with Weight for length Z Score, it was also statistically highly significant (p=0.011 in ANOVA). On the other hand Triceps and Subscapular skin fold thickness values were found positively correlated with Length for age Z score values but those were not statistically significant (p=0.097 and p=0.645 respectively). The following figures describes this scenario in details.

Figure 1 shows the correlation between Weight for Length Z score (Wasting) with Triceps Skin Fold Thickness (TST) Z score by Linear Regression Analysis which found a positive correlation between these variable and it was highly significant (p=0.008 in ANOVA).

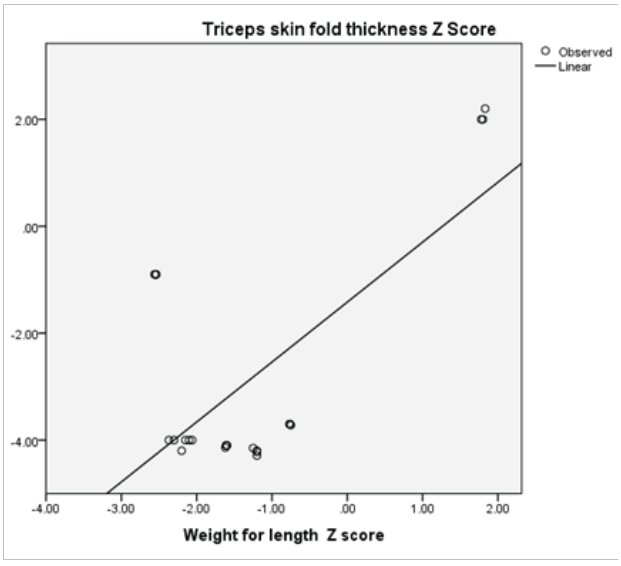

Figure-1: Linear Regression Analysis of Triceps Skin Fold Thickness Z score (TSTZ) and Weight for Length Z score (WLZ) correlation analysis.

Figure 2 shows the correlation between Weight for Length Z score (Wasting) with Subscapular Skin Fold Thickness (SST) Z score by Linear Regression Analysis which found a positive correlation between these variable and it was highly significant (p=0.011 in ANOVA).

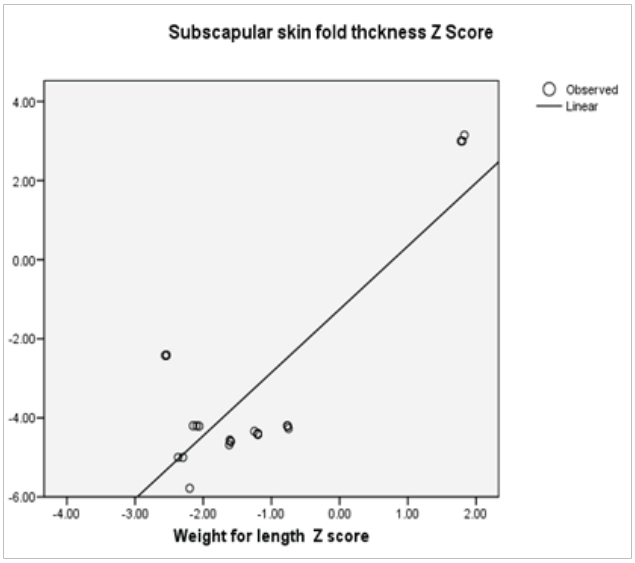

Figure-2: Linear Regression Analysis of Subscapular Skin Fold Thickness Z score (SSTZ) and Weight for Length Z score (LAZ) correlation analysis.

Discussion:

This study was done to evaluate the nutritional status of the cholestatic infants who were and we found most of them were severely underweight (44.7%), moderately stunted (46.8%) and mildly wasted (44.68%). This result indicates a quite high prevalence of stunting among cholestatic infants (87.2%) in respect to our national prevalence of stunting (14%) according to Bangladesh Demographic Health Survey 2014[9].

Present study showed the mean age was 3.19 (±1.67) months and minimum age was 1 month and maximum being 7 months. Hamid F, Afroza A and Ray P C [1] compared between Biliary Atresia(BA) group with Neonatal Hepatitis(NH) group and showed mean age of babies in BA group was 4.13 (± 2.11SD) months, where as in NH babies mean age was 3.86 (± 2.11SD) months. We found most of the patients were male 41 (87%), male female ratio 6.83:1 among them. Among cholestatic babies Biliary atresia has been reported to be common in female infants in several others previous studies. Where as, we found only 1 case out of 29 Biliary atresia cases is female. However, in Karim ASM B and Kamal M[11] series, 10 of 16 infants with BA were male (62.5%). This indicates a male preponderance in our region. Most of our study subjects came from rural (72.3%) low socioeconomic (57%) background where there is custom of giving more privilege to male child than female. There is also some difference we found in relation to initial diagnosis of the babies. Our study found BA in 29 (61.7%) cases and NH in 15 (31.91%) cases where as Hamid F, Afroza A and Ray P C [1] showed BA in 12 (40%) cases and NH 18 (60%) cases which is almost reverse picture of our study. Both the studies done in BSMMU but the difference may be due to the time of study, the former was done in 2007. In these long 9 years Paediatricians became more conscious about the surgical treatment option of BA cases in BSMMU and thus responded promptly in referral which was also reflected in our study subject's age of enrollment (72.34% before 3 months of age).

Silva F et al.[12] study done over 91 children with cholestasis, with current median age of 12 months. WAZ and HAZ indices below −2 Z-scores were observed in 33% and 30.8% of patients, respectively; where as in our study it was 57.44% and 59.57% respectively. Concerning the WLZ index and BMI, only 12% and 16% of patients, respectively, were below −2 Z-scores and in our study WLZ below -2 was 31.91%. Regarding Mid Upper Arm Circumference (MUAC), 43.8% of 89 evaluated patients had some depletion. Observing the Triceps Skin Thickness(TST), 64% of patients had depletion, and 71.1% of the 45 evaluated patients had some degree of depletion regarding the MUAC. They concluded as, the use of weight for nutritional evaluation may underestimate the detection of malnutrition in patients with chronic liver diseases due to visceromegaly, subclinical edema, and/or ascites. The anthropometric indices that consider weight and height, such as WLH and BMI, may also not reveal the real degree of depletion attributable to chronic clinical condition of these patients, in which both weight and height are impaired. TST and MUAC measures appear to be more accurate parameters for nutritional evaluation of patients with liver diseases and cholestasis. Our study found 36 infants out of 47 were malnourished (weight for length Z score below -1). We found most of them were severely underweight (44.7%), moderately stunted (46.8%) and mildly wasted (44.68%). This result indicates a quite high prevalence of stunting among cholestatic infants (87.2%) in respect to our national prevalence of stunting (14%) according to Bangladesh Demographic Health Survey 2014. Skin fold thickness also reflects the same picture. We could measure skin fold thickness in ≥3 months age group babies and found 23 study subjects and in those cases triceps and sub-scapular skin fold thickness Z-score mean was -2.83 (±2.21) and -3.26 (±2.62) respectively. If we consider the same gradation for skin fold thickness Z-score as we used for weight for length Z-score, this would indicate the babies were moderate to severely malnourished. Only 6(26%) out of 23 babies were found Z-score above -3 in both triceps and sub-scapular skin fold thickness measurement. Although several previous studies found MUAC as an important and relevant indicator of nutrition particularly in this age group, We found only 7 babies ≥6 months age group and MUAC was also measured in those babies and found mean 112.50 (±7.50) mm, all of them were in malnourished category including 4 babies in the category of severe acute malnutrition(SAM). This result strengthened our present study.

We have also done the analysis of correlation between Skin fold Thickness with Stunting and, Skin fold Thickness with

Wasting and found positive correlation in all aspects between these but degree of stunting was not significantly associated with degree of skin fold thickness Z score (TSTZ with LAZ p value was 0.097 and SSTZ with LAZ p value was 0.645) as shown in Figures. We have shown that TSTZ and SSTZ was highly significantly associated with Wasting that is WLZ (TSTZ with WLZ p value was 0.008 and SSTZ with WLZ p value was 0.011).

We also found cholestatic babies with anaemia, raised serum marker of parenchymal and biliary epithelial cell damage like raised Alanine Aminotransferase (ALT), raised Alkaline Phosphatase (ALP), raised Gamma-glutamyl transpeptidase (GGTP) and most importantly they had coagulopathy as reflected by raised PT. Their mean Haemoglobin was 9.60 (±2.32) gm/dl, mean total S. bilirubin was 11.31 (±3.66) mg/dl, mean Direct S. Bilirubin was 6.97(±3.39) mg/dl, mean S. ALT was 142.46 (±83.72) IU/L which was 3.2 times higher than upper limit normal value, mean GGTP was 333.76 (±263.35) IU/L which was 2.5 times higher than upper limit normal for any age, mean ALP was 695.48 (±855.04) IU/L and mean Prothombin Time (PT) was 36.53 (±39.95) seconds which was 2.25 times higher than upper limit normal value . Mattar et al[13] study showed mean ALP (U/l) 1,708 (938-2,020), GGTP (U/l) 216 (104-351), Total bilirubin (mg/dl) 9.6 (5-13.1), Direct bilirubin (mg/dl) 6.9 (4-11.6), Prothrombin activity (%) 77 (39-98). It has been reported that serum bilirubin rarely exceeds 12 mg/dL (and may be as low as 5-8 mg/dL) in infants with BA despite complete bile duct obstruction, whereas it may exceed 20 mg/dL in those with NH[11]. In the series by Karim ASM B and Kamal M[11], mean serum bilirubin was 10.4 mg/dL in infants with BA and 14.1 mg/dL in those with NH_2. Serum GGTP level was the only biochemical test found to be of discriminating value between hepatocellular Cholestasis and Biliary atresia. GGTP values less than 200 U/L correlated with the diagnosis of hepatocellular cholestasis while GGTP values more than 200 U/L favored the diagnosis of biliary atresia. However, our study did not correlated with such findings of GGTP. We did not find any cholestatic baby having non-glucose reducing substance in urine.

During Ultrasonography of hepatobiliary system we found 24 (51.1%) babies with abnormal findings like absent or non-contraction of gall bladder, hepatomegaly, splenomegaly, ascites. Hepatobiliary scintigraphy also done in all patients and found absent tracer activity in small intestine in 25 (53.2%) babies.

Suchy FJ[8] study proposed some nutritional interventions for cholestatic babies like: giving energy dense food which will fulfill 125-130% of RDA of the age group, giving Medium Chain Triglyceride (MCT) which does not require bile acid for absorption, adequate protein intake (2-3gm/kg/day in infants), giving vitamin A 5000-25000 U/d of water miscible preparation, Vitamin D 0.05-0.3 mcg/kg/day, Vitamin E d-alpha tocopherol polyethylene glycol-1000-succinate 15-25 u/kg/day, Vitamin K Phytomenadione 2.5-5 mg twice weekly up to 5 mg/day orally or Inj IM 2-5 mg every 4 weekly. Sokol RJ and Stall C[10] also recommends Calcium 50 mg/kg/day orally, Phosphate 25mg/kg/day orally , Zinc 1 mg/kg/day orally. Suchy FJ[8] also recommends that the diet of the cholestatic babies should be free of Copper, Aluminium and Manganese.

Conclusion:

Cholestasis is an important cause of growth failure in neonate and in early infancy which can be prevented by early identification and nutritional management. These babies were found mild to moderately malnourished in our study. Skin fold thickness is found to be correlated with the standard procedure of anthropometry for diagnosis of malnutrition at an early age.

Conflict of Interest: None.

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References:

1. Hamid F, Afroza A, Ray PC. Aetio-Clinical Profile of Cholestatic Jaundice During Infancy-Study of 30 Cases in a Tertiary Care Hospital. Bangladesh Medical Journal. 2012; 41(2): 34-39.

https://doi.org/10.3329/bmj.v41i2.18804

2. Tricia Lacy Gomella. Hyperbilirubinemia, Direct(Conjugated Hyperbilirubinemia). In: Gomella T.L., Cunningham M.D., and Eyal F.G., Neonatology Management, Procedures, On-Call Problems, Diseases, and Drugs. New York Chikago, USA: Mc Graw Hill LANGE; 2009.

3. Bhatia V, Bavdekar A, Matthai J, Waikar Y, sibal A. Management of Neonatal Cholestasis: Consensus Statement of the Pediatric Gastroenterology. Indian Pediatrics. 2015; 51: 203-210.

https://doi.org/10.1007/s13312-014-0375-2

PMid:24736908

4. Gilmour SM. Prolonged neonatal jaundice: When to worry and what to do. Paediatric Child Health. 2004; 9(10): 700-704.

https://doi.org/10.1093/pch/9.10.700

PMid:19688078

5. Dick MC, Mowat AP. Hepatitis syndrome in infancy: an epidemiologic survey with 10-year follow up. Arch Dis Child.1985; 60: 506-512.

6. Mowat AP, Davidson LL, Dick MC. Earlier identification of biliary atresia and hepatobiliary diseases:selective screening in the third week of life. Arch Dis Child.1995; 72: 90-92.

https://doi.org/10.1136/adc.72.1.90

PMid:7717750 PMCid:PMC1510987

7. John M. Evaluation of cholestatic jaundice in young infants. Indian Pediatrics. 2001; 38: 893-8.

8. Suchy FJ. Neonatal Cholestasis. Pediatrics in Review. 2004; 25: 388-296.

https://doi.org/10.1542/pir.25-11-388

PMid:15520084

9. Bangladesh Demographic Health Survey 2014. Dhaka. National Institute of Population Research and Training, Ministry of Health and Family Welfare, Bangladesh 2015.

10. Sokol RJ, Stall C. Anthropometric evaluation of children with chronic liver disease. Am J Clin Nutr. 1990; 52: 203-208.

https://doi.org/10.1093/ajcn/52.2.203

PMid:2375285

11. Karim ASM B, Kamal M. Cholestatic jaundice during infancy: experience at a tertiary-care center in Bangladesh. Indian J Gastroenterol. 2005; 24: 52-54.

12. Silva F, Ferri PM, Queiroz TC, Barbosa PS, Oliveira MC, Pereira LJ, et al. Nutritional evaluation of children-with chronic cholestatic disease. J Pediatr (Rio J). 2016; 92:197-205.

https://doi.org/10.1016/j.jped.2015.07.006

PMid:26632247

13. Mattar RHGdM, Azevedo RAd, Speridiao PGL, Neto UFN, Morais MBd. children with chronic hepatic disease with and without cholestasis. J Pediatr (Rio J). 2005;81:317-24.

DOI: https://doi.org/10.3329/medtoday.v32i1.44822

ORIGINAL ARTICLE OPEN ACCSESS

EEG as a Predictor of Poorly- Controlled Childhood Epilepsy

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Abstract

Introduction: *The aim of the present study was to determine electroencephalographic factors associated with poorly controlled epilepsy.* ***Materials and Methods:*** *This retrospective study was performed from January 2007 to December 2008 at Paediatric Neurology outpatient department in Bangabandhu Sheikh Mujib Medical University, Dhaka, among the children with epilepsy of 7 months to 15 years age who had history of at least 6 months treatment with rational antiepileptic drugs daily with adequate compliance. There were two groups of patients; group 1, consisted of 50 poorly controlled epilepsy patients and group 2, comprised 50 well-controlled epilepsy patients. We retrospectively reviewed EEGs and medical records from these children. Features of initial EEGs findings were compared between the two groups.* ***Results:*** *Significant electroencephalographic predictors of poorly controlled epilepsy were: abnormal initial EEG ($p=0.025$), EEG background abnormality ($p<0.001$), frequent sharp wave/spike ($p<0.001$) and hypsarrhythmia ($p=0.046$). No significant difference was noticed between the two groups in respect to the location of spikes/ sharp waves. With multiple logistic regression, independent predictors of poor seizure control were EEG background abnormality and frequent sharp wave/spike.* ***Conclusion:*** *The study showed several Electroencehalographic factors that can be identified early in the course of childhood epilepsy which can predict development of poor seizure control. Knowledge of these factors will help us to discriminate our patients and pay more attention to those at risk of developing poorly controlled epilepsy.*

Keywords: *Electroencephalogram (EEG), Poorly controlled epilepsy.*

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Introduction:

Seizures are common in the paediatric age group and occur in approximately 10% of the children[1]. The largest number of newly diagnosed patients with epilepsy occurs between birth and 2 years of age[2]. Epilepsy is considered to be present when two or more unprovoked seizures occur at an interval greater than 24 hours apart[1]. One or more seizures per month over a period of 6 months or more even after experiencing trials of at least two different antiepileptic drugs alone or in combination at optimum doses with adequate compliance is called intractable epilepsy[3]. Epilepsy is refractory when seizures are so frequent or severe that they limit the patient's ability to live life fully according to his or her wishes or necessitate the use of medications that, although effective, produce adverse effects[4].

Fortunately, most children with epilepsy do well when treated with an antiepileptic drug[5]. Numerous studies have reported predictors of favorable outcome of epileptic seizures in children and adults. Most report that 70-80% of patients eventually become seizure free[6]. But 10-20% of children with epilepsy have persistent seizures refractory to drugs, and those cases pose a diagnostic and management challenge[1]. However, what becomes of the group of children who continue to have seizures despite adequate trials of drugs? Although this is a small group of children, the medical, social, and economic consequences of poorly controlled seizures can be enormous. These children are at high risk for behavioral and academic difficulties[7]. In addition to the often catastrophic effects of uncontrolled seizures on the child, the burden on the parents of dealing with the multitude of daily problems accompanying intractable epilepsy cannot be underestimated[2].

Most studies regarding poor seizure control are concentrated towards the clinical predictors. Very little is known about the childhood epilepsies in Bangladesh[8]. Factors most predictive of poor seizure remission were: multiple types of seizures, poor cognition at presentation, high rates of seizures, associated motor disability and EEG abnormalities[8].

One Indian study showed that EEG was abnormal in 69% cases with background abnormality being present in 20% of intractable epilepsy[9]. Another study from Iran showed that the 1st EEG was abnormal more in patients of intractable epilepsy than those of well controlled epilepsy[10]. The outcome of seizure is poorer if EEG shows background abnormality and focal epileptiform activity[11]. In general patients with anterior temporal and frontal focus tend to have lower remission rate than those with mid temporal focus[11]. But another study did not notice any significant difference in severity of epilepsy

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with frontal and temporal focus[9]. Focal EEG slowing was associated with an increased risk of intractableepilepsy[12]. Children with intractable epilepsy were more likely to have abnormal EEG background, frequent discharges (>1/60s) and focal spike and waves than those with well controlled epilepsy[13]. Generalized epileptiform activity, whether early or well along in the course of new cases of epilepsy, is associated with a lower chance of remission[14].

As literatures were reviewed there were no published data about this type of study in our country. This study is intended to provide electroencephalographic information regarding poorly controlled epilepsy that may guide the physician for early identification of these patients for counseling their families, selecting patients for intensive investigations and treatment, as well as early consideration of epilepsy surgery to prevent consequences of poorly controlled epilepsy on quality of life.

Materials and Methods:

This case-control study was conducted from January 2007 to December 2008 at Paediatric Neurology OPD, Bangabandhu Sheikh Mujib Medical University. 50 cases of intractable epilepsy in group-1 and 50 cases of well-controlled epilepsy in group-2 were enrolled in the study. Children of either sex with epilepsy, aged 7 months to 15 years with history of at least one unprovoked seizure per month for 6 or more months and history of treatment with 2 or more rational antiepileptic drugs (AED) daily, either singly or in combination for at least 6 months and adequate compliance to antiepileptic drugs were considered as case. Children of either sex with epilepsy, aged 7 months to 15 years who had been seizure-free during 6 months after start of treatment were considered as control. Poorly controlled epilepsy was defined as one or more seizure per month over a period of 6 months or more even after experiencing trials of at least two different antiepileptic drugs alone or in combination at optimum doses with adequate compliance. Well-controlled epilepsy was defined as no seizure during 6 months after start of treatment with AED.

Once the child was reporting, a structured questionnaire was completed, containing pre-defined variables of clinical and EEG information that may help predicting the intractability. Then a detailed history including gender, age of onset, number of seizures before starting the treatment, type of epilepsy, character of seizure, status epilepticus, initial seizure frequency, history of neonatal seizures, family history of epilepsy, history of complex febrile seizure, treatment history, etc. were recorded and medical records were reviewed. Details regarding antiepileptic drugs were recorded i.e. number of drugs, duration of therapy, dosage and compliance. It was noted whether the choice of drugs were correct or incorrect in relation to seizure type and the dosage schedule was proper according to body weight. Thorough physical examination including neurodevelopmental and psychological assessment was done. All study patients were seen by a consultant Paediatric Neurologist of the unit. Seizures were classified using the International League Against Epilepsy (ILAE) classification of epileptic seizure. Interictal EEG recordings obtained with a 19 channel Electroencephalograph, employing scalp electrodes placed according to the international 10-20 system were studied. Patients who had EEGs done from the same institute were only considered and EEGs were interpreted by 2 Pediatric Neurologists expert in EEG interpretation. Although many of the children had multiple EEGs, information obtained from the first EEG was used. EEG findings were grouped into two main categories: `normal' for the age and state of the child and `abnormal'. Abnormal EEG was defined as the presence of interictal epileptiform discharges and/or the presence of background abnormal activity. Specific epileptiform abnormalities like-focal spikes, multifocal spikes, generalized spikes and waves, frequent sharp wave/ spike, hypsarrhythmia as well as background abnormalities were coded separately. Serum drug level was not measured. An antiepileptic drug was considered to have failed if it did not control seizures in spite of good compliance or if medication was discontinued because of unacceptable side effects. A drug used acutely to treat status epilepticus was not counted as one of the two AEDs. Child was a case or a control. Group1 consisted of poorly controlled epilepsy patients and Group2 comprised well-controlled epilepsy patients. A comparison of various EEG and clinical factors between the two groups was done. Data were analyzed using statistical package SPSS (version 15.0). Standard tests of significance, such as Chi-square test was applied for categorical variables and't' test was done for quantitative variables. The Odds ratio (OR) was used as estimation of risk to indicate the magnitude of association between each factor and poorly-controlled epilepsy. 95% confidence interval (CI) and p values were also computed. P-value of <0.05 was considered significant. Multivariate analysis was performed to choose independently significant factors among the many significant factors in bivariate analysis.

Results:

Table I shows that 54% of the poorly-controlled epilepsy patients and 44% of well controlled epilepsy patients were between 1-5 years age group. However there was wide spread distribution in different age groups. The mean age was found to be 4.59 ± 3.19 years in poorly-controlled group and 5.91 ± 3.72 years in well- controlled group. No significant statistical difference was observed between poorly-controlled and well-controlled group in respect to age ($p=0.062$). In poorly-controlled group 60.0% were males and 40.0% were females, while in well-controlled group males and females were 66.0% and 34.0% respectively. There was no significant statistical difference between the two groups in respect to sex ($p=0.534$).

Table-I: Distribution of the subjects by demographic variables (n=100).

Variables	Groups		p value
	Group1 (n=50) No (%)	Group2 (n=50) No (%)	
Age (year)			
○ ≤1year	5 (10.0)	1 (2.0)	
○ 1-5 years	27 (54.0)	22 (44.0)	
○ 5-10 years	16 (32.0)	20 (40.0)	
○ >10 years	2 (4.0)	7 (14.0)	
Mean ± SD	4.59 ± 3.19	5.91 ± 3.72	0.062[NS(a)]
Sex			
○ Female	20 (40.0)	17 (34.0)	
○ Male	30 (60.0)	33 (66.0)	0.534[NS(b)]

aUnpaired t test was done to measure the level of significance.
bChi-square test was done to measure the level of significance.

Table II shows that 96.0% of the patients in poorly-controlled epilepsy group had abnormal EEG in contrast to 82.0% of the patients in well-controlled epilepsy group (p<0.05)

Table II: Distribution of the subjects by initial EEG status (n=100).

EEG	Groups		OR (CI)	p value
	Group1(n=50) No (%)	Group2(n=50) No (%)		
Normal	2 (4.0)	9 (18.0)	5.27(1.08-25.78)	0.025*
Abnormal	48 (96.0)	41 (82.0)		

Chi-square test was done to measure the level of significance. ***p<0.05**

This figure shows that in poorly controlled group location of spikes/ sharp waves was predominantly generalized (38.0%) followed by multifocal (20%) and focal (20%), focal with secondary generalization being least common (6.0%). On the other hand in well-controlled group location of spikes/ sharp waves is predominantly focal (32.0%) followed by generalized (22.0%), multifocal (12%) and focal with secondary generalization (4.0%). But no significant statistical difference was noticed between the two groups in respect to location of spikes/sharp waves (p>0.05).

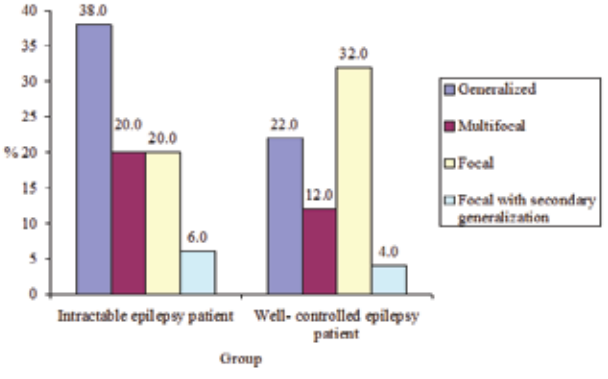

Figure-1: Multiple bar diagram showing location of spikes/sharp waves of EEG in two groups (n=100).

Table III demonstrates 64.0% of the patients in poorly-controlled group had frequent sharp waves/ spikes (>1/60 sec) compared to 24.0% in well-controlled group (p<0.001). Analysis also shows that hypsarrhythmia was found to be present in 16.0% of the patients in poorly-controlled group as compared to only 4.0% of the patients in well-controlled group(p<0.05).

Table-III: Distribution of the subjects by frequent sharp wave /spike (>1/60 sec) and hypsarrhythmia (n=100).

Variables	Groups		OR (CI)	p value
	Group1 (n=50) No (%)	Group2 (n=50) No (%)		
Frequent sharp wave /spike	32 (64.0)	12 (24.0)	5.63 (2.38-13.32)	<0.001**
Hypsarrhythmia	8 (16.0)	2 (4.0)	4.57 (1.03-19.98)	0.046*

Chi-square test was done to measure the level of significance.

Table IV demonstrates that no significant difference was noticed between the two groups in the development of poor seizure control in respect to the location of focal spikes/-waves (p>0.05). In case of multifocal spikes/waves involvement of fronto-temporal (8.0%) and tempo-parietal lobes (6.0%) were more in poorly-controlled group in comparison to well-controlled group in which case involvement of these lobes were only 2% respectively. But no significant difference was noticed between the two groups in the development of poor seizure control in respect to the location of multifocal spikes/waves (p>0.05).

Table-IV: Distribution of the subjects by location of the spike/sharp wave in different lobe/lobes of cerebral hemispheres (n =100).

Location of spike/sharp wave in lobe/lobes of cerebral hemispheres	Groups		OR (CI)	p value
	Group1 (n=50) No (%)	Group2 (n=50) No (%)		
If focal				
○ Frontal	6 (12.0)	2 (4.0)	3.27 (0.71-14.86)	0.269[NS]
○ Temporal	2 (4.0)	5 (10.0)	0.38 (0.08-1.78)	0.436[NS]
○ Parietal	1 (2.0)	2 (4.0)	0.49 (0.06-3.89)	0.999[NS]
○ Occipital	0 (0.0)	4 (8.0)		0.117[NS]
○ Central	3 (6.0)	3 (6.0)		0.999[NS]
If multifocal				
○ Fronto-temporal	4 (8.0)	1 (2.0)	4.26 (0.61-29.18)	0.362[NS]
○ Temporo-parietal	3 (6.0)	1 (2.0)	3.13 (0.43-22.46)	0.617[NS]
○ Centrotemporal	1 (2.0)	4 (8.0)	0.234 (0.03-1.64)	0.362[NS]

Fisher Exact test was done to measure the level of significance.

Table V demonstrates that 78% of the patients in Poorly-controlled group had background EEG abnormality compared to 30% of the patients in well-controlled group (p<0.001). Diffuse background slowing was present in 18.0% of the patients of poorly-controlled epilepsy compared to 4.0% in well-controlled patients (p=0.025). Focal slowing was present in 16.0% cases of well-controlled group compared to 8.0% in poorly controlled group. But no significant difference was noticed between the two groups.

Table-V: Distribution of the subjects by EEG background findings (n=100).

EEG background	Groups		OR (CI)	p value
	Group1 (n=50) No (%)	Group2(n=50) No (%)		
Normal	11 (22.0)	35 (70.0)	8.27 (3.36-20.39)	<0.001**
Abnormal	39 (78.0)	15 (30.0)		
Slowing				
○ Focal	4 (8.0)	8 (16.0)	0.46 (0.13-1.63)	0.218^{NS}
○ Diffuse	9 (18.0)	2 (4.0)	5.27 (1.20-22.73)	0.025*

Chi-square test was done to measure the level of significance.***p<0.05**

Table VI demonstrates logistic regression analysis for the EEG predictors of poorly-controlled epilepsy. Out of the 3 variables, abnormal EEG background (OR = 11.12; 95% CI=3.66-33.74) and frequent sharp wave/ spike (OR=8.06; 95% CI=2.67-24.31) were found to be the independent predictors of poorly-controlled epilepsy.

Table-VI: Logistic regression analysis for electroencephalographic predictors of Poorly-controlled childhood epilepsy.

Predictors	Model I	Model II	Model III
	Unadj. OR (CI)	Adj. OR (CI)	Adj. OR (CI)
Abnormal EEG background	8.27* (3.36-20.39)	11.62* (3.90-34.58)	11.12* (3.66-33.74)
Frequent sharp wave/ spike		8.29* (2.77-24.80)	8.06* (2.67-24.31)
Hypsarrhythmia			1.42 (0.24-8.60)

*Significant predictors at 5% level of significance. Absent was the reference category.

Discussion:

This study was done to search the elctroencephalographic factors that are associated with the poor control of childhood epilepsy. While there had been a number of recent studies designed to evaluate predictors of medical intractability in children, most have concentrated on clinical factors. This retrospective study demonstrated that there were a number of electroencephalographic factors that were associated with poor seizure control. Early identification of these factors might help in planning early intervention.

Table I illustrates the demographic characteristics of the patients. Age (mean± SD) at presentation of poorly controlled group was 4.59 ± 3.19 years and that of well-controlled group was 5.91 ± 3.72 years. No significant statistical difference was observed between poorly controlled and well-controlled group in respect to age (p=0.534). In poorly controlled group males were (60.0%) and females were (40.0 %) and in well-controlled group males and females were 66.0% and 34.0% respectively. In both groups males were predominant. No significant statistical difference was observed in this study in respect to sex between poorly controlled and well-controlled group (p=0.062) that is poor seizure control occurs with equal frequency in both sexes.

This finding was supported by the study done by Kwan P et al.[15] who did not find any significant difference in sex between the groups that become seizure free and the group with uncontrolled seizure. They found that 52% of the patients with uncontrolled epilepsy were male and 47% were female, whereas 47% male and 53% female were found in patients who were seizure free. But Malik et al.[16] found that male gender was a risk factor for intractable seizures (p=0.001) and this finding was similar to that previously done by Akhondian et al.[10] The male predominance in this study was more likely to be because of a selection bias.

Table II shows that 96.0% of the patients in poorly controlled group have abnormal EEG in contrast to 82.0% of patients in well-controlled group. EEG abnormality was significantly associated with poor seizure control of childhood epilepsy (p= 0.025). This finding was similar to those done by many authors.Singhviet al.[9] found that EEG was abnormal more in poorly controlled epilepsy patients (69% cases). Banuet al.[8] found that EEG was abnormal in 80.8% cases and it was found to be associated with more than 4 times (OR= 4.09) risk to poor seizure remission (p= 0.0016). Similar finding was seen by Akhondianet al.[10] who found that the first EEG was abnormal in 96.1% of patients in the poorly controlled epilepsy group and in 83.8% of the patients in the well-controlled group (p=0.031). Das et al.[17] found that an abnormal EEG was a significant factor for recurrence (75% versus 16% in cases with normal EEG). Another study carried out by Malik et al.[16] found that higher proportion of the patients with abnormal EEG continued to have seizures during the study period compared to patients with normal EEG (63% vs. 19%; OR= 7.28; 95% CI= 4.34-12.18; p-value<0.001).

The figure in this study shows that generalized location of spikes/ sharp waves is higher in poorly controlled group (38.0%) compared to well-controlled group (22.0%). In well- controlled group focal spikes/ sharp waves was found to be higher (32.0%). This finding was supported by the study done by Shafer et al.[14] who found that the absence of generalized epileptiform activity was associated with epilepsy remission, defined as 5 years without any seizure. The authors also found that focal epileptiform activity on the first EEG was associated with a good prognosis, perhaps reflecting a high percentage of patients with benign Rolandic epilepsy. But the findings by Ko and Holmes[13] shows that focal spikes and wave discharges were more common in the medically intractable group. Study by Singhviet al.[9] also noticed that focal epileptiform discharges were commoner (36%) than generalized discharges (21%). In this study multifocal spikes/ sharp waves were more common in poorly controlled epilepsy group than well controlled group (20% vs. 12%). But it was not statistically significant (p>0.05). This is similar to the finding by Ko and Holmes[13] who found that children with poorly controlled epilepsy group were more likely to have multifocal sharp waves and spikes than those with well controlled seizures (45.8% vs. 30.8%) but failed to show statistical significance.

This study showed that in case of focal spikes/waves, frontal lobe involvement was higher (12.0%) in poorly controlled group compared to well-controlled group (4.0%) (Table IV). Okuma & Kumashiro[11] also found that patients with anterior temporal or frontal focus tended to show lower remission rate of seizure than those with other topography of paroxysmal activity (p=<0.01). In case of multifocal spikes/waves we found that involvement of fronto-temporal (8.0%) and tempo-parietal lobes (6.0%) were more in poorly controlled group. This was similar to the finding by Singhviet al.[9] who found that frontotemporal lobe involvement was in 10% cases followed by temporal (8%) and frontal (6%) lobe. Ohtsukaet al.[18] also described that the rate of poorly controlled epilepsy patients was high in frontal lobe epilepsy and multi lobar epilepsy.

Frequent sharp wave /spike (>1/60 sec) was found to be a significant predictor for poorly controlled epilepsy (table III). In multivariate analysis it was found to be an independent predictor of poor seizure control (Table VI). Ko and Holmes[13] found that 52.1% of children with poorly controlled epilepsy had frequent sharp wave /spike (>1/60s) compared to 23.1% of children with well-controlled epilepsy (p<0.01). Hypsarrhythmia was found to be significantly associated with poor seizure control (p=0.046) but it was no longer significant in multivariate analysis (Table VI). Boyd &Harden[21] described that hypsarrhythmia may develop as a result of an early cerebral insult, whether focal or multifocal, such as infarction, porencephalic cysts, microangiopathy or in infants with developmental anomalies. They also described that whatever the clinical picture, the presence of hypsarrhythmia was an indicator of poor prognosis.

Among the EEG abnormalities EEG background abnormality was the most common abnormality noticed in the poorly controlled group (78.0%) (Table V). It was significantly associated with poorly controlled epilepsy (p=0.001). In multivariate analysis EEG background abnormality was found to be an independent predictor of poor seizure control (Table VI). This finding was supported by several studies. Ko and Holmes[13] found that abnormal EEG background was present in 73.6% cases in poorly controlled epilepsy group. Banuet al.[8] found that abnormal background activity was present in 34.4% cases and was associated with poor seizure remission. Study done by Okuma & Kumashiro[11] also showed that the remission rate was lowest in patients with severely and diffusely abnormal EEG background activity (p<0.001). Study carried out by Laroiaet al.[19] showed that EEG background abnormalities strongly predicted the occurrence of electrographic seizures in neonates concomittently or in the subsequent 18-24 hrs of recording. Among background abnormalities this study found that diffuse slowing was associated with increased risk for poor seizure control (p=0.025). Ko and Holmes[13] showed that diffuse slowing was the most significant factor for poorly controlled epilepsy (p=0.01). But Berg et al.[12] showed that focal EEG slowing (p=0.02) was an independent predictor of poorly controlled epilepsy. Shinnaret al[20] also found that focal slowing was associated with a high risk of recurrence. In the present study, underlying causes were not studied or correlated with the EEG changes.

After multiple logistic regression of significant electroencephalographic factors in bivariate analysis, we found that EEG background abnormality and frequent sharp wave/ spike were significant independent predictors of poorly controlled epilepsy (Table VI).

Conclusion:

This study shows that there are a number of electroencephalographic factors that can be identified early in the course of childhood epilepsy that are predictive of poor seizure contro[l]. Knowledge of these factors will help us to discriminate our patients and pay more attention to those at risk of developing poorly controlled seizures.

Conflict of Interest: None.

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References:

1. Johnston MV. Seizures in childhood. In: Behrman RE, Kleigman RM and Jenson HB, editors. Nelson Text book of Paediatrics. 17th ed. Philadelphia: Saunders; 2004:1993-2009.

2. Holmes GL, Engel J Jr. Predicting medical intractability of epilepsy in children. How certain can we be? Neurology. 2001; 56:1430-1431.

https://doi.org/10.1212/WNL.56.11.1430

PMid:11402096

3. Chawla S, Aneja S, Kashyap R, Mallika V. Etiology and clinical predictors of intractable epilepsy .Pediatr Neurol. 2002; 27(3): 186-191.

https://doi.org/10.1016/S0887-8994(02)00416-2

4. Devinsky O. Patients with refractory seizures. The new England journal of Medicine.1999; 340(20): 1565-1570.

https://doi.org/10.1056/NEJM199905203402008

PMid:10332020

5. Camfield PR, Camfield CS, Gordon K, Dooley JM. If a first antiepileptic drug fails to control a childhood epilepsy what are the chances of success with the next drug? J Pediatr. 1997; 131: 821-824.

https://doi.org/10.1016/S0022-3476(97)70027-1

6. Annegers JP, Hausen WA, Elveback LR. Remission of Seizures and Relapse in patients with Epilepsy', Epilepsia. 1979; 20: 729-737.

https://doi.org/10.1111/j.1528-1157.1979.tb04857.x

PMid:499118

7. Holmes GL. Epilepsy in the developing brain: lessons from the la boratory and clinic. Epilepsia. 1997; 38: 12-30.

https://doi.org/10.1111/j.1528-1157.1997.tb01074.x

PMid:9024181

8. Banu SH, Khan NZ, Jahan A, Parveen M, Rahman N, Boyd SH, et al. Profile of childhood epilepsy in Bangladesh. Developmental Medicine and Child Neurology. 2003; 45: 477-482.

https://doi.org/10.1017/S0012162203000884

https://doi.org/10.1111/j.1469-8749.2003.tb00943.x

PMid:12828402

9. Singhvi JP, Sawhney IM, Lal V, Pathak A, Prabhakar, S. Profile of intractable epilepsy in a tertiary referral centre. Neurol India. 2000; 48(4): 351-356.

10. Akhondian J, Heydarian F, Jafari, SA. Predictive factors of pediatric intractable seizure. Arch Iranian Med. 2006; 9(3): 236-239.

11. Okuma T, Kumashiro H. Natural history and prognosis of epilepsy: Report of a multi-institutional study in Japan. Epilepsia. 1981; 22: 35-53.

https://doi.org/10.1111/j.1528-1157.1981.tb04331.x

PMid:7472299

12. Berg AT, Shinnar S, Levy SR, Testa FM, Smith-Rapaport S, Beckerman B. Early development of intractable epilepsy in children: a prospective study. Neurology. 2001; 56: 1445-1452.

https://doi.org/10.1212/WNL.56.11.1445

PMid:11402099

13. Ko TS, Holmes GL. EEG and clinical predictors of medically intractable childhood epilepsy. Clinical

Neurophysiology.1999; 110: 1245-1251.

https://doi.org/10.1016/S1388-2457(99)00068-1

14. Shafer SQ, Hauser WA, Anneger JF, Klass DW. EEG and other early predictors of epilepsy remission: a community study. Epilepsia. 1988; 29(5): 590-600.

https://doi.org/10.1111/j.1528-1157.1988.tb03767.x

PMid:3409845

15. Kwan P, Brodie MJ. Early identification of refractory epilepsy. N Eng J Med. 2000; 342: 314 - 319.

https://doi.org/10.1056/NEJM200002033420503

PMid:10660394

16. Malik MA, Hamid MH, Ahmed TM, Ali Q. Predictors of intractable childhood epilepsy. Journal of the College of Physicians and Surgeons Pakistan. 2008;18(3):158-162.

17. Das CP, Sawhney IMS, Lal V, Prabhakar S. Risk of recurrence of seizures following single unprovoked idiopathic seizure. Neurol India. 2000; 48: 357-360.

18. Ohtsuka Y, Yoshinaga H, Kobayashi K, Murakimi N, Yamatogi Y, Oka E, et al. Predictors and underlying causes of medically intractable localized related epilepsy in childhood. Pediatric neurology. 2001; 27:181- 191.

19. Laroia N, Guillet R, Burchfiel J, Mc Bride, MC. EEG background as Predictor of Electrographic Seizures in High Risk Neonates. Epilepsia. 2006; 39(5): 545-551.

https://doi.org/10.1111/j.1528-1157.1998.tb01418.x

PMid:9596208

20. Shinnar S, Berg AT, Moshe SL, O'Dell C, Alemany M, Newstein D, et al. The risk of seizure recurrence after a first unprovoked afebrile seizure in childhood: an extended follow-up. Pediatrics.1996; 98: 216-225.

21. Boyd SG, Harden A. Clinical neurophysiology of the central nervous system. In: Brett EM, editor. Paediatric Neurology. 3rd ed. New York: Churchill Livingstone;1997: 747-821.

DOI: https://doi.org/10.3329/medtoday.v32i1.44823

ORIGINAL ARTICLE OPEN ACCSESS

Clinical Profile and Underlying Cause of Acute Pancreatitis among a Group of Bangladeshi Patients

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Ali Monsur Md. Shariful Islam[4], Arifa Akhter[5], Md. Mamun-ur Rashid[6]

Abstract

Introduction: *Acute Pancreatitis is a medical emergency, which is one of the most common conditions for hospital admission. Very few studies have yet investigated in Bangladesh. Objective of this study was to define demographic characteristics, clinical profile & underlying etiologies.* ***Materials and Methods:*** *This hospital based cross sectional descriptive study was performed in Sir Salimullah Medical College Mitford Hospital, Dhaka & North East Medical College, Sylhet, Bangladesh from January 2015 to December 2017. One hundred and five consecutive patients of acute pancreatitis (AP) were enrolled for this study. Clinical features and investigations were systematically recorded. Diagnosis of acute pancreatitis was made by the presence of the two of the three following criteria: i. abdominal pain consistent with the disease ii. serum amylase and /or lipase greater than three times from the upper limit of normal, and/or iii. characteristic findings of abdominal imaging.* ***Results:*** *Total of 105 patients took part in the study, of them 65 were male. The mean age was 42.76 ±15.88. Abdominal pain & vomiting was the most common mode of presentation. Gall stone and hypertriglyceridaemia were responsible of 20% of acute pancreatitis. Ascariasis also causes acute pancreatitis in two patients.* ***Conclusion:*** *Acute pancreatitis is a condition associated with high morbidity and mortality. Ascariasis also causes acute pancreatitis in endemic area. Patients usually respond conservative treatment but endoscopic treatment is effective in few cases. Surgery is rarely required.*

Keywords: *Pancreatitis, Gallstone, Serum lipase.*

Number of Tables: 03; Number of References: 13; Number of Correspondence: 07.

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Introduction:

Acute Pancreatitis is a potentially serious condition with an overall mortality of 10%. But more than 80% cases are mild & have a good outcome[1]. The incidence rate of acute pancreatitis seems to be increasing without any changes in case fatality rates[2]. The population is increasingly over weight. So the incidence of gallstones is also rising in our society.This is the most common cause of acute pancreatitis[3].

Early diagnosis is important for its management. Symptoms of acute pancreatitis vary considerably. So clinician must carefully evaluate the patient with history, physical examination, laboratory tests & imaging studies before arriving a correct diagnosis. If the cause of the attack can be eliminated there will be no further attack and the pancreas will return to normal in terms of its morphology and function[4]. Mortality associated with acute pancreatitis has decreased overtime. Death is more likely in certain subgroups of patients including elderly, co-morbidity, severe coexisting hospital acquired infection & organ dysfunction[5].

The main objective of this study was to observe the clinical presentation, underlying cause and outcome among hospital admitted patients in acute pancreatitis.

Materials & Methods:

This cross sectional descriptive study was carried out in the Department of Gastroenterology, Sir Salimullah Medical College Mitford Hospital, Dhaka & North East Medical College, Sylhet, Bangladesh from January 2015 to December 2017. Patients attending at indoor & outpatient department of gastroenterology with acute pancreatitis were the study population. Informed written consent was taken from the patient or guardian before the study. Data were

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recorded in a predesigned form.

Inclusion criteria were: Patients with age 18 years or more diagnosed as acute pancreatitis were included in this study.

Exclusion criteria were: Patient with chronic pancreatitis & pancreatic malignancy, patient having liver disease, pulmonary or cardiac disease or other serious co morbid condition were excluded from this study. Diagnosis of acute pancreatitis was made by the presence of the two of the three following criteria: i. abdominal pain consistent with the disease ii. serum amylase and/or lipase greater than three times from the upper limit of normal, and/or iii.characteristic findings of abdominal imaging[6]. Patients were classified as mild, moderate & severe acute pancreatitis based on Atlanta Revision Criteria (2013). Complete blood count, liver function test, renal function test, serum amylase, serum lipase, fasting blood sugar, lipid profile, serum calcium, CRP, chest X-ray & ultrasonography of whole abdomen were done in all patients. Computed tomography (CT) of abdomen was done when indicated & CT severity index was calculated. Severe acute pancreatitis was defined as patients having persistent two or more organ failure for more than 48 hours e.g. Systolic blood pressure ≤ 90 mm Hg or PaO2≤ 60%, renal failure (S.creatinine ≥2mg/dl after rehydration) & gastrointestinal bleeding > 500ml of blood loss/24 hrs[5]. Patients with severe acute pancreatitis were managed in ICU. Mild & moderate acute pancreatitis managed in ward. Surgery was done in patients who did not improve on intensive medical management.

All data were collected and analyzed by SPSS-22. p value < 0.05 was accepted as significant. The summarized data were interpreted accordingly and was then presented in the form of tables. Categorical data were tested with Chi-square test and continuous data were tested with unpaired t-test.

Results:

A total of 105 acute pancreatitis patients were enrolled in this study. Of the 105 cases, 65(61.9%) were male. Age of the patient ranged from 21-60 years. Incidence of acute pancreatitis below 50 years was significantly higher ($p<0.01$). Among them 58 (55.2%) hailing from rural area in our study & most were belongs to lower middle socioeconomic condition 55(52.4%). Detail demographic, clinical profile of study population is shown in Table-I. The most common presentation were upper abdominal pain (100%) & vomiting (77%). Among the patient 63(60%) patient was suffering from fever. Ascites & pleural effusion were present in 04(3.8%) & 03(3%) respectively. Ascites disappeared in patient before discharge from the hospital. Leukocytosis was present in 54 patients. All patients undergone sonographic evaluation. Swollen/enlarged pancreas revealed in nineteen patients. Cholelithiasis detected eight patients.

Sludge/Microlithiasis detected in gallbladder in 05 patients. Choledocholithiasis was not detected but dilated common bile duct was seen in 04 patients. Endoscopic examination was done in 74 admitted patients. Only 08 patients found positive findings. Only 02 patients were expired due to multiple organ dysfunction syndrome (MODS). In this study gallstone was associated with 17 patients. Hypertriglyceridemia was found 04 cases, biliary ascariasis found in 02 patients & alcoholism was found 01cases. No underlying cause was found in 81 patients.

Table-I: Showing demographic and clinical findings of Acute pancreatitis (n=105).

Variables		Frequency	Percent
Age group			
	≤50	69	65.7
	>50	36	34.3
	Mean±SD	42.76±15.88	
	P value	0.001	
Sex			
	Female	40	38.1
	Male	65	61.9
	P value	0.015	
Residence			
	Rural	58	55.2
	Urban	47	44.8
	P value	0.143	
Occupation			
	Service	12	11.4
	Business	25	23.8
	Student	12	11.4
	Housewife	35	33.3
	Unemployed	5	4.8
	Others	16	15.2
Economical status			
	Poor	4	3.8
	Lower middle class	55	52.4
	Higher middle class	34	32.4
	Rich	12	11.4
Clinical presentation	Abdominal Pain	105	100
	Vomiting	81	77
	Fever	63	60
	Abdominal tenderness	28	26.7
	Jaundice	4	3.8
	Ascites	4	3.8
	DM	34	32.4
	HTN	30	28.6
	Smoking	29	27.6
	Alcohol	1	

Table-II: Endoscopic finding of the patient

Biliary ascariasis	02
Duodenal ulcer	02
Gastric ulcer	02
Gastritis	02

Table-III: Etiology of the patient

Gall stone/sludge	17
Hypertriglyceridemia	04
Biliary ascariasis	02
Alcohol	01
No cause	81

Discussion:

This study was conducted to find out the etiological pattern, presentation & demographic of sufferers of acute pancreatitis in Bangladesh. All consecutive patients of acute pancreatitis visited in hospital were included in this study. Out of 105 number of patients male were more, similar observation has also been reported by Ahmed et al[7]. This may be due to male predominance in our OPD & female attend hospital in more severe stage of disease. Mean age was found 42.76 ± 15.88 yrs (Table-I). More than 47% patient was below 40 years of age. One study of our country peak incidence was found in the fourth decade.[7] in alcoholic pancreatitis peak incidence is in the fifth decade in western countries. Whereas pancreatitis associated with gallstones & other causes peak in the 7th decade. In our country the lower age incidence may be due to lower life expectancy of Bangladeshi people[7].

Upper abdominal pain (100%) radiates to the back & nausea, vomiting (77%) were the most common clinical presentation (Table-I), also reported by a study[7]. Fever was present in 63% patient which was due to inflammatory process & some cases due to viral infection(in younger age group). Abdominal tenderness was the most common sign in our study which was also reported in another Bangladeshi study[8]. Ascites is a well-known complication of acute pancreatitis. Ascites was found in severe pancreatitis 18% to 60%[9, 10]. Pleural effusion was found in 2.8% cases, which was low in incidence than that of others study[11, 12].

We tried to find out the underlying cause of acute pancreatitis. In the present study gall stone was found 16.19% cases (Table-III). One study in Bangladesh shows gall stone causes 18% acute pancreatitis[7]. More than two third cases were idiopathic. Extensive investigation is needed to determine the cause. In many studies show that alcohol is the leading cause of acute pancreatitis, but socio-cultural & religious factor discourage alcohol consumption in our society.

Hypertriglyceridemia was responsible in four cases in our study which was consistent with others[7,8]. Triglyceride level more than 1000mg/dl increases the risk of pancreatitis. All patient of this study underwent ultrasonographic investigation. In sixty nine (65.71 %) patients pancreas appeared to be normal which may be due to poor visualization of the organ. CT scan was done in 23 (21.9%) patients who had severe acute pancreatitis having normal sonographic findings. In CT, we found seven additional pancreatic findings. Upper GIT Endoscopy was done in 15 patient of them biliary ascariasis was found in 2 cases.

The overall mortality rate was 1.9%. Mortality of severe acute pancreatitis was higher. Death was mostly due to multi organ dysfunction syndrome (MODS) in elderly patient. However, Keya et al showed higher mortality 13.6%[11,12,13]. For these type of patient intensive care unit (ICU) is necessary in tertiary care hospital. But ICU bed is inadequate in regional tertiary care centre.

Conclusion:

Acute pancreatitis is a condition associated with high morbidity and mortality. Ascariasis also causes acute pancreatitis in endemic area. Patients usually respond conservative treatment but endoscopic treatment is effective in few cases. Surgery is rarely required. Further study with large sample size; multicenter, prospective randomized controlled study is needed to determine the underlying etiology & prognostic criteria in our population.

Conflict of Interest: None.

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References:

1. Omar E EL, McLean MH. Gastroenterology. Raltson SH, Penman ID, Stachan MWJ, Hobson RP. Davidson's Principles and Practice of Medicine. 23th edition. Elsevier. 2018; 837-39.

2. Conwell DL, Banks PA. Acute pancreatitis. Greenberger NJ, Blumberg RS. Current diagnosis & treatment Gastroenterology, Hepatology& Endoscopy. 3rd edition. McGraw-Hill Education. 2016; 335-42.

3. Tenner S, Steinberg WM. Acute Pancreatitis. Feldman M, Friedman LS, Brandt LJ. Sleisenger and fordtran's gastrointestinal and liver disease: pathophysiology/ diagnosis/management. 9th ed. Philadelphia: Elsevier; 2010; 959-82.

https://doi.org/10.1016/B978-1-4160-6189-2.00058-5

4. Norton J, Berger G, Philip P. Acute and Chronic pancreatitis. Harrison's Principles of Internal Medicine. Newyork McGraw Hill. 19th edition. 2015; 2097-2114.

5. Edward W. Campion M. Acute Pancreatitis, Review article. Forsmark CE, Vege SS, Wilcox CM. N Engl J Med. 2016; 375:1972-81.

https://doi.org/10.1056/NEJMra1505202

PMid:27959604

6. Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology Guideline: Management of Acute Pancreatitis. Am J Gastroenterol. 2013; 108(9):1400-15; 1416.

7. Ahmed KU, Ahad MA, Alim MA, Ekram ARMS. Clinical profile of acute pancreatitis in a teaching hospital. Bang Med J Khulna. 2016; 49:7-12.

https://doi.org/10.3329/bmjk.v49i1-2.31818

8. Datta IK, Haque MN, Bhuiyan TM. Clinical profile, degree of severity and underlying factor of acute pancreatitis among a group of Bangladeshi patients. IMC J Med Sci. 2018; 12(1): 06-10.

https://doi.org/10.3329/imcjms.v12i1.35170

9. Durenier T, Laterre PF, Reynaert Ms. Ascites fluid in severe acute pancreatitis: from pathophysiology to therapy. Acta Gastroenterol Belg. 2000; 63: 264-68.

10. Maringhini A, Ciambra M, Patti R, Randazzo MA, Dardononi G, et al. Ascites pleural and pericardial effusion in acute pancreatitis. A prospective study of incidence, natural history and prognostic role. Dig Dis Sci. 1996; 41:848-52.

https://doi.org/10.1007/BF02091521

PMid:8625753

11. Kaya E, Dervisoglu A, Polat C. Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis. World J Gastroenterol. 2007; 13(22): 3090-4.

https://doi.org/10.3748/wjg.v13.i22.3090

PMid:17589925 PMCid:PMC4172616

12. Beaux AC, Palmer KR, Carter DC. Factors influencing morbidity and mortality in acute pancreatitis; an analysis of 279 cases. Gut. 1995; 37:121-126.

https://doi.org/10.1136/gut.37.1.121

PMid:7672660 PMCid:PMC1382782

13. Chatzicostas C, Roussomoustakaki M, Vlachonikolis IG, Notas G, Mouzas I, Samonakis D, et al. Comparison of Ranson, APACHE II and APACHE III scoring systems in acute pancreatitis. Pancreas. 2002; 25: 331-335.

https://doi.org/10.1097/00006676-200211000-00002

PMid:12409825

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ORIGINAL ARTICLE

OPEN ACCSESS

Clinical, Pathological, Treatment Pattern and Post-management Follow-up Features of Differentiated Thyroid Carcinoma (DTC) in a Tertiary Level Health Care Center of Bangladesh

Parvez Ahmed[*1], Subrata Ghosh[2], Mostafa Shamim Ahsan[3], Nasrin Begum[4], Mosharruf Hossain[5], M.M. Arif Hosen[6]

Abstract

Introduction: *This study evaluates clinical, pathological, treatment pattern and post-management follow-up features of differentiated thyroid carcinoma affected patients who had attended Rajshahi Medical College Hospital as well as private clinics and then Institute of Nuclear Medicine & Allied Sciences, Rajshahi for primary surgical management and post-operative radioiodine ablation therapy respectively.* ***Materials and Methods****: It is a retrospective study. Clinico-pathological, treatment and post-management follow-up features of 254 patients of histologically proved differentiated thyroid carcinoma (DTC) were recorded from their ultrasonography report, pre-operative cytology, operation note, post-operative histopathology and radioiodine ablation therapy related follow-up book between 2011 and 2015 and analyzed using statistical software IBM SPSS v. 16.* ***Results:*** *Among the sample (n=254), 211 (83 %) were female and 43 (17 %) were male. Mean age group was 30-39 years. In relation to FNAC findings of thyroid nodules among this study sample (n=254), 223 (88%) had malignant cytology, 16 (6%) had borderline cytology and 15 (6%) had benign cytology. Regarding the histopathological findings of thyroid nodules, 241 (95 %) had papillary carcinoma and 13 (5 %) had follicular carcinoma. Regarding radioiodine ablation dose, 198 (78%) had been given 138 millicurie (5.1 gigabecquerel) and 56 (22%) had been given 178 millicurie (6.6 gigabecquerel).* ***Conclusion:*** *Early and successful management of differentiated thyroid carcinoma is required to practice widely and equitably in order to significantly reduce mortality and morbidity related to such conditions.*

Keywords: *Differentiated thyroid carcinoma (DTC), Clinical features, Pathological features, Treatment pattern, Post-management follow-up features.*

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Introduction:

Well-differentiated thyroid carcinoma that arises from the thyroid follicular epithelial cells is the most common endocrine neoplasia[1,2]. Among the well-differentiated thyroid carcinomas, the incidence of papillary thyroid carcinoma rose by a factor of three around the world from 1973 to 2009 and most of the increased incidence is traceable to more frequent diagnostic evaluation with improved technology-namely, more widespread and aggressive use of ultrasound and image-guided biopsy[3,4,5,6]. Regarding treatment, well-differentiated carcinomas of the thyroid are treated by surgery combined with radioactive iodine therapy[7]. In fact, various surgical techniques have been devised for tumor invasion confined to the neck depending on the site and longitudinal and transverse extent of tumor[8].

Materials and Methods:

It is a retrospective study. Clinico-pathological, treatment and post-management follow-up features of 254 patients of histologically proved differentiated thyroid carcinoma (DTC) who had attended Rajshahi Medical College Hospital and then Institute of Nuclear Medicine & Allied Sciences, Rajshahi for primary surgical management and post-operative radioiodine ablation therapy respectively were recorded from their ultrasonography report, pre-operative cytology, operation note, post-operative histopathology and radioiodine ablation therapy related follow-up book between 2011 and 2015. Their data were analyzed using statistical software IBM SPSS v. 16.

Results:

Among the sample (n=254), 211 (83 %) were female and 43 (17 %) were male (Figure-1).

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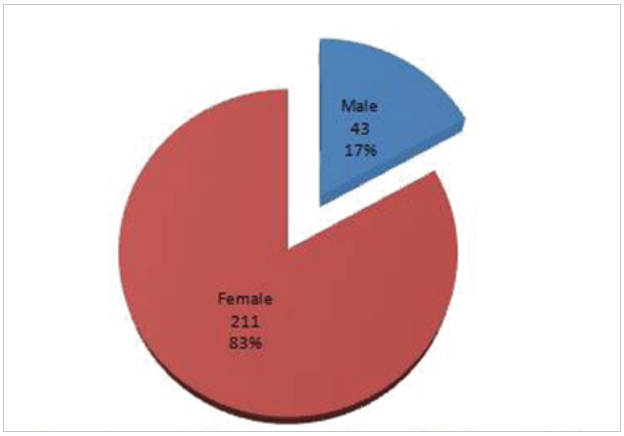

Figure-1: Distribution of gender (n=254).

Mean age group was 30-39 years (Figure-2).

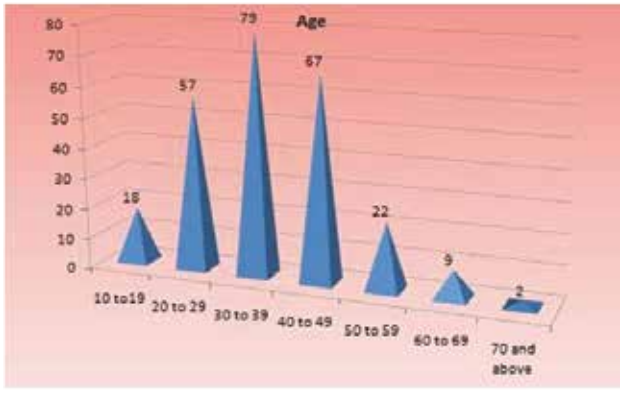

Figure-2: Distribution of age (n=254).

Regarding the USG findings of thyroid gland, 187 (74 %) had unilateral nodule and 67 (24 %) had bilateral nodules (Figure-3).

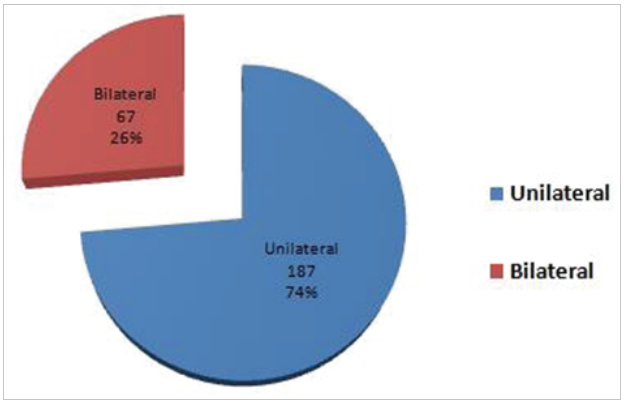

Figure-3: HRUS findings of thyroid nodules (n=254).

And, in relation to FNAC findings of thyroid nodules among this study sample (n=254), 223 (88%) had malignant cytology, 16 (6%) had borderline cytology and 15 (6%) had benign cytology (Figure-4).

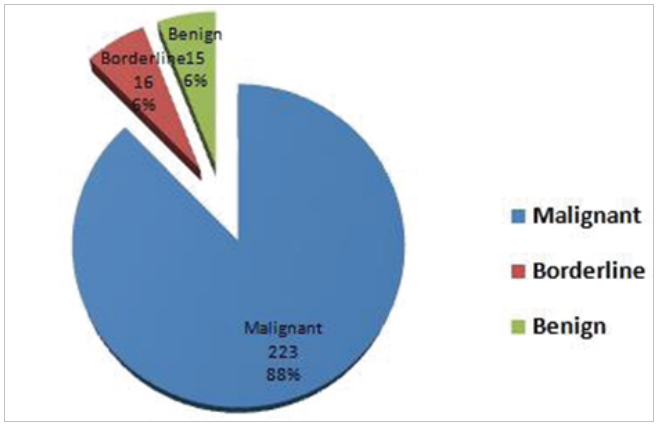

Figure-4: FNAC findings of thyroid nodules (n=254).

Regarding the histopathological findings of thyroid nodules, 241 (95 %) had papillary carcinoma and 13 (5 %) had follicular carcinoma (Figure-5).

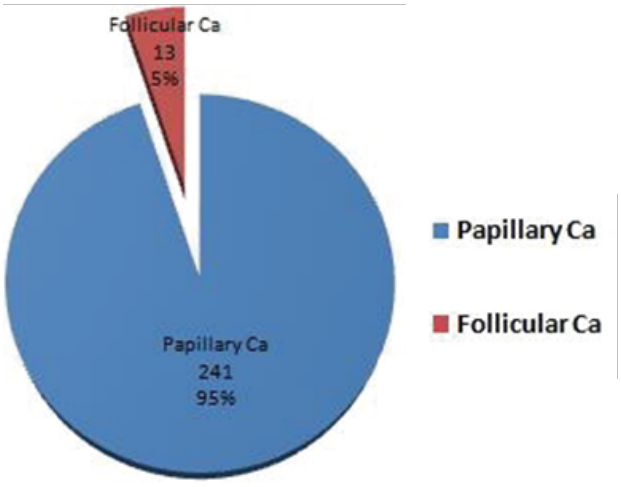

Figure-5: Histopathology findings of thyroid nodules (n=254).

In relation to surgical management, 185 (73%) had undergone total thyroidectomy with central clearance, 48 (19%) had undergone total thyroidectomy with selective neck dissection, 13 (5%) had undergone completion thyroidectomy and 8 (3%) had undergone total thyroidectomy with modified radical neck dissection (Figure-6).

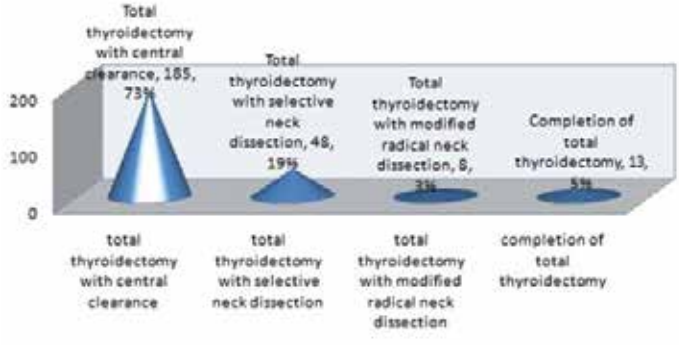

Figure-6: Types of undertaken neck surgery (n=254).

Regarding radioiodine ablation dose, 198 (78%) had been given 138 millicurie (5.1 gigabecquerel) and 56 (22%) had been given 178 millicurie (6.6 gigabecquerel) And, in relation to treatment outcome from 2011 to 2016 among the sample (n=254), 26 (10%) were treated in 2011 and had recurred once (in 2014), 49 (20%) were treated in 2012 and had recurred twice (once in 2015 and once in 2016), 46 (18%) were treated in 2013 and had recurred twice in 2016, 49 (19%) were treated in 2014 and had recurred once in 2016 and 84 (33%) were treated in 2015 and had no recurrence upto 2016.

Discussion:

Thyroid cancer is the most common endocrine malignancy. The most frequent histologic subtype of thyroid carcinoma is papillary (accounting for 80.2% of cases), followed by follicular carcinoma (11.4% of cases), which are commonly collectively referred to as well-differentiated thyroid cancer[9]. In the systematic investigation of thyroid nodules, risk stratification is performed on the basis of the physical, ultrasonographic, and scintigraphic findings, and the diagnosis is generally established by fine-needle biopsy and cytology[10].

Papillary thyroid microcarcinoma (PTMC) is a specific subgroup of papillary thyroid carcinoma (PTC) and defined by WHO on the largest dimension of 1.0 cm or less. Most of PTMC are not detectable at clinical examination and are diagnosed incidentally during pathologic examination of thyroid specimens after surgery for benign thyroid diseases, or in autopsies. Characteristic cytologic features of PTC help make the diagnosis by FNA or after surgical resection; these include psammoma bodies, cleaved nuclei with an "orphan-Annie" appearance caused by large nucleoli, and the formation of papillary structures. Moreover, the increased accuracy of the pathologic thyroid examination, in particular with the thinness and the number of the anatomical slices obtained for thyroid specimens, led to a more frequent pathologic; diagnosis of incidental PTMC. On the other hand, the widespread use and the technical improvement of thyroid ultrasonography and fine-needle aspiration biopsy (FNAB) contributed to an increase in the rate of preoperative diagnosis of PTMC over the last few decades[11].

Conclusion:

The incidence of thyroid cancer has increased dramatically during the past three decades and it is now the fastest growing cancer in women[12].Therefore, early and successful management of differentiated thyroid carcinoma is required to practice widely and equitably in order to significantly reduce mortality and morbidity related to such conditions. Through the findings of this study, such well-established management protocol has been highlighted at this tertiary level at Rajshahi, Bangladesh. However, large-scale relevant studies are required to evaluate ongoing management protocol so that global standard can be achieved.

Conflict of Interest: None.

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References:

1.Ceresini G, Corcione L, Michiara M. Thyroid cancer incidence by histological type and related variants in a mildly iodine-deficient area of Northern Italy, 1998 to 2009. Cancer. 2012; 118: 5473–5480. [PubMed]

https://www.ncbi.nlm.nih.gov/pubmed/22517468
DOI:10.1002/cncr.27591

2. Robert Koch-Institut und Gesellschaft der epidemiologischen Krebsregister in Deutschland e. V. (eds.): Krebs in Deutschland 2009/2010. 9. Ausgabe. Berlin, 2013.

https://www.aerzteblatt.de/int/archive/references?id=171082

DOI: 10.3238/arztebl.2015.0452

3. Morris LGT, Sikora AG, Tosteson TD, Davies L. The increasing incidence of thyroid cancer: The influence of access to care. Thyroid. 2013; 23: 885–891. [PMC free article] [PubMed]

https://www.ncbi.nlm.nih.gov/pubmed/23517343

DOI: 10.1089/thy.2013.0045

4. Tuttle RM, Ball DW, Byrd D. Thyroid carcinoma. J Natl Compr Canc Netw. 2010; 8: 1228–1274. [PubMed]

https://www.ncbi.nlm.nih.gov/pubmed/21081783

PMID: 25505208

5. Davies L, Welch HG. Increasing incidence of thyroid cancer in the United States, 1973-2002. JAMA. 2006; 295: 2164-2167.

https://www.ncbi.nlm.nih.gov/pubmed/16684987

DOI: 10.1001/jama.295.18.2164

6. Cooper DS, Doherty GM, Haugen BR. Management guidelines for patients with thyroid nodules and differentiated thyroid cancer. Thyroid. 2006 ;16:109-142.

https://www.ncbi.nlm.nih.gov/pubmed/16420177

DOI: 10.1089/thy.2006.16.109

7. Dralle H, Musholt TJ, Schabram J, et al. German Association of Endocrine Surgeons practice guideline for the surgical management of malignant thyroid tumors. Langenbecks Arch Surg. 2013;398:347–375. [PubMed]

https://www.ncbi.nlm.nih.gov/pubmed/23456424

DOI: 10.1007/s00423-013-1057-6

8. Lorenz K, Niederle B, Steinmüller T, Dralle H. The European Society of Endocrine Surgeons perspective of thyroid cancer surgery: an evidence-based approach. Langenbecks Arch Surg. 2014; 399:135–139. [PubMed]

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4515787/
DOI: 10.3238/arztebl.2015.0452

9. Annual Report to the Nation on the status of cancer, 1973-1999, featuring implications of age and aging on U.S. cancer burden

https://onlinelibrary.wiley.com/doi/full/10.1002/cncr.10593?sid=nlm%3Apubmed

DOI: 10.1002/cncr.10593

10. American Association of Clinical Endocrinologists, Associazione Medici Endocrinologi, and European Thyroid Association Medical guidelines for clini... - PubMed - NCBI

https://www.ncbi.nlm.nih.gov/pubmed/20551008

DOI: 10.4158/EP.16.3.468

11. Papillary thyroid microcarcinoma: extrathyroidal extension, lymph node metastases, and risk factors for recurrence in a high prevalence of goiter a... - PubMed - NCBI

https://www.ncbi.nlm.nih.gov/pubmed/20052467

DOI: 10.1007/s00268-009-0375-x

12. Bilimoria KY, Bentrem DJ, Ko CY. Extent of surgery affects survival for papillary thyroid cancer. Ann Surg. 2007; 246 :375–381. [PMC free article] [PubMed]

https://www.ncbi.nlm.nih.gov/pubmed/17717441

DOI: 10.1097/SLA.0b013e31814697d9

DOI: https://doi.org/10.3329/medtoday.v32i1.44825

ORIGINAL ARTICLE OPEN ACCSESS

Comparison of Haemodynamic Effects of Levobupivacaine and Bupivacaine in Sub-arachnoid Block for Total Abdominal Hysterectomy

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Abstract

Introduction: *Altered haemodynamics and arterial hypotension are the most prevalent untoward effects of sub-arachnoid block with bupivacaine. Use of levobupivacaine as pure S (-) - enantiomer of bupivacaine has progressively increased due to its lower cardiotoxicity, neurotoxicity and shorter duration of motor block. This study aimed to compare the haemodynamic status of levobupivacaine and bupivacaine when used with fentanyl in Sub-arachnoid block (SAB) among patients undergone total abdominal hysterectomy.* ***Materials and Methods:*** *This randomized clinical trial enrolled 80 gynaecological patients scheduled for total abdominal hysterectomy under SAB. Forty patients were randomly assigned as trial group (levobupivacaine+fentanyl) and forty as control group (bupivacaine+fentanyl). Main outcome measures in both groups considered intraoperative haemodynamic stability (acute hypotension), cardiotoxicity (bradycardia or tachycardia) and time of administration of first dose of postoperative analgesics.* ***Results:*** *Bupivacaine caused comparatively more significant slowing of heart rate at 25 and 35 minutes of operation. Though no significant differences were found in systolic blood pressure between two groups, bupivacaine caused significant reduction of diastolic blood pressure in comparison to levobupivacaine at 6, 9, 45 minutes and at the end of surgery. Mean pressure found significantly lowered at 6 minutes and at the end of surgery following bupivacaine administration. The incidence of acute hypotension was significantly low in trial group and postoperative analgesia was maintained longer duration with levobupivacaine in control group.* ***Conclusion:*** *Levobupivacaine in comparison to bupivacaine showed more effective and satisfactory haemodynamic stability in sub-arachnoid block with less incidence of intraoperative acute hypotension and provides prolonged postoperative analgesic effect.*

Keywords: *Bupivacaine, Levobupivacaine, Sub-arachnoid block.*

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Introduction:

Sub-arachnoid block (SAB) or spinal anaesthesia is now-a-days a popular technique in gynaecological surgery including several advantages of rapid onset of profound anaesthesia, lower total drug dosages, and higher level of patient's satisfaction[1,2]. Level of sensorial blockade is one of the most important factors for successful anaesthesia and baricity is the most important one for local anaesthetic distribution[3]. Isobaric solutions remain in close proximity of injection site but hyperbaric solutions gravitate to the dependent areas. The duration of spinal anaesthesia is, therefore, related to dose and baricity of local anaesthetic administered[3].

Bupivacaine is an amide local anaesthetic of hydrophilic nature with moderately rapid onset and long duration of action. Levobupivacaine is isobaric, and formulated by using normal saline as a diluent with specific gravity of 1000 at 37°C whereas bupivacaine heavy in 8% glucose is hyperbaric, having specific gravity of 1021 at 37°C[4]. Hyperbaric bupivacaine attains higher sensory levels of intrathecal anaesthesia in comparison to equal doses of isobaric levobupivacaine[5]. Both isobaric levobupivacaine and hyperbaric bupivacaine have been used for sub-arachnoid block with good results[4].

Addition of opioids (fentanyl) with local anaesthetics for spinal anaesthesia is increasingly common both to enhance anaesthetic effect and to provide postoperative analgesia[6]. The dose of local anaesthetics can also be reduced by adding fentanyl. Small doses of opioids administered to the central nervous system not only provide adequate analgesia but also reduce the side effects of intravenous analgesics like pruritus, nausea and vomiting or respiratory depression[7].

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Cardiac toxicity; the most commonly encountered catastrophe, should be taken into immense consideration before selection of local anaesthetic agent. Hyperbaric bupivacaine solutions may cause hypotension or bradycardia after mobilization of anaesthetized patient but isobaric solutions are favored with respect to their less sensitivity to posture issues properties[8]. Levobupivacaine, being the S (-) - enantiomer of bupivacaine, is less cardiotoxic due to its lower affinity to sodium channel and also less neurotoxic with shorter duration of motor block than hyperbaric bupivacaine. Thus its use has been increasing progressively[9].

Though hyperbaric bupivacaine has satisfactory quality of analgesia following intrathecal administration, it regresses rapidly with more side effects. Isobaric levobupivacaine seems to provide a slow regression of analgesia with fewer undesirable haemodynamic effects except for its control of spread of analgesia[10]. But if the dosages as well as the speed of administration of isobaric solution are well adjusted, it is believed to be safe and reliable with an excellent level of analgesia for abdominal surgeries like hysterectomy.

This study was designed to compare the intraoperative haemodynamic effects of levobupivacaine and bupivacaine in spinal anaesthesia among patients undergone total abdominal hysterectomy. The study also compared postoperative analgesic effect by documenting the time of administration of first dose of analgesics in postoperative period.

Materials and Methods:

This randomized clinical trial was conducted in the Department of Anesthesiology of SSMC & Mitford Hospital, Dhaka, from January 2015 to June 2015. Eighty gynaecological patients, aged 30-65 years with physical status-I & II according to American Society of Anesthesiologists (ASA) scheduled for routine total abdominal hysterectomy under sub-arachnoid block were enrolled for the study. Patients with significant cardiac, pulmonary, hepatic or renal diseases, abnormal coagulation profile, spinal deformities, evidences of skin infection at the site of injection, disabling neuropsychiatric disorders, known hypersensitivity to bupivacaine, levobupivacaine or fentanyl, chronic drug abusers and alcoholics were excluded from this study. Forty patients were randomly assigned for isobaric levobupivacaine designated as "trial group" and forty for hyperbaric bupivacaine designated as "control group". Baseline heart rates, systolic, diastolic and mean arterial blood pressures (MAP) were recorded preoperatively. Patients in "trial group" received 0.5% levobupivacaine 15 mg (3 ml) + 25 microgram fentanyl (0.5 ml) (total volume 3.5 ml) and "control group" received 0.5% bupivacaine heavy 15 mg (3 ml) + 25 microgram fentanyl (0.5 ml) (total volume 3.5 ml) slowly at a rate of 1 ml/5 sec in subarachnoid space at L_3-L_4 or L_4-L_5 level. Each patient was immediately turned to supine position. After intrathecal injection, heart rate, non-invasive systolic, diastolic and mean arterial blood pressures were recorded at every 3 minutes for 15 minutes, thereafter at every 10 minutes up to 45 minutes and at the end of surgery. The time when first dose of postoperative analgesic required was determined in each patient. Acute hypotension was defined as decrease in mean arterial blood pressure more than 20-30% from baseline, bradycardia as heart rate less than 45 beats/min and tachycardia as heart rate more than 100 beats/min. All data were recorded in a structured questionnaire by an anesthesiologist involved.

Results:

Mean age of study patients was 45.6 ± 7.1 and 44.9 ± 5.2 years in trial & control group respectively. Heart rate, systolic, diastolic and MAP were almost homogenously distributed between two groups at baseline with no significant differences (Table I). Bupivacaine caused comparatively more significant slowing of heart rate at 25 and 35 minutes (Table II). Though no significant ($p>0.05$) differences were found in systolic blood pressure between two groups, bupivacaine caused significant ($p<0.05$) reduction of diastolic blood pressure in comparison to levobupivacaine at 6, 9, 45 minutes and at the end of surgery (Table III). Mean arterial pressure also significantly ($p<0.05$) lowered at 6 minutes and at the end of surgery following bupivacaine administration (Table III). The incidence of acute hypotension was significantly ($p<0.05$) low in trial group than control group (27.5% vs. 50.0%). Postoperative analgesia was maintained longer duration with levobupivacaine in control group as time of postoperative 1st dose analgesic requirement was significantly later (Table IV).

Table-I: Baseline haemodynamic parameters of study population.

Baseline parameters	Trial group	Control group	p value
Heart rate (beats/min)	93.0 ± 17.1	95.2 ± 15.5	0.479
Systolic blood pressure (mmHg)	139.4 ± 20.4	134.1 ± 16.4	0.201
Diastolic blood pressure (mmHg)	84.6 ± 11.6	81.9 ± 11.4	0.306
Mean arterial pressure (mmHg)	100.1 ± 18.4	101.1 ± 15.1	0.786

Values expressed as Mean ± standard deviation, p value was obtained by Chi-square test.

Table-II: Comparison of heart rate at different time interval.

Heart rate (beats/min)	Trial group	Control group	p value
At 3 minutes	94.0 ± 22.0	92.0 ± 16.0	0.654
At 6 minutes	91.0 ± 21.0	85.0 ± 15.0	0.151
At 9 minutes	85.0 ± 19.0	90.0 ± 13.0	0.242
At 12 minutes	82.0 ± 17.0	78.0 ± 11.0	0.193
At 15 minutes	81.0 ± 15.0	76.0 ± 10.0	0.078
At 25 minutes	80.0 ± 14.0	73.0 ± 9.0	**0.013**
At 35 minutes	77.0 ± 13.0	71.0 ± 8.0	**0.032**
At 45 minutes	76.0 ± 14.0	73.0 ± 10.0	0.162
At the end of surgery	79.0 ± 15.0	76.0 ± 12.0	0.264

*Values expressed as Mean ± standard deviation, p value was obtained by Chi-square test.

Table-III: Comparison of systolic, diastolic and mean arterial pressure at different time interval.

Variables	Systolic BP		p value	Diastolic BP		p value	Mean pressure		p value
	Trial group	Control group		Trial group	Control group		Trial group	Control group	
At 3 minutes	120.6 ± 23.0	115.4 ± 15.8	0.241	73.8 ± 16.2	69.9 ± 13.1	0.231	87.2 ± 18.6	82.7 ± 13.0	0.203
At 6 minutes	116.2 ± 24.6	106.9 ± 16.1	0.052	69.5 ± 16.0	63.1 ± 13.9	**0.035**	85.0 ± 20.0	76.4 ± 14.1	**0.029**
At 9 minutes	110.3 ± 23.1	104.9 ± 16.0	0.230	67.0 ± 14.4	60.8 ± 12.8	**0.047**	79.1 ± 18.2	74.7 ± 14.5	0.235
At 12 minutes	108.1 ± 20.9	102.2 ± 14.8	0.145	64.6 ± 15.3	60.9 ± 12.6	0.239	77.9 ± 17.3	73.3 ± 14.3	0.199
At 15 minutes	106.9 ± 19.6	103.7 ± 17.8	0.437	66.1 ± 15.6	62.6 ± 12.9	0.271	76.2 ± 14.0	73.1 ± 14.9	0.342
At 25 minutes	100.4 ± 12.4	99.0 ± 15.5	0.668	59.6 ± 11.8	57.1 ± 12.0	0.342	70.7 ± 12.6	70.8 ± 13.9	0.993
At 35 minutes	98.0 ± 12.0	96.7 ± 13.1	0.698	60.0 ± 11.9	56.9 ± 11.4	0.282	69.2 ± 10.3	68.5 ± 12.3	0.776
At 45 minutes	99.4 ± 12.6	97.4 ± 12.5	0.484	61.9 ± 13.3	56.7 ± 9.8	**0.043**	72.3 ± 12.4	67.6 ± 10.8	0.072
At end of surgery	102.5 ± 15.0	100.3 ± 10.6	0.439	63.8 ± 11.5	58.6 ± 8.3	**0.022**	75.8 ± 13.3	70.8 ± 8.9	**0.050**

*Values expressed as Mean ± standard deviation, p value was obtained by Chi-square test.

Table-IV: Per-operative complications encountered and requirement of postoperative analgesic.

Variables*	Trial group (n = 40)	Control group (n = 40)	p value
Bradycardia (< 45 beats/min)	2 (5.0)	1 (2.5)	0.50†
Tachycardia (> 100 beats/min)	6 (15.0)	5 (12.5)	0.745†
Acute hypotension	11 (27.5)	20 (50.0)	0.039†
Time of postoperative analgesic requirement (min)	233.0 ± 20.0	161.4 ± 24.5	< 0.05‡

***Values :** Expressed as numbers (n) and percentages (%) in parentheses or Mean ± SD

†p value : Obtained by Chi-square test

‡p value : Obtained by Student's t-test

Discussion:

The goal of this study was to compare the intraoperative haemodynamic effects of levobupivacaine and bupivacaine when used with fentanyl in SAB for TAH. Forty patients were randomly assigned for levobupivacaine + fentanyl (trial group) and 40 for bupivacaine + fentanyl (control group). Intraoperative haemodynamic parameters including heart rate, systolic, diastolic and mean pressure were recorded at different time interval.

Following SAB, heart rate decreased insidiously from baseline to 35 minutes in both groups but bupivacaine caused comparatively more significant slowing of heart rate at 25 and 35 minutes. Fattorini et al. showed that levobupivacaine and bupivacaine decreased heart rate over 30 minutes after anesthesia in major orthopedic surgery without significant inter group differences[11]. Systolic and diastolic BP in both study groups exhibited a sharp fall at 3-6 minute of intervention. Thereafter the fall of systolic BP was insidious up to 35 minutes and became stable at the end of surgery without significant differences between study groups. Conversely, the fall of diastolic BP was slow and steady up to 12 minutes with another sharp fall at 25 minutes and stabilized at the end. Though in this study bupivacaine caused comparatively significant reduction of diastolic BP than levobupivacaine at 6, 9, 45 minutes and at the end of surgery, no significant inter group differences of systolic and diastolic BP were noted by Fattorini et al.[11] In another prospective observational study, Herrera et al. found a decrease ($p < 0.05$) in systolic and diastolic BP at 30 minutes intraoperatively[12]. This study also found significantly lowered mean arterial pressure (MAP) at 6 minutes and at the end of surgery following bupivacaine administration. But no inter group difference in MAP was observed by Fattorini et al.[11] Contrary to this study Guler et al. found bradycardia more common in bupivacaine fentanyl group. The incidence of acute hypotension was significantly ($p<0.05$) low in trial group (levobupivacaine) than control group (bupivacaine) (27.5% vs. 50.0%). This findings support several other studies[12,13]. Though bupivacaine heavy yields satisfactory quality of analgesia following

SAB, it regresses rapidly[10]. Postoperative analgesia was maintained longer duration with levobupivacaine in this study. Erdil et al. similarly showed isobaric levobupivacaine provides a slow regression of analgesia[10]. Guler et al. also concluded that the time of first analgesic requirement was earlier in group bupivacaine compared to levobupivacaine[13].

Conclusion:

Levobupivacaine when co-administered with fentanyl in SAB maintains intraoperative haemodynamic stability better than the bupivacaine-fentanyl combination. The incidence of acute intraoperative hypotension is appreciably reduced and duration of postoperative analgesia also becomes prolonged following levobupivacaine administration.

Recommendation

Intrathecal levobupivacaine-fentanyl combination could be an effective and reliable alternative to bupivacaine-fentanyl combination in patients undergoing total abdominal hysterectomy under sub-arachnoid block.

Conflict of Interest: None.

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References:

1. Velde MV, Teunkens A, Hanssens M. Intrathecal sufentanil and fetal heart rate abnormalities: a double-blind, double placebo-controlled trial comparing two forms of combined spinal epidural analgesia with epidural analgesia in labor. Anesth Analg. 2004; 98: 1153-59.

https://doi.org/10.1213/01.ANE.0000101980.34587.66

PMid:15041616

2. Nakamura G, Ganem EM. Effects on mother and fetus of epidural and combined spinal-epidural techniques for labor analgesia. Rev Assoc Med Bras. 2009; 55: 405-09.

https://doi.org/10.1590/S0104-42302009000400014

PMid:19750306

3. Akbay KB, Boztepe A, Sezen YG. A comparison of bupivacaine solutions with or without fentanyl intrathecally Türkiye Klinikleri. J Med Sci. 2007; 5: 118-23.

4. Kokki H, Tuovinen K, Hendolin H. Spinal anesthesia for paediatric day-case surgery: a double-blind, randomized, parallel group, prospective comparison of isobaric and hyperbaric bupivacaine. Br J Anaesth. 1998; 81: 502-06.

https://doi.org/10.1093/bja/81.4.502

PMid:9924220

5. Hallworth SP, Fernando R, Columb MO, Stocks GM. The Effect of Posture and Baricity on the Spread of Intrathecal Bupivacaine for Elective Cesarean Delivery. Anesthesia & Analgesia. 2005; 100(4): 1159-1165.

https://doi.org/10.1213/01.ANE.0000149548.88029.A2

PMid:15781538

6. Parlow JL, Money P, Chan P. Addition of opioids alters the density and spread of intrathecal local anesthetics? An in vitro study. Can J Anesth. 1999; 46: 66-70.

https://doi.org/10.1007/BF03012518

PMid:10078407

7. Santos LM, Santos VCJ. Intrathecal morphine plus general anesthesia in cardiac surgery: effects on pulmonary function, postoperative analgesia and plasma morphine concentration. Clinics. 2009; 64: 279-85.

https://doi.org/10.1590/S1807-59322009000400003

PMid:19488583 PMCid:PMC2694465

8. Povey HMR, Jacobsen J, Westergaard-Nielsen J. Subarachnoid Analgesia with Hyperbaric 0.5% Bupivacaine: Effect of a 60-Min Period of Sitting. Acta Anaesthesio- logica Scandinavica. 1989; 33(4): 295-297.

https://doi.org/10.1111/j.1399-6576.1989.tb02911.x

PMid:2655366

9. Panula J, Pihlajamaki H, Mattila VM. Mortality and cause of death in hip fracture patients aged 65 or older: a population-based study. BMCm Musculoskelet Disor. 2011: 12.

https://doi.org/10.1186/1471-2474-12-105

PMCid:PMC3118151

10. Erdil F, Bulut S, Demirbilek S. The effects of intrathecal levobupivacaine and bupivacaine in the elderly. Anaesthesia. 2009; 64: 942-46.

https://doi.org/10.1111/j.1365-2044.2009.05995.x

PMid:19686477

11. Fattorini F, Ricci Z, Rocco A. Levobupivacaine versus racemic bupivacaine for spinal anaesthesia in orthopedic major surgery. Minerva Anestesiol. 2006; 72: 637-44.

12. Herrera R, Andres J, Estan L, Olivas FJM, Martínez-Mir I and Steinfeldt T. Haemodynamic impact of isobaric levobupivacaine versus hyperbaric bupivacaine for subarachnoid anesthesia in patients aged 65 and older undergoing hip surgery. BMC Anesthesiology. 2014; 14: 97.

https://doi.org/10.1186/1471-2253-14-97

PMid:25371654 PMCid:PMC4218988

13. Guler G, Cakir G, Ulgey A, Ugur F, Bicer C, Gunes I, et al. A Comparison of Spinal Anesthesia with Levobupivacaine and Hyperbaric Bupivacaine for Cesarean Sections: A Randomized Trial. Open J Anesthesiol. 2012; 2: 84-89.

https://doi.org/10.4236/ojanes.2012.23020

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ORIGINAL ARTICLE

OPEN ACCSESS

Estimation of Prevalence of Musculoskeletal Pain & Rheumatic Disorders in a Rural Community of Bangladesh

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Abstract

Introduction: *Rheumatic disorders are one of the largest health problems in the world both in developed & developing countries. They are the most common causes of chronic health problems and long term physical disability, work loss and loss of income. The study was conducted among the 15 plus years aged people in selected areas based on broad objective of exploring the prevalence of rheumatic disorders.* ***Materials and Methods:*** *It was an Observational cross sectional study. The study was conducted among the people of nineteen small villages of Sonargaon upazilla in Narayanganj district, Bangladesh. This study was conducted From January 2010 to December 2011. All subjects of the defined area aged ≥ 15 years were included in this study.* ***Results:*** *The point prevalence rate of definite rheumatic disorders was 23.7%. Age, sex and occupations were associated with the overall prevalence rate. The prevalence was higher (51.8%) in older aged (65years and above) population. Females (34.5%) were affected more than males (18.6%) in this rural area.* ***Conclusion:*** *These diseases are associated with some form of disability and work loss, as well as loss of income. More community-based studies with the design for the identification of associate's factors for individual rheumatic disease are needed. So that appropriate messages can be given to the community for prevention and treatment of rheumatic disorders.*

Keywords: *Prevalence, Rheumatic disorders, Rural community.*

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Introduction:

Musculoskeletal disorders are common and disabling but are low priorities for public health resources and medical education curricula (Dequeker et al., 2000)[2]. In the world much effort has directed at killing diseases, whereas crippling diseases are relatively neglected and yet the social and economic burden which the later imposes is probably greater.

Prevalence studies for musculoskeletal pain and rheumatic disorders were performed in different western countries and data for major rheumatic disorders are available (Badely et al., 1994)[1] (Reyes-Liernea et al., 2009)[3]. But for giving emphasis on rural communities in developing countries, WHO & International League of Association for Rheumatology (ILAR) jointly founded the Community Oriented Program for Control of Rheumatic Disorders (COPCORD) in 1981. Primary objective of the COPCORD is reduction of community burden of rheumatic disorders, particularly in developing countries (Chaimnuay et al., 1998)[4]. COPCORD has played a crucial role in collecting data on rheumatic complaints and disability in rural areas especially in developing countries. The epidemiological studies of rheumatic diseases by WHO-ILAR-COPCORD have already been performed in urban and rural Filipinos (Dans LF et al., 1997)[5], in rural & urban Indonesians (Darmawan J et al., 1992)[6], And in many others country all over the world. These studies denote the prevalence of musculoskeletal pain has varied from 11.6% to 45.5%.

The Point Prevalence estimates of musculoskeletal pain in rural, urban slum and affluent urban communities were 26.2% (women 31.3%, men 21.1%), 24.9% (women 27.5%, men 22.6%), and 27.9% (women 35.5%, men 18.6%), respectively was reported by COPCORD study group of Bangladesh. The overall point prevalence of musculoskeletal pain was 26.3% but the point prevalence of definite rheumatic disorders was 24% (S. A. Haq et al., 2005)[7]. In Bangladesh commonest rheumatic disorders were osteoarthritis of the knees, nonspecific low back pain, lumber spondylosis, fibromyalgia and soft tissue rheumatism. Their prevalence estimates were 7.5%, 6.6%, 5%, 4.4%, 2.7% respectively, in the rural, 9.2%, 9.9%, 2.0%,

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3.2% and 2.5%, respectably, in the urban slum, and 10.6%, 9.2%, 2.3%, 3.3% and 3.3% in the urban affluent community (S. A. Haq et al., 2005)[7]. There were large differences between different communities in the prevalence of pain at any site and of different rheumatic disorders. This difference may be attributed to differences in customs, habits, occupation and living conditions, but may also arise, at least partly, from differences in the methodology, adopted operational definitions and classification of symptoms or diseases.

To determine exactly, the socio-demographic characters, prevalence rate and disability burdens of musculoskeletal pain and rheumatic disorders in Bangladesh it is necessary to collect authentic data from the rural community, so that appropriate preventive measures can be taken to prevent and control the rheumatic morbidities. The population of Sonargaon upazilla of Narayangonj district was selected for community study. For effective supervision, quality control, reduction of cost, as well as compare with previous study the same rural area near Dhaka city was selected for the study.

Objectives

To estimation the prevalence of definite rheumatic disorders in a rural community and also try to search the socio-demographic factors associated with the rheumatic disorders of the study population.

Materials and Methods:

It was an Observational cross sectional study. The study was conducted among the people of nineteen small villages of Sonargaon upazilla in Narayanganj district, Bangladesh. This study was conducted From January 2010 to December 2011. All subjects of the defined area aged ≥ 15 years were included in this study.

Inclusion criteria are subjects of both sex aged ≥15 years were included in this study.

Exclusion criteria are critically ill patients (such as stroke, severe respiratory failure, unconscious patients), mentally handicapped persons (mental defect congenital or acquired preventing or resisting a person from participating in normal life or limiting their capacity to work) and relatives of the residents who had come to visit the study area for short period.

Data Processing

After collecting data were checked thoroughly for constancy and completeness. Data were checked to exclude any error or inconsistency.

Statistical Analysis

All analysis was done by appropriate statistical methods using spss are software for windows.

Ethical Issues

All ethical issues, which were related the research involved with human subject were followed according to the guideline of ethical review committee.

Sample Size

Sample size was calculated by using the following formula- $N = \frac{Z^2 pq}{d^2}$

Here,

N = required sample size

Z = 95% Confidence interval (1.96)

P = Prevalence or proportion of occurrence of rheumatic diseases in rural area 26% (0.26)

q = 100-p or proportion of person not affected by the disease, 74% (0.74)

d = Acceptable (allowable) error, 10% of P, 2.6% (0.026)

So, sample size was 1093(one thousand ninety-three). We considered 10% dropout, so approximate sample size was 1202, rounded up to 1200(one thousand two hundred).

NB: As prevalence studies all subjects in nineteen villages, aged 15 and more than 15 years were included for this study.

Sampling Technique

Sampling was done on the basis of some predetermined idea or subjective judgment. Here study area and study population was prefixed. Field workers with the supervision of the investigator identified persons with musculoskeletal pain by using culturally adapted and validated modified Bengali version of COPCORD questionnaire. Sample was enrolled on the basis of inclusion and exclusion criteria.

Observations and Results:

Total 5005 study subjects were selected for the study. Among them, 4850 (male-2447, female- 2403) were interviewed giving and here total response rate 96.9%. Out of 4850 respondents, musculoskeletal pain and rheumatic disorder positive respondents were 1283 (male-454, female-829) and total Positive respondents rate 26.45%. Male positive respondents rate 18.55% and female respondents rate 34.92%. Rheumatic Disorder positive rate is higher among female then male (Fig.1).

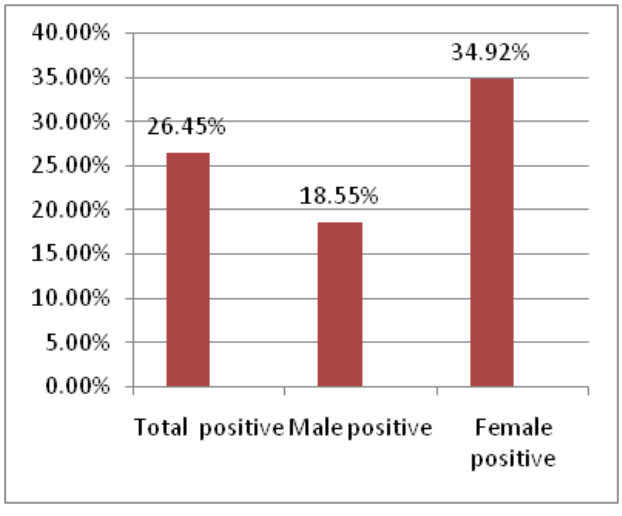

Figure-1: Prevalence of rheumatic disorders.

Prevalence of positive respondents by age group and sex:

The distribution of positive respondents by age group and sex is shown in table-I

Table-I: Point prevalence of musculoskeletal pain by occupational group.

Occupation	Interviewed population		Positive respondents	
	Number	Percent	Positive	Percent
Students	638	13.2	38	5.9
Cultivators	116	2.4	46	39.7
Service (in door)	195	4.0	18	9.2
Retired persons	229	4.7	86	37.6
Housewives	1850	38.1	688	37.2
Day Laborers	63	1.3	24	38.1
Workers	442	9.1	60	13.6
Weavers	667	13.8	186	27.9
Garments worker	56	1.2	7	12.5
Drivers	44	0.9	8	18.2
Rickshaw/Van driver	13	0.3	8	61.5
Businessmen	400	8.2	55	13.8
Tailors	16	0.3	4	25.0
Others	121	2.49	55	45.45

The prevalence of musculoskeletal complaints increased with the increase of age. It was 12.6% in the aged group 15-24 years and 51.8% in 65 years and above aged group (table-II).

Table-II: Prevalence by age group and sex distribution

Age Group	Interviewed population			Positive respondent		
	Male	Female	Total	Male	Female	Total
15 – 24	741 (30.2)#	766 (31.8)	1503 (31.0)	60 (8.1)	130 (17.0)	190 (12.6)
25 – 34	671 (27.4)	618 (25.7)	1289 (26.6)	117 (17.4)	166 (26.9)	283 (22.0)
35 – 44	442 (18.1)	441 (18.4)	883 (18.2)	93 (21.0)	191 (43.3)	284 (32.2)
45 – 54	272 (11.1)	276 (11.5)	548 (11.3)	68 (25.0)	164 (59.4)	232 (42.3)
55 – 64	197 (8.1)	177 (7.4)	374 (7.7)	62 (31.5)	103 (58.2)	165 (44.1)
65 +	124 (5.1)	125 (5.2)	249 (5.1)	54 (43.5)	75 (60.0)	129 (51.8)
Total	2447	2403	4850	454	829	1283

#Data in the parentheses indicate percentage

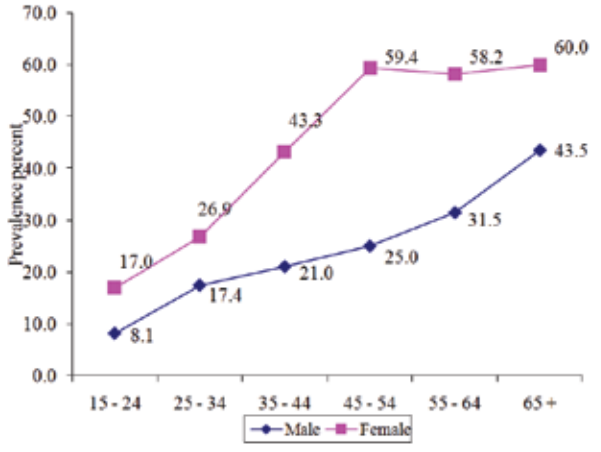

Figure-2: Prevalence of musculoskeletal pain by age group and sex.

Prevalence of musculoskeletal pain by occupation:

By occupation the prevalence of musculoskeletal pain was more common among the Rickshaw/Van drivers, cultivators, day laborers, retired persons and housewives. The prevalence of rheumatic pain was 61.5%, 39.7%, 38.1%, 37.6% and 37.2% respectively among rickshaw/van drivers, cultivators, day laborers, retired persons and housewives (table -III).

Table-III: Prevalence of major rheumatic diseases by occupations.

Occupations	OA Knee (%)	LBP (%)	Lumber spondylosis (%)	Fibromyalgia (%)
Student	0.31	1.88	0.00	0.94
Cultivator	17.24	5.17	4.31	1.72
Sales person	1.54	1.54	1.54	0.00
Retired persons	23.14	3.49	9.17	2.18
Housewife	13.14	11.24	2.81	2.27
Day Laborers	15.87	4.76	1.59	0.00
Worker	3.17	3.85	0.45	1.58
Weaver	1.80	10.49	0.90	1.95
Garments worker	0.00	3.57	0.00	0.00
Rickshaw/Van driver	3.51	5.26	0.00	1.75
Business	1.75	4.00	1.25	1.75
Others	12.04	7.41	2.78	3.70

The frequency of musculoskeletal pain was 19.0% in the smokers and 47.8% in the tobacco leaf users (table-IV).

Table -IV: Prevalence of musculoskeletal pain by habit:

Habit	Interviewed population			Positive respondent		
	Male	Female	Total	Male	Female	Total
Smoking	546 (22.3)#	21 (0.9)	567 (11.7)	102 (18.7)	6 (28.6)	108 (19.0)
Tobacco leaf	183 (7.5)	369 (15.4)	552 (11.4)	73 (39.9)	191 (51.8)	264 (47.8)
Others	19 (0.8)	6 (0.2)	25 (0.5)	5 (26.3)	1 (16.7)	6 (24.0)
Total	2447	2403	4850	454	829	1283

#Data in the parentheses indicate percentage

Table-V: Frequency by Economic status.

Social status	Interviewed population		Positive respondents	
	Number	Percent	Positive	Percent
Upper class	228	4.7	56	24.6
Middle class	714	14.7	187	26.2
Lower class	3908	80.6	1040	26.6
Total	**4850**		**1283**	

Economic status of the positive respondents:

The frequency of MSK pain was 24.6%, 26.2% and 26.6% respectively in upper, middle and lower class (table-VI).

Table-VI: Prevalence of MSK pain and rheumatic diseases.

Name of diseases	Male (N=2447)	Female (N=2403)	Total (N=4850)	95% CI
RA	4 (0.2)#	28 (1.2)	32 (0.7)	0.47 – 0.93
SSA-AS	20 (0.8)	25 (1.0)	45 (0.9)	0.69 -1.24
SSA-PSA	0 (0.0)	2 (0.1)	2 (0.04)	0.01 - 0.15
Reactive arthritis	1 (0.04)	3 (0.1)	4 (0.1)	0.03 - 0.21
OA other than knee	44 (1.8)	74 (3.1)	118 (2.4)	2.04 - 2.91
OA-Knees	106 (4.3)	275 (11.4)	381 (7.9)	7.13 - 8.65
Fibromyalgia	36 (1.5)	52 (2.2)	88 (1.8)	1.48 - 2.23
Frozen Shoulder	2 (0.1)	11 (0.5)	13 (0.3)	0.16 – 0.46
STR	41 (1.7)	61 (2.5)	102 (2.1)	1.74 - 2.55
SR-UBP	37 (1.5)	37 (1.5)	74 (1.5)	1.22 - 1.91
SR-LBP	115 (4.7)	249 (10.4)	364 (7.5)	6.80 - 8.28
VS (No specific disease)	55 (2.2)	81 (3.4)	136 (2.8)	2.38 - 3.31
VSR-PIVD	6 (0.2)	14 (0.6)	20 (0.4)	0.27 - 0.64
VSR-Radiculopathy	1 (0.04)	9 (0.4)	10 (0.2)	0.11 - 0.38
TRM	19 (0.8)	12 (0.5)	31 (0.6)	0.45 – 0.91
Total (MSK pain)	**454 (18.6)**	**829 (34.5)**	**1283 (26.5)**	**25.23 - 27.71**

* Multiple responses

#Data in the parentheses indicates percentage.

Discussion:

This was an observational cross-sectional study to explore the musculoskeletal pain and rheumatic morbidity in the populations aged ≥15 years in the nineteen small villages of Sonargaon upazilla of Narayangonj district. This study was conducted in two phases. The screening phase was conducted by eight trained field workers and examination phase by eight physicians including two rheumatologists. The response rate during screening and examination phases were 96.9% and 96.8% respectively.

The point prevalence rate of musculoskeletal pain was 26.5% .The prevalence of definite rheumatic disorders was 23.7% and the remaining 2.8% had no identifiable musculoskeletal syndrome. Age, sex and occupations were associated with the overall prevalence rate. The prevalence was higher (51.8%) in older aged (65yers and above) population. Females (34.5%) were affected more than males (18.6%) in this rural area. The commonly affected occupational groups were rickshaw/van drivers, cultivators, day laborers, retired persons and housewives respectively. The highest prevalence was recorded in lower class and then middle and upper class respectively. Among positive respondents tobacco leaf users were more frequently affected.

The most common site of pain of the body was low back (21%) followed by knee (14%), shoulder (7%), hip (7%), and ankle (6%). OA knee 7.9 %,(95% CI 7.13-8.65) was the most frequent rheumatic disease identified in the rural population followed by low back pain 7.5 %,(95% CI 6.80-8.28) OA other than knee 2.4 %,(95% CI 2.04-2.91), STR 2.1%, (95% CI 1.74- 2.55) fibromyalgia 1.8%, (95% CI 1.48- 2. 23) and upper back pain 1.5% (95% CI 1.22-1.91). Rheumatoid arthritis was found in 0.7% of the population and others inflammatory arthritis were uncommon. Females were affected more frequently than males in all cases.

Functional disability in various forms was identified among positive respondents and only few of them had complete inability to do specific tasks. Nineteen percent of rheumatic patients had work loss with a mean duration of 18 days in the preceding 12 months among positive respondents. Majority of positive cases commonly used NSAIDS or analgesic for relieving pain with or without prescription.

Conclusion:

Rheumatic diseases are common in the Bangladeshi rural community. The study revealed wide spectrum rheumatic diseases of which degenerative and mechanical diseases were the main group of disorder. These diseases are associated with some form of disability and work loss, as well as loss of income. More community-based studies with the design for the identification of associate's factors for individual rheumatic disease are needed so that appropriate messages can be given to the community for prevention and treatment of rheumatic disorders.

Conflict of Interest: None.

Acknowledgment: This study was conducted by self-funded.

Bibliography:

1. Badley EM, Rasooly I, Webster GK. Relative importance of musculoskeletal disorders as a cause of chronic health problems, disability and health care utilization: Findings of 1990 Ontario Health Survey. J Rheumatol. 1994; 21: 505-14.

2. Dequeker J, Rasker JJ, Woolf AD. Educational issues in rheumatology. Baillieres Rheumatol. 2000; 14: 715-29.

https://doi.org/10.1053/berh.2000.0109

3. Reyes-Lierena GA, Guibert - Toledano M, Cardiel MA, Prevalence & Barden of Rheumatic diseases in Cuba. A COPCORD study. J clin Rheumatol. 2009; 15 (2): 51-5.

https://doi.org/10.1097/RHU.0b013e31819b61cb

PMid:19265344.

4. Chaiamnuay P, Darmawan J, Muirden KD, Assawatana bodee P. Epidemiology of rheumatic diseases in rural Thailand: a WHO-ILAR-COPCORJD study. Community Oriented Program for the Control of Rheumatic Disease. J Rheumatol. 1998; 25: 1382-7.

5. Dans LF, Tankeh-Torres S, Amante CM, Penserga EG. The prevalence of rheumatic disease in a Filipino urban population; a WHO-ILAR-COPCORD study. J Rheumatol.1997; 24: 1814-9.

https://doi.org/10.1016/S0895-4356(98)90015-4

6. Darmawan J, Volkenburg HA, Muirden KD, Wigley RD. Epidemiology of rheumatic disease in rural and urban Indonesia: a WHO-ILAR-COPCORD study, stage -1, phase- 2.Ann rheum dis.1992; 51: 525-8.

https://doi.org/10.1136/ard.51.4.525

PMid:1586254 PMCid:PMC1004706.

7. S.A. Haq, Darmawan John, Islam MN. Prevalence of rheumatic diseases and associated outcomes in rural and urban communities in Bangladesh: A COPCORD study. J Rheumatol. 2005; 32: 348-353.

DOI: https://doi.org/10.3329/medtoday.v32i1.44829

ORIGINAL ARTICLE OPEN ACCSESS

Clinico-Biochemical Abnormalities in Hospitalized Neonates with or without Seizure

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Abstract

Introduction: *To determine the clinical types and etiological factors and to assess the biochemical abnormalities in neonates with seizure.* ***Materials and Methods:*** *This was a Cross-sectional, comparative hospital based study. Ninety neonates, age up to 28 days of both sexes were evaluated for clinical types, etiological factors and biochemical abnormalities with seizure and compared with those having no seizures. The variables were analyzed using student t- test. All the data was processed and analyzed by computer software SPSS version 15.0. Level of significance was considered as p value less than 0.05.* ***Results:*** *Most of the neonates having seizures (72%) within 3 days of life. The seizures were common in male babies (62%). 35% of the mother of baby with seizure gave history of prolonged labour. HIE was diagnosed 56.67% neonates with seizure whereas infection were found in 25% cases with seizure. Among the seizure subtypes, subtle seizures were 58% followed by clonic seizures 30%. Significant biochemical changes we found in 43.34% of neonates with seizures. Hypocalcemia (46%) was most common followed by hypoglycemia(38%).* ***Conclusion:*** *Hypoxic ischemic encephalopathy was the commonest cause of neonatal seizure followed by neonatal infections including meningitis and sepsis.. Biochemical abnormalities are more common in neonates with seizure than neonates free from seizure. Among the biochemical abnormalities hypocalcaemia and hypoglycemia occurs most commonly followed by hyponatraemia and hypomagnesaemia.*

Keywords: *Neonate, Seizure, Biochemical abnormality.*

Number of Tables: 03; Number of Figures: 02; Number of References: 24; Number of Correspondence: 04.

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Introduction:

Neonatal seizures are common and may be the first manifestation of neurological dysfunction after a variety of insults[1]. Neonatal seizures are clinically significant because very few are idiopathic[1,2]. Moderate to severe acute hypoxic ischemic encephalopathy (HIE) accounts for approximately two third of all cases of neonatal seizures[3]. Biochemical disturbances occur frequently in neonatal seizures either as an underlying cause or as an associated abnormality[4,5,6]. Among the biochemical abnormalities hypocalcemia and hypoglycemia were most common showed in different studies. Hypoglycemia is common among neonates whose mothers have diabetes, large for gestational age, having HIE or other stresses[7]. Early onset (first 72 hours) hypocalcemia usually associated with preterm LBW, IUGR, asphyxia, infant of diabetic mother where as late onset hypocalcemia is seen in child fed evaporated cow's milk & other improper formulas[3,8]. About half of neonates with seizures secondary to hypocalcemia, also have hypomagnesemia and should be considered if seizures continue after given therapy[7,9]. Prognosis of neonatal seizures however is related to pathogenesis i.e. prolong or recurrent hypoglycemia permanently affect the CNS[7]. Thus early recognition and treatment of biochemical disturbances is essential for optimal management and satisfactory long term outcome[1,2]. The present study is designed to assess the biochemical abnormalities associated with neonatal seizure and to compare the findings with those of neonates having no seizures.

Materials & Methods:

The present study was a cross sectional, comparative study conducted in the department of pediatrics, Mymensingh Medical College Hospital, Mymensingh, Bangladesh from 1st July 2008 to 30th June 2009. Total 90 neonates within 28 days of life were included in the

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study. Among them 60 were with seizures and rest 30 were without seizures. Both study group and reference group were taken according to inclusion criteria and selected randomly. Seizures were diagnosed and classified by author himself or by authorized person. The neonatal seizures were classified according to Volpe's classification. Hypoglycemia were defined as blood sugar < 40 mg/dl, and hypocalcaemia when total serum calcium was < 7.0 mg/dl. Age, sex, etiological factors and biochemical parameters were recorded in a predesigned data sheet. The data was analyzed by using SPSS version 15.0.

Results:

Total 90 neonates who were admitted into neonatal unit in MMCH included in this study. Among them 60 having convulsion and rest are without convulsion. Out of 90 neonates 54 (60%) were male and 36(40%) were female. 37(61.67%) of the neonates with seizure were male. On the other hand 17(56.66%) males having no seizure among control group. Most of the seizures about 43 (71.66%) cases occurring in our study within first three days of life. In the present study 32 babies had seizure in the range of 1-15 minutes. 27 babies had seizures less than 1 minute of duration and one baby had seizure persisting more than 15 minutes. Among the mothers of neonates with seizure, 34(56.6%) suffered from different types of problems during pregnancy or labour. Twenty one (56.6%) having history of prolong labour and 5 (8.3%) having PROM. Majority about 42(70%) in neonates with seizure and 18(60%) in neonates without seizure had normal vaginal delivery and caesarian section were 16(26.6%) and 12(40%) respectively. Thirty five (58%) neonates with seizure were born in home. Most of the neonates with seizure 58(96.67%) and without seizure 27 (90%) under study were born within normal gestational period. Maximum patient under this study were born at term about 91%. About 16 (26.66%) of the neonates were fed with foods other than colostrums in neonates with seizure. Neonates were subsequently fed with breast milk in 41 cases in neonates with seizure. Hypoxic ischemic encephalopathy (HIE) was the commonest diagnosis found in 34 (56.67%) neonates with seizure group and other causes are septicemia, meningitis intracranial hemorrhage and low birth weight. No cause could be identified in 4 (6.67%) cases of neonates with seizure, out of which 3 (5%) suspected clinically due to primary metabolic disorder. Subtle types of seizure were noticed in 35 (58.33%) cases and subsequently focal or multifocal clonic were 18 (30%) and 7 (11.67%) patients had tonic type of seizures. One of them was generalized tonic convulsion. There was no myoclonic type of seizure diagnosed. 26(43.33 %) patients had eye manifestation and 16 (26.67%) patients had mouth deviation. The biochemical parameters done in the present study were the blood glucose, serum calcium, serum sodium, serum magnesium, serum potassium and serum inorganic phosphate level and to see if any significant biochemical changes in different types of seizure occurring due to different causes and it was compared with that in non-seizure neonates.

The mean serum sodium (134.8 ± 4.3 meq/L) was found to be much lower in the neonates with seizure than the neonates without seizure (139.74 ± 3.5 meq/L). The mean blood glucose level in the neonates with seizure was 2.72 ± 0.91 m mol/L which was also much lower than the other group where it was 4.18 ± 0.96 mmol/L. The neonates with seizure also had lower mean serum calcium level 7.25 ± 0.4 mg/dl which was 8.51 ± 0.72 mg/dl in case of neonates without seizure **(Table III)**.

Table-I: Values expressed as number and percentages. The total no. of significant biochemical changes are more due to multiple response i.e. multiple biochemical changes in a single case.

Table-I: Types of seizure with biochemical changes.

Seizures type	Total no of cases	Significant biochemical change	Hypoglycemia	Serum Na		Hypocalcaemia	Hypomagnesemia	Hypokalaemia	Hyperphosphatemia
				Hypo	Hyper				
Subtle	35	16 (45.71%)	5	2	0	6	2	1	0
Clonic	18	10 (55.55%)	4	0	1	4	1	0	0
Tonic	7	4 (57.14%)	1	1	0	2	0	0	0
Myo clonic	0	0	0	0	0	0	0	0	0
Total	60	26 (43.33%)	10 (38.4%)	3 (11.5%)	1 (3.8%)	12 (46.1%)	3 (11.5%)	1 (3.8%)	0

Fig 1: Hypoxic ischemic encephalopathy (HIE) was the commonest diagnosis was found in 56.67% neonates with seizure group. Infection in the form of septicemia and meningitis were found in 25% cases with seizure.

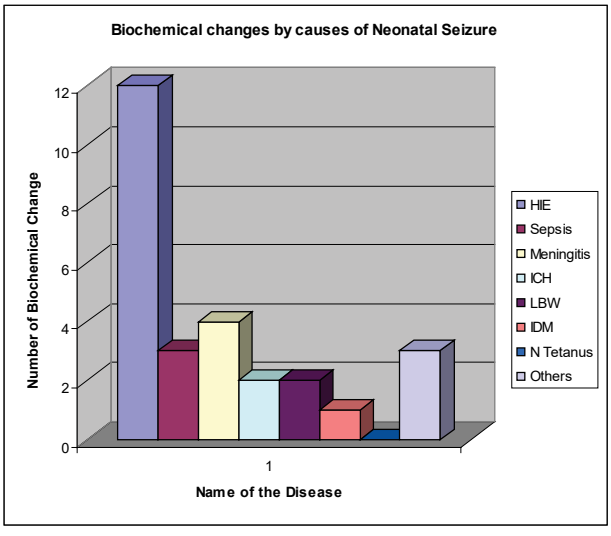

Figure-1: Clinical diagnosis of the neonates with seizur.

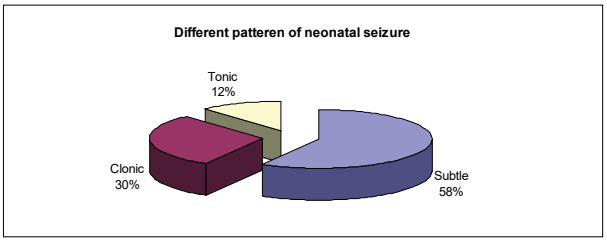

Figure-2: Types of seizure among Study group (n=60).

Table II. shows hypocalcemia and hypoglycemia were more common in neonates with seizure where as hypoglycemia and hyponatraemia were found in neonates without seizure.

Table-II: Distribution of seizure and non seizure neonates with biochemical changes.

Biochemical parameters	Neonates with Seizures (n=60)	Neonates without Seizures (n=30)
Hypoglycemia	10 (16.67%)	1 (3.34%)
Hypocalcaemia	12 (20%)	0
Hypomagnesaemia	3 (5%)	0
Hyponatraemia	3 (5%)	1 (3.34%)
Hypernatraemia	1 (1.67%)	0
Hypokalaemia	1 (1.67%)	0
Hyperphosphatemia	0	0

Table III: Statistical T-test was done to see if there is any statistically significant difference in the biochemical levels of the neonates with seizure and no seizure. Statistically significant difference ($p < 0.001$) was found in cases of Blood glucose, Serum calcium, Serum sodium and Serum magnesium level between the two groups.

Table-III: T-test: Neonates with seizure and no seizure by biochemical changes.

Biochemical parameters	Seizure	Number	Mean	SD	T-value	P-value
Blood Glucose	Yes	60	2.72	.91	7.02	< .001
(m mol/L)	No	30	4.18	.96		
Serum Sodium	Yes	60	134.8	4.3	5.3	< .001
(meq/L)	No	30	139.74	3.5		
Serum Calcium	Yes	60	7.25	.40	10.4	< .001
(mg/dl)	No	30	8.51	.72		
Serum Potassium	Yes	60	4.28	.68	.79	>.05
(meq/L)	No	30	4.4	.66		
Serum Magnesium	Yes	60	1.91	.35	3.5	< .001
(mg/dl)	No	30	2.35	.29		
Serum Phosphate	Yes	60	2.1	.33	.88	>.05
(mmol/L)	No	30	2.03	.40		

Discussion:

Neonatal seizure is an acute emergency which is responsible for very high morbidity and mortality[10,11]. Neonatal seizure observed more in male patient (61.67%) than female (38.33%) in this study which is consistent with study done by Taksande AM et al[10] showed 66.4% male and 33.6% in female newborn. Shah et al[11] also found male predominant in neonatal seizure. The most common seizure type in term and preterm babies is the subtle seizure[12,13]. In this study, majority of the patients (58.33%) showed subtle type of seizure whereas clonic and tonic seizures were 30% and 11.67% respectively. Ross et al[14] showed 40.6% subtle, 35.59% clonic, 8.9% generalized tonic and 27.78% myoclonic type of seizures. Soni A et al[15] reported 25% subtle and 37.5% tonic seizures and Taksande AM et al[10] showed 31.1% subtle and 46.7% clonic seizures in term babies whereas 48.4% had subtle and 27.2% had clonic seizures in preterm babies and tonic seizures observed in 19.4% and 24.2% in term and preterm babies respectively. Shah et al[11] reported 42.2% subtle, 44.4% clonic, 11.1% tonic and 2.2% myoclonic seizures in a observational study conducted in Nepal.

Coen RW et al[16] found 81% of babies had early onset seizures (< 72 hrs). Ross AL et al[14] and Taksande AM et al[10] also found early onset seizures in 50.33% and 85.45% babies respectively whereas in this study we found 71.66% neonates had seizures within 72 hrs. Brown JK et al[17] observed 43% neonates had seizures in the first 4 days of life while Ronen GM et al[18] reported 83% seizures in the first week of the life.

The etiology of neonatal seizure is not disease specific and may be due to a combination of abnormalities[4]. The most common cause of seizure encountered in this study was birth asphyxia, occurred in 34 (56.67%) cases. Sood et al,[4] Kumar et al,[5] and Shah et al[11] reported birth asphyxia as etiology of seizure was 45.71%, 48.27% and 44% cases respectively which are comparable with the result of present study. Similar observation seen by Mizrahi where HIE as the cause of convulsion in 46% cases[19]. Taksande AM et al[10] reported 42.7% and Brown et al[17] showed 65% cases of neonates of perinatal asphyxia experienced with seizure. Eriksson M et al,[20] Painter MJ et al [21] and Holden KR[22] et al also showed similar results.

Sepsis in neonate is often associated with meningitis and is a cause of significant morbidity and mortality[12]. A study conducted by Legido A et al[23] reported 5% septicemia and 12.25% meningitis. Ross et al[14] showed 9.5% babies had septicemia. Shah et al[11] and Taksande AM et al[10] both of the study reported about 20% cases suffered from infection; sepsis and meningitis combined. These results are concordance with our study where sepsis were diagnosed in 13.33% cases and meningitis in 11.67% cases of neonates with seizure. Preterm babies are more vulnerable to intraventricular hemorrhage either spontaneously or as a result of Perinatal asphyxia[13]. Taksande AM et al[10] showed 1.29% term neonate and 18.18% preterm neonate had IVH whereas Ross et al[14] and Scher MS et al[24] also reported higher incidence of IVH in preterm babies which are comparable with present study where we found out of three neonates (5%) having IVH which were diagnosed clinically and one of them were preterm.

The incidence of primary metabolic cause of convulsion is about 10%[12]. In this study, 4 (6.67%) cases no other cause of seizure could be detected. Out of them 3 (5%) were clinically suspected as primary metabolic disorder. One of them was hypoglycemic and other two cases showed significant hypocalcaemia. Hypomagnesaemia was associated with one of the hypocalcemic baby. Kumar et al shown primary metabolic disorder accounted 25.7% causes of seizure[5]. This is in contrast to report from western countries where improvements in infant feeding practices have made this category an uncommon cause of seizures[8]. In a study by Cockburn et al it was found that 55% of neonatal seizures were due to primary disturbance of mineral metabolism[6].

Hypocalcaemia was the most common biochemical abnormalities among 12(46.15%) neonates in this study (Table I)

similar to the study done by Sood et al[4] about 48.27% and Kumar et al[5] about 31.8% cases. Hypoglycemia was the second most common biochemical abnormalities found in 38.46% cases which similar to Sood et al[4] and Kumar et al[5] where values were 48.27% and 50% respectively. Shah et al[11] reported 22% whereas Taksande et al[10] experienced only 8.1% of hypoglycemia in neonatal seizures. Three (11.53%) neonates with seizure present with hyponatraemia and 1 (3.84%) with hypernatraemia, 1(3.84%) with hypokalaemia and 3(11.53%) with hypomagnesaemia which is comparable with study done by Sood et al[4] and Kumar et al[5] where reported 17.24% and 45.45% cases of hyponatraemia respectively. Hypernatraemia and hypokalaemia we found was associated with HIE. Two baby of hypomagnesaemia were associated perinatal asphyxia and one was suspected primary metabolic disorder. Two cases were associated with hypocalcaemia. Kumar et al found hypomagnesaemia in 9% case of neonatal seizures[5] which is consistent with our study.

Biochemical changes we found 6.67% cases of neonates without seizures and 43.33% neonates with seizures. Among these non-seizure cases, one (3.3%) was hyponatraemic diagnosed as a case of birth asphyxia and another one was hypoglycemic diagnosed as neonatal sepsis. The biochemical changes found in the non-seizure neonates may be attributed to the disease conditions with which the neonates were admitted to the hospital. The mean of all the biochemical parameters except serum phosphate were found to be higher (within normal range) in the non-seizure cases. The difference in the mean value of glucose, calcium, magnesium and sodium between the two groups was found to be statistically significant ($p < 0.001$).

In this study, though individual variation in respect of race, geography and socioeconomic condition are present even then most of the study findings are consistent with that done in abroad.

Conclusion:

Perinatal asphyxia leading to Hypoxic ischemic encephalopathy was the commonest cause of neonatal seizure followed by neonatal infections including meningitis and sepsis. Most seizures occurred within first 72 hours of postnatal age. Meningitis mostly occurs after first week of life. Subtle seizure was the commonest type of seizure that can easily missed, as it is very mild. Biochemical abnormalities are more common in neonates with seizure than neonates free from seizure. Among the biochemical abnormalities hypocalcaemia and hypoglycemia occurs most commonly followed by hyponatraemia and hypomagnesaemia.

Conflict of Interest: None.

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References:

1. Evans D, Levene M. Neonatal seizures. Arch Dis Child Fetal Neonatal Ed. 1998; 78: F70-F75.

https://doi.org/10.1136/fn.78.1.F70

PMid:9536846 PMCid:PMC1720746

2. Gomlla T L. Neonatal seizures. In: Neonatology: Management, Procedures, On-call problems, Diseases, and Drugs. 5th edn, McGraw Hill, New York; 2004: 496-501.

3. Fenichel GM. Paroxismal disorder of newborn. In: Clinical Pediatric Neurology. 4th Ed. W B Saunders Company Philadelphia; 2001:1-5.

4. Sood A, Grover N and Sharma R. Biochemical abnormalities in Neonatal seizures. Indian J Pediatrics. 2003; 70(3): 221-224.

https://doi.org/10.1007/BF02725588

PMid:12785293

5. Kumar A, Gupta V, Kacchawaha J.S. and Singla P.N. Biochemical Abnormalities in Neonatal Seizures. Indian Pediatrics. 1995; 32: 424-428.

6. Cockburn F, Brown JK, Belton NR, Forfar JO. Neonatal convulsions associated with primary disturbance of calcium, phosphorus, and magnesium metabolism. Arch Dis Child. 1973; 48: 99-108.

https://doi.org/10.1136/adc.48.2.99

PMid:4690525 PMCid:PMC1648180

7. Neonatal seizure disorders. Neurologic disorders in children. [Internet] Merck manual professional. Available from: http://www.merck.com/mmpe/sec 19/ch283html

8. Calciolari G, Perlman JM, Volpe JJ. Seizures in the intensive care unit of the 1980s. Types, etiologies, timing. Clin Pediatr. 1988; 27: 119-123.

https://doi.org/10.1177/000992288802700301

PMid:3342595

9. Kalra V. Neonatal seizure. In: Practical Pediatric Neurology. 1st ed., New Delhi: Arya Publications; 2002: 25-30.

10. Taksande AM, Vilhekar K, Jain M, Lakra M. Clinico-

biochemical profile of neonatal seizures.Pediatric oncall [serial online] 2005. Available from: http://www.Pediatric on call.com/For doctor/medical_original_articles/Neonatal_seizures org.

11. Shah GS, Sing MK, Budhathoki S, Kalakheti BK, Baral DD. Clinico-biochemical profile of Neonatal Seizure. J Nepal Pediatr Soc. 2008; 28(1): 7-9.

https://doi.org/10.3126/jnps.v28i1.1398

12. Rennie J M. Neonatal neurology. In: McIntosh N, Helms PJ, Smyth RL, editors. Forfar & Arneils Textbook of Pediatrics. 6th ed. Edinburgh, UK; 2003: 309-24.

13. Kuban KCK and Filiano J. Neonatal seizures. In: Cloherty JP, Stark AR, Ed. Manual of neonatal care.4th ed. Philadelphia; 1997: 493-505.

14. Ross AL, Lombroso C. Neonatal seizures state: A study of clinical, pathology and electrographic features in 137 full term babies with long term follow up. Pediatrics. 1970; 45: 404-425.

15. Soni A, Sabarawal AK, Amita K. Clinical profile of seizures in neonatal intensive case unit. In: Manual abstract of XXIII, Annual convention of national neonatology forum. 2003; P 43: 109-110.

16. Coen RW, Mc Cutchen CB, Warma D. Continuous monitoring of electroencephalogram following perinatal asphyxia. J. Pediatr. 1982; 100: 628-630.

https://doi.org/10.1016/S0022-3476(82)80773-7

17. Brown JK, Cockburn F and Forfur JO. Clinical and chemical correlate in convulsion of new born. Lancet. 1992; 1: 135-138.

https://doi.org/10.1016/S0140-6736(72)90694-0

18. Ronen GM, Penny S and Andrew W. The epidemiology of clinical neonatal seizures in new foundland- A population based study. J Pediatrics. 1999; 134: 71-75.

https://doi.org/10.1016/S0022-3476(99)70374-4

19. Mizrahi EM. Neonatal Seizures: Problems in diagnosis and classification. Epilepsia 1987; 28: 546-55.

https://doi.org/10.1111/j.1528-1157.1987.tb05757.x

PMid:3113930

20. Eriksson M, Zetterstrom R. Neonatal convulsion. Incidence and causes in Stockholm area. Acta Pediatr Scand. 1979; 68: 807-811.

https://doi.org/10.1111/j.1651-2227.1979.tb08216.x

PMid:539404

21. Painter MJ, Bergmann I and Cumrine P. Neonatal Seizures. Pediatr Clin North Am. 1986; 33(1): 91-105.

https://doi.org/10.1016/S0031-3955(16)34971-9

22. Holden KR, Mellitus D and Freeman JM. Neonatal Seizures I. Correlation of Prenatal and Perinatal events with outcome. Pediatrics. 1982; 70: 165-176.

23. Legido A, Clancy RR, Berman PH. Neurologic outcome after electroencephalographically proven neonatal seizures. Pediatrics. 1991; 88: 583-596.

24. Scher MS, Aso K, Beggarly ME, Hamid MY, Steppe DA and Painter MJ. Electrographic seizures in preterm and full-term neonates: clinical correlates, associated brain lesions, and risk for neurologic sequelae. Pediatrics. 1993; 91: 128-134.

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ORIGINAL ARTICLE OPEN ACCSESS

Morphology of Right Atrioventricular Valve -A Postmortem Study

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Abstract

Introduction: *Heart valves serve the important function of preventing backflow, or regurgitation, in the healthy heart. It is well known that cardiac valves can suffer from congenital and acquired disease. Most frequent acquired valvular abnormalities are stenoses of the aortic and mitral valves, which account for approximately 2/3rd of all valve disease. For the management of valvular disease morphology of right atrioventricular valve is essential.* ***Materials and Methods:*** *Study design was descriptive type of study. Place and period of study was Department of Anatomy, Sylhet MAG Osmani Medical College, Sylhet from July 2015 to June 2016.* ***Results:*** *Present study was performed on 70 post mortem human hearts of age ranging from 9 to 70 years. Human heart was collected from the unclaimed dead bodies autopsied in the department of Forensic medicine in Sylhet MAG Osmani Medical College during the study period fulfilling the inclusion criteria.* ***Conclusion:*** *The collected samples were divided into 3 groups depending on age. Group – A: (9 - 21 Years), Group - B: (22 - 41 Years), Group - C: (42 -70) Years. Each group was subdivided into two groups depending on their sex.*

Key words: *Atrioventricular valve, Heart.*

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Introduction:

The right atrioventricular or tricuspid valve guards the right AV orifice [1]. Through the right atrioventricular (tricuspid) orifice, the inflow part of the right ventricle receives blood from the right atrium ,located posterior to the body of the sternum at the level of the 4th and 5th intercostal space [2]. The right atrioventricular orifice is somewhat oval or circular in outlet and is oriented almost vertically. It measures on average 11.4 cm in circumference in males and 10.8 cm in females [1,3]. The A-V valves (the tricuspid and mitral valves) prevent backflow of blood from the ventricles to the atria during systole. These valves close and open passively. That is, they close when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure [4]. The atrioventricular valve is developed by local proliferations of mesenchymal tissue after the fusion of atrioventricular endocardial cushions [5,6]. Replacement of damaged cardiac valves with prostheses has now become a common and often life-saving mode of therapy. This advancement has undoubtedly called for an improved anatomical knowledge on the part of the surgeon. Besides for correction of cardiac abnormalities modern cardiac surgery demands precise methods of investigation to provide accurate anatomical details. It is only when such information is available that a precise diagnosis can be planned [7]. This sort of information is undoubtedly best gathered through experience in the living subject, secondly to that approach and clearly prerequisite to the study of representative specimen such as anatomical study through autopsy.

Materials and Methods:

This descriptive study was conducted in the Department of Anatomy, in collaboration with the Department of Forensic Medicine, Sylhet MAG Osmani Medical College, Sylhet during the period from July 2015 to June 2016. Seventy human postmortem hearts were collected from the dead bodies autopsied within 36 hours of death. Considerable signs of decomposition or decomposed dead body, any gross heart disease were excluded. The obtained Hearts were classified according to the age and sex. Particulars of dead body was collected from police inquest report and chalan. Heart was collected after standard procedure of autopsy. Unwanted tissues were cleared and heart was washed thoroughly with normal saline and was gently squeezed to remove the clotted blood from the cavity of the heart. Congenital anomalies of heart was excluded after dissection. Each specimen was duly tagged by a piece of waxed cloth bearing an identifying number which was considered as serial number. Then the specimen was fixed and preserved in 10% formalin.

Grouping and distribution of cases

The collected samples were divided into 3 groups depending on age.

Group - A: 9 - 21 Years.
Group - B: 22 - 41 Years.
Group - C: 42 -70 Years.

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Each group was subdivided into two groups depending on their sex. Parameter: Circumference of the right atrioventricular orifice.

Measurement of Parameter of the Right Atrioventricular Valve:

Measurement of right atrioventricular orifices: For the measurement of the circumference of the right atrioventricular orifices the annulus of each orifice was cut open and stretched flat. A metric scale and non-stretchable nylon thread was used for this purpose. The latter used on base (annulus) of the cusps and subsequently stretched and compared with the metric scale. The values was expressed in centimeter.

Results:

Distribution of circumference of right atrio-ventricular orifice among different age group.

The circumference of right atrio-ventricular orifice was 6.62 ± 1.80 cm in the age group-A (9-21 years), 9.02 ± 1.27 cm in the age group-B (22-41 years) and 8.73 ± 1.33 cm in the age group-C (42-70 years). There was significant difference of circumference of right atrio-ventricular orifice between age group-A and age group-B (p<0.001); and age group-A and age group-C (p<0.001); but no significant difference between age group-B and age group-C (p=0.504). Distribution of circumference of right atrio-ventricular orifice among different age group was shown in table I.

Table-I: Distribution of circumference of the Right atrio-ventricular orifice among different age group (n=70).

Group	n=70	Minimum cm	Maximum cm	Mean ±SD	P	Remark
A	20	6.62 ± 1.80	9.02 ± 1.27	6.62 ± 1.80	A vs B, p<0.001	Signifant between Group A and B and A and C
B	32			9.02 ± 1.27	A vs C, p<0.001	
C	18			8.73 ± 1.33	B vs C, p=0.504	Not significant between B and C

Data were expressed as mean ±SD n : number of specimen
Group – A : 9 - 21 Years Group – B : 22 - 41 Years
Group – C : 42 -70 Years SD : Standard deviation
P : probability value

Distribution of circumference of right atrio-ventricular orifice between male and female:

The circumference of right atrio-ventricular orifice was 8.56 ± 1.57 cm in male and 7.54 ± 2.05 cm in female. There was significant difference of circumference of right atrio-ventricular orifice of heart between male and female (t=2.266; p=0.027). Distribution of circumference of right atrio-ventricular orifice between male and female was shown in table II.

Table-II: Distribution of circumference of the right atrio-ventricular valve of heart between male and female (n=70).

Sex	n=70	Minimum cm	Maximum cm	Mean ±SD	t	p	Remark
Male	49	4.47±2.53	10.47±1.33	8.56 ± 1.57	2.266	0.027	Significantly greater in male
Female	21	2.03±1.03	8.56±1.54	7.54 ± 2.05			

Data were expressed as mean±SD n : number of specimen
SD : Standard deviation P : probability value

Discussion:

This study showed that the circumference of right atrio-ventricular orifice was 6.62 ± 1.80 cm in the age group-A (9-21 years), 9.02 ± 1.27 cm in the age group-B (22-41 years) and 8.73 ± 1.33 cm in the age group-C (42-70 years). There was significant difference of circumference of right atrio-ventricular orifice between age group-A and age group-B (p<0.001); and age group-A and age group-C (p<0.001); but no significant difference between age group-B and age group-C (p=0.504). The circumference of right atrio-ventricular orifice was 8.56 ± 1.57 cm in male and 7.54 ± 2.05 cm in female. There was significant difference of circumference of right atrio-ventricular orifice of heart between male and female (p=0.027).

Farzana[8] stated that the mean circumference of right atrioventricular orifice was 7 ± 1.57, 9.03 ± 1.15 and 9.51 ± 0.94 cm respectively. The mean difference of circumference of right atrioventricular orifice between Groups A and B, B and C and A and C was statistically significant at p<.05 level.

Begum[9] found that the mean circumference of the tricuspid orifice in male hearts was 9.24 ± 1.19 cm and in females was 9.35 ± 1.13 cm. Statistical analysis however, showed no significant difference (P>0.05) in the circumference of tricuspid orifice between males and females.

Yavuz et al[10] measured the mean circumference of the tricuspid orifice was 12.4 ± 1.1 cm in males and 11.8 ± 1.3 cm in females.

Mannan[11] measured the mean circumference of the tricuspid orifice and found 9.12 cm in males and 8.53 cm in females.

Datta[12] described that the right atrio-ventricular orifice is somewhat oval or circular in outline (depending upon the phase of the cardiac cycle) and the circumference of the opening, on an average, varies between 10 cm and 12 cm.

Standring et al[1] stated that circumference of the tricuspid orifice in males 11.4 cm and 10.8 cm in females.

The difference of the values of present study with the above cited literature is assumed to be due to racial cause.

Conclusion:

From the results a single set of conclusion could not drawn, but are expected to provide an idea about the circumference of the tricuspid orifice of the heart and their changes in relation to age and gender of Bangladeshi people.

Conflict of Interest: None.

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References:

1. Standring, S. Gray's Anatomy. 40th ed. Churchill Livingstone: Elsevier; 2008: 1245-5

2. Moore KL, Dalley AF, Agur AMR. Clinically Orientated Anatomy. 6th ed. Philadelphia: Lipincott Williams and Wilkins; 2010: 139.

3. McKinley M, O'Loughlin V. Human Anatomy. 3rd. New York: McGraw-Hill; 2012: 830-1.

4. Hall JE. Guyton and Hall Textbook of Medical Physiology. 12th eds. Philadelphia: Elsevier and Saunders; 2011: 103-9.

5. Sadler TW., editor. Langman's Medical Embryology. 12th ed. Philadelphia, Lippincott :Williams and Wilkins; 2012: 175.

6. Moore KL, Persaud TVN. The Developing Human: Clinically Oriented Embryology. 8th ed. Elsevier Saunders; 2010: 350.

7. Anderson JR, Zakkar M. Cardiac surgery. In: William N.S., Bulstrode, C.J.K. O'Connell, P.R., editors. Bailey and Love's Short Practice of Surgery. 26th ed. Boca Raton: Taylor & Francis; 2013: 823-49.

8. Farzana T. Anatomical Study on Post-Mortem Hearts with Its Vessels of Bangladeshi People (M Phil thesis). Dhaka: University of Dhaka; 2014.

9. Begum J. An Anatomical study of atrio-ventricular valves, muscular part of interventricular septum and papillary muscle of heart among the adult Bangladeshi people [Thesis]. Dhaka: University of Dhaka; 1996.

10. Yavuz KA, Govsa F, Ekin O, Boidak B, Ismail C. Structure of the human tricuspid valve leaflets and its chordae tendineae in unexpected death. Saudi Med J. 2004; 25(8): 1051-1058.

11. Mannan S. An anatomical study on post-mortem heart of Bangladeshi adult people (M Phil thesis). Dhaka: University of Dhaka; 2004.

12. Datta, A.K. Essentials of Human Anatomy. 9th ed. Kolkata: Current Books International. 2010; 1: 70.

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ORIGINAL ARTICLE

OPEN ACCSESS

Sensitivity of Alvarado Score among the Suspected Appendicitis Patients at a Tertiary Level Teaching Hospital

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Abstract

Introduction: *The aim of this study was to screening the Sensitivity of Alvarado score among the hospitalized suspected appendicitis patients at Dhaka National Medical College Hospital for determine the diagnostic accuracy.* ***Materials and Methods:*** *It was an observational type of descriptive study, conducted in the Dhaka National Medical College Hospital, Dhaka, during the study period of July 2015 to December 2015. The study was approved by the institutional ethical committee.* ***Results:*** *Most of the appendicitis patients belonged to the between 21-30 years which was 64 (32%). Male appendicitis patients (52%) are more than the female patients (48%). Majority of the patients (69%) complains pain occurs in the Right iliac fossa. The sensitivity of Alvarado scores was 81.60%, specificity 74.58%, accuracy 79.35%, positive and negative predictive values were 87.18% and 65.67% respectively.* ***Conclusion:*** *Alvarado score has more specificity.*

Keywords: *Alvarado score, Appendicitis patients.*

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Introduction:

Acute appendicitis is the most common cause of 'Acute Abdomen' in young adults. Appendicectomy is the treatment of choice and it is the most frequently performed urgent abdominal surgery and is often the first major procedure performed by a surgeon in training[1]. Multiple scoring system have been developed for the diagnosis of acute appendicitis among which Alvarado is the most commonly used scoring system[2]. However this scoring system was created in west and when applied in different environments, such as middle east and asia the sensitivity and specificity levels achieved very low[3,4]. According to the Alvarado score, if score 1-4: acute appendicitis very unlikely, Score 5-7: acute appendicitis probable, Score 8-10: acute appendicitis definite. The aim of this study was to screening the sensitivity of Alvarado score among the patient presented with right iliac fossa pain and who are suspected of acute appendicitis in Dhaka National Medical College Hospital for determine the diagnostic accuracy. The Alvarado score system are given below.

Table A: Alvarado appendicitis score system.

Date of assessment	
Time of assessment	
Symptoms	**Score**
Pain migration to RIF	1.0
Anorexia	1.0
Nausea and vomiting	1.0
Signs	
RIF tenderness	2.0
Rebound tenderness	1.0
Fever	1.0
Investigations	
Raised WBC count	2.0
Shift of WBC to left	1.0
Total	10

Total score is achieved by adding all the score for each category

1 = Score 8-10: acute appendicitis definite.

2 = Score 5-7: acute appendicitis probable.

3 = Score 1-4: acute appendicitis very unlikely.

Materials and Methods:

It was an observational type of descriptive study, conducted in the Dhaka National Medical College Hospital, Dhaka, during the study

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period of July 2015 to December 2015. The study was approved by the institutional ethical committee. To evaluate the sensitivity of Alvarado score among the suspected appendicitis patients, a data collection sheet was prepared. Total 200 case records were studied during the study period. The data was obtained from the hospitalized patients. All filled questionnaires were entered into the computer for subsequent analysis using SPSS method version 20.1.

Results:

Table I shows mean age of the patients were 35.83(±12.30) years, minimum age was 18 years and maximum age was 57 years. Maximum age group was between 21-30 years which was 64 (32%).

Table-I: Age group distribution of the study population (n=200).

Age in years	Frequency	Percentage (%)
<20 years	16	8%
21-30 years	64	32%
31-40 years	48	24%
41-50 years	30	15%
>50 years	42	21%
Total	200	100%
Mean±SD	35.83 (±12.30)	Range 18-57 years

According to figure 1, More than half (52%) were male patients and 48% were female patients. Male and female ratio was 1.08:1.

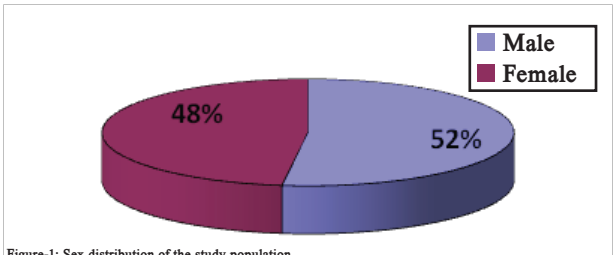

Figure-1: Sex distribution of the study population.

According to table II, in 29(14.5%) patients pain occurred at peri -umbilical region, 138(69%) patients pain occurred at Right iliac fossa and 33(16.5%) patients pain occurred at other place.

Table-II: Site of pain of the study population (n=200).

Site of pain	Frequency	Percentage (%)
Peri-umbilical region	29	14.5%
Right iliac fossa	138	69%
Other place	33	16.5%
Total	200	100%

According to table III, pathological findings 136(68%) were positive for acute appendicitis and 64(32%) were negative for acute appendicitis.

Table-III: Histo-pathological findings of the study population (n=200).

Histopathological findings	Frequency	Percentage (%)
Positive for acute appendicitis	136	68%
Negative for acute appendicitis	64	32%
Total	200	100%

According to table IV, Majority of the patients (63.5%) alvarado score ≥7.

Table-IV: Alvarado score of the study population (n=200).

Alvarado score	Frequency	Percentage (%)
<7	73	36.5%
≥7	127	63.5%
Total	200	100%

According to table V, sensitivity of Alvarado scores was 81.60%, specificity 74.58%, accuracy 79.35%, positive and negative predictive values were 87.18% and 65.67% respectively.

Table-V: Performance of the diagnostic test (n=200).

Parameter	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
Alvarado scores	81.60%	74.58%	87.18%	65.67%	79.35%

Discussion:

Acute appendicitis is one of the common causes of emergency surgery. Most of the patient presented with the right lower abdominal pain. But the diagnosis of acute appendicitis and decision to make surgery is often difficult. Because the history and presentation of these patients are variable and accurate diagnosis is not easy, but the delay in the diagnosis may cause fatal complications which in turn increase morbidity and mortality. In this present study showed mean age was 35.83 (±12.30) years, minimum age was 18 years and maximum age was 57 years. Maximum age group was between 21-30 years which was 64 (32%). Majority 52% were male and 48% were female, male: female ratio was 1.08:1. In study of Chong et al.[5] showed the mean age of the patients (92 male, 100 female) was 25.1 ± 12.7 years. In Ismail Alnjadat I, Baha Abdallah study [6] male to female ratio was 1.5:1 and mean age was 26.52 years. These results are consistent with other studies[7, 8]. In our study most of the pain occurs in right illac fossa (69%). Our study results are similar to the Nshuti et al. [9] study but the percentage is not same. In their study they stated that most of the pain occurs in right iliac fossa (95%). In this study showed sensitivity of Alvarado scores was 81.60%, specificity 74.58%, accuracy 79.35%, positive and negative predictive values were 87.18% and 65.67% respectively. Khan et al.[10] applied the Alvarado scoring system in an Asian population and only achieved a sensitivity and specificity of 59% and 23%, respectively, with a negative appendicectomy rate of 15.6%. The sensitivity of the Alvarado score achieved when applied in an oriental population, at the suggested cut-off threshold of 7.0, was similarly low at 50.6%, but achieved a high specificity of 94.5%[11]. However, this improved when the cut-off threshold was lowered to 6.0, with a sensitivity and specificity of 88.3% and 94.5%, respectively, suggesting a definite ethnic difference with regard to the Alvarado score[11]. Both the Alvarado and modified Alvarado scores lack parameters that have been shown to be important determinants in the diagnosis of acute appendicitis, such as age, gender and the duration of symptoms. Wani et al.[12] have shown that the sensitivity and specificity of the Alvarado scoring system vary with age, gender and duration of the symptoms.

Conclusion:

Alvarado score has more specificity. The Alvarado scoring systems vary with age, gender and duration of the symptoms. So it is not 100 percent accurate for all the patients.

Conflict of Interest: None.

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References:

1. Ashmawy. Evaluation of combined graded compression Ultrasonography with Alvarado score in the diagnosis of acute appendicitis. Alexandria Bulletin. 2006; 42(1): 29-31.

2. Kalan M, Talbot D, Cunliffe W J, Rich A J. Evaluation of the modified Alvarado score in the diagnosis of acute appendicitis: a prospective study. Ann R Coll Surg Engl. 1994; 76: 418-19.

3. Al-Hashemy AM, Seleem MI. Appraisal of the modified Alvarado score of the acute appendicitis in adults. Saudi Med J. 2004: 1229-31.

4. Khan I, Ur Rehman A. Application of Alvarado scoring system in the diagnosis of acute appendicitis. J Ayub Med Coll Abbottabad. 2005: 41-42.

5. Chong C F, Thien A, Mackie A J A, Tin A S, Tripathi S, Ahmad M A, et al. Comparison of RIPASA and Alvarado scores for the diagnosis of acute appendicitis. Singapore Med J. 2011: 34.

6. Ismail Alnjadat I, Abdallah B. Alvarado versus RIPASA score in diagnosing acute appendicitis. Rawal Medical Journal. 2013: 147-151.

7. Askarpour S, Pour FF, Rezaei SK. Accuracy of Diagnosis of Appendicitis and Its Relation to Perforation in Hospitalized Children in Surgery Department of Imam Khomeini Hospital-Ahwaz. Ann Pediatr Surg. 2008: 74-77.

8. Sulu B, Gunerhan Y, Palanci Y, Isler B, Caglayan K. Epidemiological and demographic features of appendicitis and influences of several environmental factors. Turk J Trauma Emerg Surg. 2010: 38-42.

9. Nshuti, R., Kruger, D., Luvhengo, E T. Clinical presentation of acute appendicitis in adults at the Chris Hani Baragwanath academic hospital. Int J Emerg Med. 2014; 7.

https://doi.org/10.1186/1865-1380-7-12

PMid:24533851 PMCid:PMC3938026

10. Khan I, Ur Rehman A. Application of alvarado scoring system in diagnosis of acute appendicitis. J Ayub Med Coll Abbottabad. 2005: 41-44.

11. Jang SO, Kim BS, Moon DJ. Application of alvarado score in patients with suspected appendicitis. Korean J Gastroenterol. 2008: 27-31.

12. Wani MM, Yousaf MN, Khan MA. Usefulness of the Alvarado scoring system with respect to age, sex and time of presentation, with regression analysis of individual parameters. Internet J Surg, 2007; Available at: www. ispub.com/ journal/the_ internet_journal_of_ surgery/ archive/ volume_11_ number_2 .html. Accessed July 1, 2008.

https://doi.org/10.5580/d56

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ORIGINAL ARTICLE OPEN ACCSESS

Renal Function Status in the Newborn with Perinatal Asphyxia

Tarun Kumar Roy*[1], Mahmood A Chowdhury Arzu[2], Wazir Ahmed[3], Didarul Alam[4], Sanjana Islam[5], Rabeya Sultana[6], Suvendu Ray[7]

Abstract

Introduction: *Renal involvement is frequent in neonates with perinatal asphyxia. It is correlated with the severity of neurological damage and degree of involvement depends upon the severity of asphyxia. To assess the status of renal function in new born suffering from prenatal asphyxia and to precise the relationship between severity cerebral damage and renal failure.* ***Materials and Methods:*** *A hospital based cross-sectional study was conducted in the department of Neonatology, Chattogram Maa O Shishu Medical College Hospital, Agrabad, Chattogram on 180 full-term neonates (150 cases and 30 control). The cases were categorized according to HIE Sarnat stages.* ***Results:*** *Among cases, 83(53.4%) were with HIE I, 53 (35.3%) were HIE II and 14(9.3%) were HIE III. The mean value of S. Creatinine was high in cases 1.64 ± 0.33 mg/dl vs 0.41 ± 0.09 mg/dl (p value <0.001) and it is highest in HIE stage III 1.85 ± 0.20 mg/dl (p value is <0.001). Mean Serum Potassium was high in cases 5.88 ± 0.59 mmol/L vs 3.99 ± 0.36 (p value <0.001) and the values are abnormally high in HIE stage III 6.25 ± 0.33 (p value <0.001). The mean value of FeNa was high in cases 2.44 ± 0.55 compared with control 1.06 ± 0.38 (p value <0.001) and it is higher in HIE stage III 2.72 ± 0.22 (p value is <0.001).* ***Conclusion:*** *Severity of renal impairment correlates well with the degree of HIE. HIE stage wise assessment of renal function status using serum creatinine level, S. potassium, FeNa, can be used to assess the outcome of perinatal asphyxia.*

Keywords: *Perinatal asphyxia, Hypoxic ischemic encephalopathy, Acute kidney injury.*

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Introduction:

Perinatal asphyxia is the most important preventable cause of cerebral injury in the neonatal period leading to very high neonatal mortality and morbidity in developing countries[1]. It occurs mostly during the first and second stage of labor[2] and is an eventually having far reaching consequences in the neonatal period. It can cause damage to almost every tissue and organ of the body; the most vulnerable ones are central nervous system (72%), followed by kidneys (42%), cardiovascular (29%), gastrointestinal tract (29%), and pulmonary (26%)[3,4,5]. It results in redistribution of blood flow towards the brain, heart and adrenals and away from kidneys, skin and the gastrointestinal tract to ensure adequate oxygen and substrate deliver to these vital organs[6,7]. Hypoperfusion with concomitant hypercapnia and acidosis contribute to these organ damage[8,9]. As kidneys are very sensitive to oxygen deprivation, renal insufficiency may occur within 24 hours of a hypoxic ischemic episode, which if prolonged, may even lead to irreversible cortical necrosis[10]. So, early recognition of renal failure is important in babies with HIE to facilitate appropriate fluid and electrolyte management as a stable biochemical milieu is vital.

A United Nations Children's Fund (UNICEF) report published on Tuesday said the under-five mortality rate in Bangladesh in 1990 was 144 per 1,000. But in 2015, the rate is 38 per 1,000 and the child mortality rate across the world was 53 percent, over the same timeframe. About two-third of this high mortality in Bangladesh is due to high perinatal mortality. And perinatal asphyxia constitutes a large portion of perinatal mortality. It is also a leading cause of admission to neonatal care services but in our country, previously no sufficient data is available on incidence of acute renal failure in neonates with perinatal asphyxia and on relationship between low Apgar score[10] and or the hypoxic ischemic encephalopathy grading

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and development of acute renal failure.

Materials and Methods:

This Cross-sectional study was conducted in the department of neonatology, Chattogram Maa-Shishu O General Hospital (CMSOGH) from January 2016 to June 2016. All Perinatal asphyxia patients admitted in the department of neonatology, Chattogram Maa-Shishu O General Hospital fulfilling the inclusion criteria were included in the study. Inborn and out born infants with perinatal asphyxia, with postnatal age between 72 – 96 hours were included as cases. Very sick neonates with perinatal asphyxia who required CPR for resuscitation and mechanical ventilation, Newborn with gestational age <34 weeks congenital anomalies were excluded from research work. Control was taken from babies of postnatal ward. They all were healthy, kept with mother in postnatal ward and age was between 72 to 96 hours.

• Reference values used in this study:

Serum Creatinine: Day 3-7: 14 – 86 (0.16 – 1.0 mg/dl)

Serum Electrolyte: S. Sodium: 131-144 mmol/L, S. Potassium: 3.2 – 5.7 mmol/L

Blood Urea: 0.7 – 4.6 mmol/L

Urinary Indices	Prerenal	Intrinsic Renal
Urine osmolality (mosmol/kg water)	>400	<400
Urine sodium (mEq/L)	31 ± 19	63 ± 35
Urine/Plasma creatinine ratio	29 ± 16	10 ± 4
FeNa (%)	<2.5	>2.5
RFI	<3.0	>3.0

Urinary indices in the Neonate used in the evaluation of acute kidney injury

- $\text{FeNa}\,\% = \frac{\text{Urine Na} \times \text{Serum Cr}}{\text{Serum Na} \times \text{Urine Cr}} \times 100$

To conduct the study ethical clearance was taken from the concerned authority of CMOSH with due procedure. A newborn with history of failure to initiate spontaneous breath immediately after birth and /or history of delayed cry or no cry at all after birth and followed by evidence of Hypoxic Ischemic Encephalopathy (HIE), fulfilling the inclusion and exclusion criteria were considered as case. In this way 150 cases were taken. The cases were categorized according to HIE Sarnat stages. The aim and objectives of the study along with its procedure were explained to the parents in easy, understandable and in local language. Then informed written consent was taken before data collection. Privacy and secrecy were maintained during the procedure. No financial cost was paid by the parents. Detailed history, careful physical examination was performed and case record form was filled in. Two milliliters venous blood was collected from the patient within 72 to 96 hours of age. Adequate amount of urine was also collected from a prefixed sterile plastic bag. Renal status was assessed by doing (1) Serum creatinine, (2) Serum Electrolytes, (3) Urinary electrolytes, (4) Urinary creatinine, (5) Fractional excretion of sodium; Investigations were done in Chattogram Ma-O-Shishu hospital laboratory by auto analyzer. All data were collected in individual structured data collection form Chattogram Ma-O-Shishu hospital. Then results were prepared. Data was analyzed by using SPSS version 15.0 (SPSS Inc. Chicago, USA) statistical software employing appropriate statistical tests like unpaired Student's "t" test, mean, SD, 95% Confidence limit, Standard error and their "P" values were obtained to see the statistical significance. P value < 0.05 was considered as significant.

Results and Observation:

Table - I: Distribution of the study subjects (n = 180).

Study Groups	Frequency	Percentage (%)
Case	150	83.3
Control	30	16.7
Total	180	100.0

*** PNA = Perinatal Asphyxia**

Cases	Frequency	Percentage (%)
PNA with HIE stage I	83	55.4
PNA with HIE stage II	53	35.3
PNA with HIE stage III	14	9.3
Total	150	100.0

*** PNA = Perinatal Asphyxia**

Table - II: Statistics of plasma creatinine among the study subjects (n = 180).

Plasma Creatinine (mg/dl)	N	Mean	± SD	Median	Range	P value*
Case (Newborn with PNA)	150	1.64	0.33	1.62	0.50 – 2.90	P < 0.001 Highly
Control	30	0.41	0.09	0.39	0.21 – 0.61	Significant
Total	180	1.43	0.55	1.56	0.21 – 2.90	* t-test

*** PNA = Perinatal Asphyxia**

The mean value of S. Creatinine of case and control are 1.64 mg/dl and 0.41 mg/dl respectively which is statistically significant (P <0.001).

Table - III: Plasma creatinine among the case (with t-test and ANOVA significance) (n = 150).

P. Creatinine	Stages			p value*
	Stage I	Stage II	Stage III	
Normal	49 (59.0)	27 (50.9)	6 (42.9)	0.422
Abnormal	34 (41.0)	26 (49.1)	8 (57.1)	
Total	**83 (100.0)**	**53 (100.0)**	**14 (100.0)**	

*Chi square test was done to measure the level of significance. Figure within parentheses indicates in percentage

Plasma Creatinine (mg/dl)	N	Mean	± SD	Median	Range	*P value**
PNA stage 1	83	1.13	0.28	1.14	0.50 – 1.6	P < 0.001
PNA stage 2	53	1.47	0.37	1.38	0.90 – 2.10	*Highly*
PNA stage 3	14	1.85	0.20	1.72	1.50 – 2.90	*Significant*
Total	150	1.64	0.33	1.62	0.50 – 2.90	* ANOVA

*** PNA = Perinatal Asphyxia**

Among the case, normal value and abnormal value of S. creatinine in HIE stage I, stage II and stage III are not statistically significant. But the mean value of the cases of HIE stage I is 1.13 ± 0.28 mg/dl, stage II is 1.14 ± 0.37 mg/dl and in stage III is 1.84 ± 0.20 mg/dl, that is significant.

Table–IV: Statistics of FeNa among the study subjects (n=180).

	FeNa (%)	N	Mean	± SD	Median	Range	*P value**
Case (Newborn with PNA)		150	2.44	0.55	2.49	1.11 – 4.31	P < 0.001 *Highly Significant*
Control		30	1.06	0.38	1.04	0.51 – 1.77	
	TOTAL	180	2.21	0.73	2.40	0.51 – 4.31	* t-test

* PNA = Perinatal Asphyxia; FeNa = Fractionated Excretion of Sodium

This table shows the mean value of Fractional Excretion of Sodium (FeNa) in case is 2.44 and in control is 1.06 which is statistically significant (P value is <0.001).

Table -V: Distribution of FeNa by stages (with t-test and ANOVA significance).

FeNa	Stages			p value*
	Stage I	Stage II	Stage III	
Normal	57 (68.7)	22 (41.5)	1 (7.1)	0.001
Abnormal	26 (31.3)	31 (58.5)	13 (92.9)	
Total	**83 (100.0)**	**53 (100.0)**	**14 (100.0)**	

*Chi square test was done to measure the level of significance.

Figure within parentheses indicates in percentage.

FeNa (%)	N	Mean	± SD	Median	Range	P value*
PNA stage 1	83	2.25	0.52	2.39	1.11 – 3.74	P < 0.001
PNA stage 2	53	2.65	0.55	2.62	1.38 – 4.31	*Highly Significant*
PNA stage 3	14	2.72	0.22	2.73	2.24 – 3.12	
Total	150	2.44	0.55	2.49	1.11 – 4.31	* ANOVA

* PNA = Perinatal Asphyxia; FeNa = Fractionated Excretion of Sodium

This table shows normal and abnormal values of FeNa in stage I; stage II and stage III are significant statistically and their mean values are also statistically significant.

Discussion:

Perinatal asphyxia is one of the important causes of neonatal morbidity and mortality in Bangladesh (BDSH 2005). The kidneys are the commonest organs to be involved in perinatal asphyxia and it is very sensitive to ischemic damage (Aurora and Snyder 2004 & Talos 1996). So, the renal function assessment in perinatal asphyxia is essential for accurate management of metabolic derangement particularly fluid, electrolytes and acid base imbalance resulting from renal function impairment. For early prediction of renal injury, markers of renal injury (i, e. S. Electrolytes, S. Creatinine, FeNa, and RFI) are more sensitive and specific in the determination of indices of renal function (Willis et al. 1997).

Traditionally, assessment of perinatal asphyxia has relied on a combination of clinical observations such as Apgar score and measurement of serum creatinine level. There was weakness in such methods because Apgar score may be influenced by metabolic and chromosomal disorders other than perinatal asphyxia. So, determination of more sensitive and specific markers of renal tubular dysfunction of renal injury in perinatal asphyxia (Willis et al. 1997) is essential. In this study 180 neonates were enrolled; among them 150 patients were cases and 30 patients were control. Among the cases, on the basis of clinical status, 83 (55%) neonates were assigned as HIE stage I, 53 (35%) neonates were assigned as HIE stage II and 14 (9%) neonates were assigned as HIE III.

Among the cases, numbers of male newborns were higher than female newborns. Presenting features coma, convulsion and hyper alert were predominant features in HIE stage III, stage II and stage I respectively. In this study it was found that perinatal asphyxia is more common in male sex than female sex (53% Vs 47%), People of rural area than urban (62.7% Vs 37.3%), Home delivery than hospital delivery (58% Vs 42%) and NVD than LUCS (65.3 Vs 34.7). The high incidence of PNA in hospital delivery and by LUCS is because from rural area mothers came to the hospital with complication and emergency delivery was done by LUCS to save mother's life and to secure the baby.

In my study among 150 cases 55% were HIE stage I, 35% were HIE stage II and 9% were HIE stage III. It is near to the findings done by D Saha, MAH Hollah, S Afroz, M Banerjee, TH Khan and CK Saha in Dhaka Medical College in January 2012 to January 2013, where among 102 cases 30.4% had mild asphyxia, 48% moderate and 21.6% had severe asphyxia. In my study, the discrepancy is higher percentage of stage I is most possible due to higher incidence of PNA in rural areas of the country and also difference in admission protocol of PNA patients in different institutions. In my study, in HIE stage I, among 83 patients 49 (59%) patients have normal S. creatinine and 34 (41%) patients have abnormally high S. creatinine. Whereas, in stage II, among 53 patients 27 (50.9%) patients have normal S. creatinine and 26 (49.1%) patients have abnormally high S. creatinine and in stage III, among 14 patients 6 (42.9%) patients have normal S. creatinine and 8 (57.1%) patients have abnormally high S. creatinine. Mean value of S. creatinine in all stages is 1.64 ± o.33 mg/dl. This value is consistent with Dr. Khalilur Rahaman study of Dhaka Shishu hospital 2007, where Serum creatinine values were significantly higher in asphyxiated neonates as compared to the control group. It is also consistent with Girish Gopal) and Group II at all, (p=0.0001).

In this study in stage I, 57 (68.7%) baby has normal FeNa and 26 (31.3%) baby has abnormally high FeNa. For stage II, 22 (41.5%) baby has normal FeNa and 31 (58.5%) baby has abnormally high FeNa and for stage III, only 1 (7.1%) baby has normal FeNa and 13 (92.9%) baby has abnormally high FeNa. There P value is 0.001, which is statistically highly significant.

Tubular dysfunction was confirmed by the fractional excretion of sodium, which was grossly abnormal in group 1 (mean 31 9%) mildly increased in group 2 (1-7%), and normal in group 3 (0-3%). That in control infants was below 1% and in sodium balance studies the fractional excretion of sodium in healthy infants of similar gestational age with a sodium intake of 2-5 mmol/kg/day has been reported to be normally less than 1%. 17 (Prediction of acute renal failure after birth asphyxia D S Roberts, G B Haycock, R N Dalton, C Turner, P Tomlinson, L Stimmler, J W Scopes).

In my study, In stage I, Mean ± SD of FeNa value is 2.25 ± 0.52, for Stage II, Mean ± SD value of FeNa is 2.65 ± 0.55 and for stage III, Mean ± SD of FeNa value is 2.72 ± 0.22 and "p" value 0.001 which is statistically highly significant. FeNa levels were higher in neonates with HIE II and III than in the control group while it was comparable in babies of HIE O and I stage with that of control babies. (Renal Failure in Asphyxiated Neonates B.D. Gupta, Pramod Sharma, Jyoti Bagla, Manish Parakh and J.P. Soni).

The high values of the FeNa signify that this AKI is intrinsic in nature.

Conclusion:

To evaluate the renal function impairment in asphyxiated infant some investigations are usually done. These are S. creatinine, S. electrolytes. But to see the extent of injury we need to do some additional investigations like Fractional excretion of sodium (FeNa). It is useful to find out specific AKI to get better outcome.

Conflict of Interest: None.

Acknowledgement:

It is a great pleasure on my part to express the best regards and gratefulness to the Department of Paediatrics, Chattogram Maa O Shishu Medical College and Hospital, Agrabad, Chattogram for completion of this research. I express my indebtedness to all my patients – living and dead, their parents who contributed in no small way to this research, but for whom this entire exercise would have been unimaginable.

References:

1. Shireen N, Nahar N, Mollah AH. Risk Factors and Short-Term Outcome of Birth Asphyxiated Babies in Dhaka Medical College Hospital. Bangladesh J Child Heal. 1970; 33(3 SE-Original Articles).

https://doi.org/10.3329/bjch.v33i3.5688

2. Roberts G. Manual of Neonatal Care. 1987; 15.

https://doi.org/10.1016/0378-3782(87)90024-7

3. Gomella TL, Cunningham MD, Eyal FG, Tuttle DJ. Neonatology: management, procedures, on-call problems, diseases and drugs. 2013.

4. Shah P, Riphagen S, Beyene J, Perlman M. Multiorgan dysfunction in infants with post-asphyxial hypoxic-ischaemic encephalopathy. Arch Dis Childhood-Fetal Neonatal Ed. 2004; 89(2): F152-F155.

https://doi.org/10.1136/adc.2002.023093

PMid:14977901 PMCid:PMC1756028

5. Martín-Ancel A, García-Alix A, Cabañas FGF, Burgueros M, Quero J. Multiple organ involvement in perinatal asphyxia. J Pediatr. 1995; 127(5): 786-793.

https://doi.org/10.1016/S0022-3476(95)70174-5

6. Kliegmassn RM, Stanton B, Geme JS, Schor NF, Behrman RE. Nelson textbook of pediatrics. 2015.

7. Covey M V, Levison SW. Pathophysiology of perinatal hypoxia-ischemia and the prospects for repair from endogenous and exogenous stem cells. Neoreviews. 2006; 7(7): e353-e362.

https://doi.org/10.1542/neo.7-7-e353

8. McIntosh N, Helms P, Smyth R, Logan S. Forfar and Arneil's Textbook of Paediatics. Churchill Livingstone; 2008.

9. Perlman JM, Tack ED, Martin T, Shackelford G, Amon E. Acute systemic organ injury in term infants after asphyxia. Am J Dis Child. 1989; 143(5): 617-620.

https://doi.org/10.1097/00132582-198910000-00022

https://doi.org/10.1001/archpedi.1989.02150170119037

PMid:2718998

10. Apgar V. A proposal for a new method of evaluation of the newborn. Class Pap Crit Care. 1952; 32(449): 97.

DOI: https://doi.org/10.3329/medtoday.v32i1.44827

ORIGINAL ARTICLE OPEN ACCSESS

Psychiatric Morbidity among the Patients Attended in a Psychiatric OPD in North East Part of Tertiary Level Hospital of Bangladesh

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Abstract

Introduction: *Mental health problem is a major public health issue in the world across the developed and developing countries. However, data in most of the developing countries including Bangladesh are scarce. In Bangladesh, socio-political situation is insecure and unstable with poverty and vulnerable to natural disaster which causes psychiatric morbidity. The pattern of psychiatric morbidity attended in OPD is quite different from that in government hospital. This study was aimed to assess the diagnostic pattern of psychiatric morbidity among the attended patients in a out patient department in a medical college.* ***Materials and Methods:*** *The study was carried out in a OPD which is situated in 500 bedded private medical college in the sylhet city. All the information including longitudinal histories of patients was recorded in files and the diagnosis was confirmed by psychiatrist. All information notes were recorded in register. Socio -demographic parameters and family history of mental illness were collected from the record file of individual patient.* **Results:** *Among 304 patients 184 (60.53%) were males and 120 (36.47%) were females. More than 50% of patients were in the age group of 18 to 37 years. Most common psychiatric disorders were schizophrenia and other psychotic disorders (39.4%), mood disorder (18.75%), borderline personality disorder (3.6%), conduct disorder (2.3), somatoform disorder (1.6%), anxiety disorder (0.7%), organic psychiatric disorder (2%), impulse control disorder (1.3%) and adjustment disorder (0.7%).* ***Conclusion:*** *Major forms of psychiatric disorders are common both in urban and rural areas of Bangladesh.*

Keywords: *Psychiatric morbidity.*

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Introduction:

Psychiatric morbidity is a major public health problem in the World across developed and the developing countries. Today mental health and mental illnesses are key public health issues. A large number of people worldwide suffer from mental disorders. According to World Health Organization at least 40 million people in the world suffer from mental disorders such as schizophrenia and dementia[1-4].

Bangladesh is a densely populated area where prevalence of psychiatric illness is not less than that of any other country in the world. A study showed that 29% of patients attending general practice were suffering from functional disorder and 6% from both functional and organic disorder. The same study demonstrated that 47% patients were suffering from neurotic disorder, 37% from psychosomatic disorder, 10% from affective disorder, 1.44% from schizophrenia, 2.88% from substance use disorder and 2% organic psychiatric syndrome[5].

Another study in Dasherkandi, a village nearby Dhaka city indicated that 6.52% people had been suffering from psychiatric illnesses[6].

Still now maximum people are out of modern treatment facilities due to poor economic condition, prevailing superstition, stigma on mental patients and lack of education and knowledge about scientific method of treatment of mental illness. Study conducted in Outpatient department of National Institute of Mental Health (NIMH), Dhaka revealed that 37.4% of patients were suffering from schizophrenia and schizophrenia like psychotic disorders, 16.14% from anxiety disorders, 11.19% from Major Depressive disorder, 8.95% from Bipolar mood disorder, 7.66 % from substance related disorder, 6.60% from somatoform disorder, 4.12% from mental retardation and [7].88% from other disorders[7]. The main objective of the present study was to observe the types of the psychiatric diagnoses among the admitted patients in a private hospital in Dhaka city, to see the relationship of psychiatric disorders with some socio-demographic parameters and also to observe the relationship between the family history of psychiatric illness and different types of psychiatric disorders.

Materials and Methods:

The study was carried out in a OPD of private medical college in sylhet city. It is a 500 bedded medical college hospital. Having psychiatric out patient department. Most of the patients came from Sylhet city & nearest thana. All the information about the patients including their thorough histories was recorded in files. Patients

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were diagnosed by the consultant psychiatrist Three hundred and four patients were admitted here throughout the year of 2018 from January to December. They were diagnosed according to Diagnostic and statistical manual for Mental disorder criteria by the Psychiatrist[8]. Necessary informations regarding patients were collected from record files. Data were processed and analyzed manually following the simple descriptive statistical procedure.

Results:

Total three hundred and four patients attended in a psychiatric OPD in a medical college of sylhet city during the period of January to December in the year 2018 were included in the study within the age group of 10 to 55 years. Out of 304 patients, 184 (60.53%) were male and 120 (36.47%) were female. 135 patients (44.4%) were married, 158 patients (50.66%) were unmarried and 15 (4.93%) were divorcee. 92.76% were muslims, 4.60% were Hindu and 2.63% were Christians (Table I). Regarding occupational status most of the patients (30.2%) were unemployed, followed by students (23.7%). Eighteen to twenty eight years of age group had more psychiatric disorder (42%), which was nearer to the finding of other study[9]. As the study was carried out in a private clinic almost all the patients belonged to medium to high social class (monthly income >10,000 Taka). Results showed that most of the patients were educated, 19% completed graduation, and 56.6% completed Higher Secondary certificate examinations (Table III). Out of three hundred and four patients 39.4% were suffering from schizophrenia and other psychotic disorders, 29.6% substance related disorder, 12.17% from bipolar mood disorder, 6.58% from major depressive disorder, 3.6% from borderline personality disorder, conduct disorder 2.3%, organic psychiatric disorder 2%, somatoform disorder 1.6%, impulse control disorder 1.3% and others 1.4%.

Table-I: Distribution of patients by sex, age group, religion and marital status.

Sex	Number	Percentage (%)
Male	184	60.53%
Female	120	39.47%
Age group		
< 18 years	23	7.6%
18-27 years	128	42%
28-37 years	107	35%
38-47 years	32	11%
48 years and above	14	4.6%
Residence		
Rural	90	29.6%
Urban	214	70.4%
Marital status		
Married	135	44.4%
Unmarried	154	50.66%
Divorcee	15	4.93%
Religion		
Islam	282	92.76%
Hinduism	14	4.60%
Christians	8	2.63%

Table-II: Distribution of patients by occupational status

Occupational status	Number	Percentage (%)
Business	65	21.4%
Student	72	23.7%
Service	29	9.6%
House wife	43	14.1%
Unemployedtg	92	30.2%
Farmer	3	1.0%

Table-III: Distribution of patients by their educational status

Educational status	Number	Percentage (%)
Primary	25	8.22%
Secondary	49	16.11%
SSC/HSC	172	56.6%
Graduate	58	19%

Table- IV: Types of psychiatric disorder among the admitted patients.

Types	Total number	Percentage (%)
Schizophrenia and other psychotic disorders	120	39.4%
Substance related disorders	90	29.6%
Bipolar mood disorder	37	12.17%
Major depressive disorder	20	6.58%
Borderline personality disorder	11	3.6%
Conduct disorder	7	2.3%
Somatoform disorder	5	1.6%
Organic psychiatric disorder	6	2%
Anxiety disorder	2	0.7%
Impulse control disorder	4	1.3%
Adjustment disorder	2	0.7%

Table-V: Distribution of different psychiatric disorders by sex.

Type of disorder	Male	%	Female	%
Schizophrenia and other psychotic disorders	77	25.3	43	14.1%
Substance related disorders	87	28.6	3	1%
Bipolar mood disorder	26	9.0	11	3.6%
Major depressive disorder	7	2.3	13	4.3%
Borderline personality disorder	3	1	8	2.6%
Conduct disorder	5	1.6	2	0.7%
Somatoform disorder	0	0	5	1.6%
Alzheimer's disease	2	0.7	0	0%
Postpartum psychosis	0	0	4	1.3%
Anxiety disorder	0	0	2	0.7%
Impulse control disorder	3	1	1	0.3%
Adjustment disorder	0	0	2	0.7%

Table-VI: Distribution of patients by family history of mental illness.

Family history of mental illness	Number	Psychiatric disorders	Number	Percentage (%)	Total
		Schizophrenia and other psychotic disorders	52	17.1%	
Present				7%	26.3
	80	Bipolar mood disorder	21		
		Substance related disorders	7	2.3%	
Absent	224				73.7

Discussion:

Schizophrenia and psychotic disorders were the commonest psychiatric disorders attending psychiatric OPD (39.4%). The study showed that next to schizophrenia was substance related disorder (29.6%). Substance use disorder is a rising problem of present day and a serious threat to our

social integrity and cohesion. A significant number of our young generation has been abusing illicit drugs and substances. Present study revealed that drugs use was high among the age group of 18 to 37 years, similar to other study. Among ninety cases of substance related disorder three were female and rests were males. Less access to narcotics to female abusers may justify less prevalence of substance use disorder among females. This finding was consistent to other study[6]. Next to substance related disorder 12.17% patients of bipolar mood disorder were admitted and more among males. Present study also showed patients of anxiety disorders (0.7%) were admitted less frequently as majority of them were treated in the out patient department[6]. Study revealed that major depressive disorder (6.58%), somatoform disorder (1.6%), anxiety disorder (0.7%) were more among females probably because of stressful life events, the effects of child birth and behavioral model of learned helplessness[5,7]. Borderline personality disorder was present in 3.6% of patients and also more among females[9]. Conduct disorder was common among boys (1.6%) than in girls (0.7%)[10].

As the study place was in the sylhet city, most of the patients (70.4%) were from urban background and from rural area 29.6% patients. Psychiatric morbidity was higher among urban people. Because these people are facing the daily life stresses and thereby more vulnerable to psychiatric illness. In this study a substantial number of patients (30.2%) were unemployed. This could be due to presence of psychiatric disorders[11]. Next to unemployment psychiatric morbidity appeared to be higher among students (23.7%), who were mostly of adolescents and of early adulthood and thereby were most vulnerable for most of the psychiatric disorders. Findings were consistent to other study[6]. There are enough evidences that psychiatric disorders particularly major psychiatric disorders have substantial contribution of genetic hereditability in their causation. One of our aim was also to estimate the relationship between positive family history of mental illness and major psychiatric disorders. Study showed positive family history for mental disorder was in 26.3% cases and it was highest (17.1%) among schizophrenia and schizophrenia like disorder. Next to schizophrenia for bipolar mood disorder it was about 7% followed by 2.3% in substance related disorder. Finding was consistent to other study[12].

Conclusion:

Psychiatric disorders are common in both rural and urban areas, which create hazards in personal, occupational or social level. Countrywide advertisement to increase people's awareness, co-operation of the Government and efficiency and commitment of service providers will be needed. Awareness about psychiatric illness is gradually increasing day by day among the people of Bangladesh. So, the number of patients seeking treatment is also increasing. To meet the need of the people the number of mental health professionals and facilities for mental health services are needed to be increased in government level as well as private sector.

Conflicts of Interest: None.

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References:

1. Firoz AHM, Karim ME, Alam MF, Rahman AHM, Zaman MN, Chandra V. Community Based Multicentric Service Oriented Research on Mental Illness with focus on Prevalence, Medical Care, Awareness and Attitude towards Mental Illness in Bangladesh. WHO published data, 2003-2005. Bang J Psychiatry. 2006; 20 (1): 9-32.

2. Hamid MA, Chowdhury S, Mohit MA. Comparative Study of Patients in Different Out patient Department(OPD) and the Pattern of Psychiatric Disorder in a district hospital. Bang J Psychiatry. 2003; 17 (2): 5-12.

3. Alam MN. Psychiatric Morbidity in General Practices. Bang Med Res Coun Bull. 1981; 4(1) 22-39.

4. Chowdhury AKMN, Alam MN, Ali SMK. Dasherkandi Project Studies: demography, morbidity and mortality in a rural community of Bangladesh. Bang Med Res Coun Bull. 1981; 4 (1): 22-39.

5. Mohit MA. Diagnosis of Patients Attending Out Patient Department (OPD) of NIMH. Bang J Psychiatry. June 2001; 15 (1): 5-12.

6. Rahman F, Sabeka MM, Karim ME. Psychiatric co-morbidity and Sexual Dysfunction in Substance Use Disorder. Bang J Psychiatry. 2003; 17 (2): 14-21.

https://doi.org/10.1023/A:1015267826608

PMid:12046636

7. Gelder M, Gath D, Mayou R, Cowen P. Mood Disorder in Gelder M, Gath D, Mayou R, Cowen P (eds). Oxford Text Book of Psychiatry. 5th ed. Oxford. 2005: 290-294.

8. Diagnostic and Statistical Manual for Menbtal Disorders. American Psychiatric Association. 4th ed. NewYork: USA; 1994.

9. Kaplan HI, Sadock BJ. Synopsis of Psychiatry. Williams and Wilkins, PA. 2003;809 .

10. Goodman R, Scoll S. Child and Adolescent Psychiatry. Blackwell Publishing ltd. 2nd edn. UK: 2005; 64.

11. Meyer R. In: Psychopathology and Addictive disorders. NY: The Guildford Press; 1986: 3-6.

12. Islam TM, Ahmed HU, Uddin JMM, Rahman F, Firoz AHM. Relationship between Positive Family History of Mental Illness and Major Psychiatric Disorder-A study in teaching hospital. Bang J Psychiatry. 2006; 20 (2): 50-56.

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ORIGINAL ARTICLE OPEN ACCSESS

Effect of Oral Zinc Sulfate on Burning Mouth Syndrome

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Abstract

Introduction: *Burning Mouth Syndrome (BMS) is a common condition having a severe burning sensation in the mouth, mainly affects menopausal women and have no definitive treatment so far. The main aim of this study was to evaluate the efficacy of zinc supplement in improving the symptoms of burning mouth syndrome.* ***Materials and Methods:*** *70 patients were enrolled in this study and were randomly divided in 2 groups. The patients in the intervention group received triamcinolone acetonide topical past with oral zinc sulfate therapy twice daily that continued for 3 months and the patients in the control group receivedtriamcinolone acetonide topical past only.* ***Results:*** *There was a significant difference (P = 0.001) in the severity of burning sensation in the two groups after the intervention.* ***Conclusion:*** *Zinc supplement might lessen the intensity of burning mouth syndrome in patients with zinc deficiency.*

Keywords: *Zinc, Burning Mouth Syndrome, Pain.*

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Introduction:

Burning mouth syndrome (BMS) is a chronic pain disorder characterized by burning, stinging, and/or itching of the oral cavity in the absence[1-2]. Burning sensation usually felt in the normal oral mucosa, especially on the tongue, which affects other mucosal surfaces, including the lips, buccal mucosa and the floor of the mouth[3].The tongue is found to be the most common location for burning sensation in the oral cavity. BMS can be accompanied by Dysgeusia (distortion in sense of taste), Glossodynia (painful tongue), Glossopyrosis (burning tongue) & Xerostomia (dry mouth the symptoms include a burning sensation, occasional pain, taste disturbances. Burning sensation is mostly reported in postmenopausal women[4]. The most commonly affected area is tip of the tongue and also affects the lips, gingiva and palate. The condition is extremely rare in patients under 30 years and never been reported in children and adolescence[5]. The exact etiology of BMS is unknown. Although there is no definitive cause of primary BMS, there are numerous potential secondary causes of the burning mouth syndrome. Several factors play an important role in the etiology of BMS. These are grossly classified to local, systemic and psychological factors[6]. After Iron, Zinc is the 2nd most important element of the body, which enhances growth and development. Though zinc deficiency has been reported to be related to BMS in the immune system, the beneficial effects of zinc replacement therapy has not been evaluated in patients with BMS. A study conducted by Cho in 2010 concluded that 26.8% of patients with BMS exhibited zinc deficiency and zinc replacement therapy experienced a decrease in burning sensation[7]. A burning sensation may also occur in other oral diseases like lichen planus and geographic tongue[8]. Burning mouth syndrome is associated with nutritional deficiencies including vitamin B1, B2, B6 and B12 deficiencies. Though, some studies have shown that zinc deficiency can also lead to BMS. Recent diagnostic criteria include continuous everyday pain in the oral cavity in association with normal oral mucosa after excluding other local and systemic diseases[9]. Metabolic endocrine and nutritional disorders may also lead to BMS. Some nutritional disorders, such as deficiencies in iron, zinc and vitamin B group, especially vitamin B12[10-11]. The early diagnosis of BMS is based on the exclusion of etiologic factors. Significant clinical recovery from BMS is possible only when systemic, local and psychological factors are treated or their eliminated[12].

Objectives

This study was conducted to evaluate the effect of zinc replacement therapy on BMS symptoms.

Materials and Methods:

This clinical trial study was conducted in oral and maxillofacial surgery department, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka between January 2015 and August 2016. Patients with BMS and who had zinc deficiency in laboratory evaluations, were included in the study. We performed a complete blood count (CBC) and serum zinc. Burning sensation in all or some parts of the oral cavity with or without symptoms subjects were divided to case and control groups . All the study population were examined before treatment and the burning severity was evaluated based on numeric rating scale (NRS), with '0' indicating there is absent of

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burning sensation and '10' indicating the most severe burning sensation experienced by the study subject. Study subject was asked to determine the severity of their burning sensation from 0 to 10. The patients in the case group were treated with zinc sulphate were administered twice daily and the zinc therapy was continued for three months. In the control group placebo were administered two times daily for three months. After three months, burning sensation severity was evaluated and measured. The serum zinc levels were determined and if these levels were not normal, treatment with zinc sulfate capsules continued for another month. After this period, the subjects were evaluated again and burning sensation severity was determined. After the study, the control group patients also received zinc replacement therapy. Data were analyzed with the SPSS software. The t-test was used to evaluate the means of burning sensation scores and serum zinc levels of the subjects.

Results:

In the present study, 70 patients with BMS, who had zinc deficiency, were evaluated, with 35 subjects in the case and 35 in the control groups. In the case group, there were 32 females and 3 males; in the control group, there were 30 females and 5 males. The mean age of the subjects were 43 ± 5.02 and 45.6 ± 7 years in the case and control groups, respectively, with no statistically significant differences between the two groups. The mean durations of BMS were 2±0.50 and 3.1±.54 years in the case and control groups, respectively, with no statistically significant differences between the two groups . Before intervention there was no difference in sensation severity and serum zinc level between the groups. At the end of the study there was a significant difference in sensation severity and serum zinc level between the two groups.

Table-I: Severities of Burning Sensation and Serum Zinc Levels Before and After Treatment (n=30).

	Case (n=35) n (%)	Control (n=35) n (%)	p-value
Burning sensation before the intervention	5.6 ± 1.3	4.9 ± 1.1	0.05
Burning sensation after the intervention	1.4 ± 0.9	4.9 ± 0.69	0.001
Serum zinc level before the intervention, g/dl	60 ± 10.5	62 ± 8.05	0.4
Serum zinc level after the intervention, g/dl	83 ± 0.92	65 ± 2.38	0.008

t-test was used to evaluate the means of burning sensation scores and serum zinc levels of the subjects

Discussion:

The results of this study showed that zinc supplementation lessen the severity of burning sensation in patients with BMS. Although the short term follow up studies show potential symptomatic improvement with treatment in patients with BMS, the long-term outcomes for BMS remain unclear. There is an important role of Zinc in the growth and development of the immune system and the neurologic functions. Zinc deficiency also affect the immune system and the repair processes of the mucosa and may cause BMS. Another study conducted by Maragou P et al showed lower serum levels of zinc in patients with BMS compared to healthy subjects[13]. Pekiner conducted a study in 2009 in 30 BMS patients and 30 healthy subjects and compared salivary levels of magnesium, zinc and copper among them and reported there were no significant differences between the two groups4. In a study of Cho et al. in 2010, 20 rats were divided to two groups of 10, where one group received a zinc deficient diet and the other group received a standard diet. After 20 weeks, hyperkeratosis and an increase in mitotic activity were observed in the dorsum area of the tongue of rats receiving the Zn-deficient diet. It is not possible to compare the results of the present study with those of other studies from a clinical point of view due to the limited number of studies on BMS serum zinc levels.

Conclusion:

The results of the present study concluded that zinc supplement reduces the symptom in patients with BMS.

Conflict of Interest: None.

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References:

1. Netto FO, Diniz IM, Grossmann SM, de Abreu MH, do Carmo MA, Aguiar MC. Risk factors in burning mouth syndrome: a case-control study based on patient records. Clin Oral Investig. 2011; 15: 571-575.

https://doi.org/10.1007/s00784-010-0419-5

PMid:20440632

2. Lamey PJ, Lamb AB. Prospective study of aetiological factors in burning mouth syndrome. Br Med J (Clin Res Ed). 1988; 296:1243-1246.

https://doi.org/10.1136/bmj.296.6631.1243

PMid:3133028 PMCid:PMC2545712

3. Pezelj-Ribaric S, Kqiku L, Brumini G, Urek MM, Antonic R, Kuis D, et al. Proinflammatory cytokine levels in saliva in patients with burning mouth syndrome before and after treatment with low-level laser therapy. Lasers Med Sci. 2013; 28(1): 297-301.

https://doi.org/10.1007/s10103-012-1149-5

PMid:22773117

4. Pekiner FN, Gumru B, Demirel GY, Ozbayrak S. Burning mouth syndrome and saliva: detection of salivary trace elements and cytokines. J Oral Pathol Med. 2009; 38(3): 269-75.

https://doi.org/10.1111/j.1600-0714.2008.00734.x

PMid:19141055

5. Grushka M, Epstein JB, Gorsky M. Burning mouth syndrome: differential diagnosis. Dermatol Ther. 2002; 15: 287-291.

https://doi.org/10.1046/j.1529-8019.2002.01535.x

6. Therapeutic options in idiopathic burning mouth syndrome: literature review. Int Arch Otorhinolaryngol. 2015; 19(1): 86-9.

7. Cho GS, Han MW, Lee B, Roh JL, Choi SH, Cho KJ, et al. Zinc deficiency may be a cause of burningmouth syndrome as zinc replacement therapy has therapeutic effects. J Oral Pathol Med. 2010; 39(9): 722-7.

https://doi.org/10.1111/j.1600-0714.2010.00914.x

PMid:20618611

8. Lamey PJ, Lamb AB. Prospective study of aetiological factors in burning mouth syndrome. Br Med J (Clin Res Ed). 1988; 296(6631):1243-6.

https://doi.org/10.1136/bmj.296.6631.1243

PMid:3133028 PMCid:PMC2545712

9. Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol. 2013; 19(5): 665-72.

https://doi.org/10.3748/wjg.v19.i5.665

PMid:23429751 PMCid:PMC3574592

10. Mock D, Chugh D. Burning mouth syndrome. Int J Oral Sci. 2010; 2(1): 1- 4.

https://doi.org/10.4248/IJOS10008

PMid:20690412 PMCid:PMC3475590

11. Muzyka BC, De Rossi SS. A review of burning mouth syndrome. Cutis. 1999; 64(1): 29-35.

12. Sun A, Wu KM, Wang YP, Lin HP, Chen HM, Chiang CP. Burning mouth syndrome: a review and update. J Oral Pathol Med. 2013; 42(9): 649-55.

https://doi.org/10.1111/jop.12101

PMid:23772971

13. Maragou P, Ivanyi L. Serum zinc levels in patients with burning mouth syndrome. Oral Surg Oral Med Oral Pathol. 1991; 71(4): 447-50.

https://doi.org/10.1016/0030-4220(91)90427-E

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ORIGINAL ARTICLE OPEN ACCSESS

Study on Diagnostic Evaluation of Pancytopenic Patients

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Abstract

Introduction: *Pancytopenia is a common hematological problem with an extensive differential diagnosis and is a challenging problem to the treating physician. Bone marrow aspiration and biopsy is an important diagnostic test for patient management. The objective of this study is to find out the incidence various causes of pancytopenia in patients attending to the Enam medical college hospital in savar.* ***Materials and Methods:*** *This prospective observational study was done in the department of haematology, Enam Medical College Hospital (EMCH) from July 2012 to June 2019.* ***Results:*** *In our study out of 66 patients, 36 (54.55%) were male, 30 (45.45%) were female and male to female ratio were 1.2:1.Generalized weakness 47 (71.21%) and fever 23 (34.85%) were the most common presenting symptoms followed by bleeding 17 (25.76%), weight loss 6 (12.12%), bodyache 6 (9.09%). Most common clinical findings were anemia 57 (86.36%) and bone tenderness 22 (33.33%). Other physical findings were purpura/brusing 13 (19.70%), splenomegaly 10 (15.15%), lymphadenopathy 4 (6.06%) and hepatomegaly 3 (4.55%). Hematological malignancy 29 (43.94%) and hypoplastic marrow 26 (39.39%) were the most common bone marrow finding of pancytopenic patients followed by megaloblastic anaemia 4 (6.06%), leishmaniasias 5 (7.58), and erythroid hyperplasia 2 (3.03%). Acute myeloid leukaemia was the common haematological malignancy 16 (24.24%), others were acute lymphoblastic leukaemia 5 (7.58%). Myelodysplastic syndrome 3 (4.55%), multiple myeloma 4 (6.06%), chronic myelogenous leukaemia in blastic crisis 1 (1.52%).* ***Conclusion:*** *So we concluded that complete workup is essential for all cases of pancytopenia to find out the treatable cases and to reduce the motality and morbidity in serious diseases.*

Key words: *Pancytopenia, Bone marrow examination, Haematological malignancy.*

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Introduction:

Pancytopenia is a common hematological problem with an extensive differential diagnosis and is a challenging problem to the treating physician. It is not a disease but a triad of anaemia, leucopenia and thrombocytopenia. Various pathophysiological mechanisms are related to the development of pancytopenia and this includes reduced or ineffective hematopoiesis and increased destruction by either sequestration or destruction by antibodies[1]. The cause of pancytopenia may be thus lie in the bone marrow, periphery or both.Various factors encompassing geographic distribution and genetic disturbances may cause variation in the incidence of disorders causing pancytopenia[2,3,4]. The presenting symptoms are often attributed to anaemia/ thrombocytopenia. Leukopenia is an uncommon cause of initial presentation but can become the most serious threat to life during the course of the disorder[5]. A detailed history, physical examination and complete blood counts with reticulocyte count and peripheral blood smear remain essential for diagnosis. Bone marrow examination is essential to determine the cause of pancytopenia, as it plays a major role in hematological malignancies, unexplained cytopenias and storage disorders[6]. Trephine biopsy is mainly undertaken when hypoplasia or aplasia of bone marrow being suspected on aspiration[7]. The severity of pancytopenia and the underlying pathology determine the management and prognosis of these patients[8]. The objective of this study is to find out the incidence various causes of pancytopenia in patients attending to Enam medical college hospital in savar.

Materials and Methods:

This prospective observational study was done in the department of haematology, Enam Medical College Hospital (EMCH) from July

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2012 to June 2019. A total of 66 pancytopenic patient were included in this study. Chemotherapy, radiotherapy and drug induced pancytopenia cases were excluded. Pancytopenia was confirmed by complete blood count and peripheral smear examination. Clinical parameters were assessed, and other necessary haematological investigations were done. Bone marrow aspiration was done thereafter. When aspirated material was inadequate or there was dry tap, trephine biopsy was done. Data was collected and subsequently analyzed.

Results:

Total 66 patients were included in our study. 36 (54.55%) were male, 30 (45.45%) were female and male to female ratio were 1.2:1. The age range of the patients were 9 years to 80 years. Generalized weakness 47 (71.21%) and fever 23 (34.85%) were the most common presenting symptoms. Other presenting symptoms were bleeding 17 (25.76%), weight loss 6 (12.12%), Bodyache 6 (9.09%). Most common clinical findings were anemia 57 (86.36%) and bone tenderness 22 (33.33%). Other findings were purpura/brusing 13 (19.70%), splenomegaly 10 (15.15%), lymphadenopathy 4 (6.06%) and hepatomegaly 3 (4.55%).

Haematological malignancy 29 (43.94%) and hypoplastic marrow 26 (39.39%) were the most common bone marrow finding of pancytopenic patients. Bone marrow finding are given in table I.

Table-I: Bone marrow aspiration finding in case of pancytopenia :

Bone marrow finding	No.of cases	Percentage(%)
Haematological malignancy	29	43.94
Hypoplastic bone marrow	26	39.39
Leishmaniasis	5	7.58
Megaloblastic anemia	4	6.06
Erythroid hyperplasia	2	3.03

Most common haematological malignancy was acute myeloid leukaemia 16 (55.17%). Haematological malignancies are summarizing in table II.

Table-II: Haematological malignancies:

Haematological malignancies	No of cases (n-29)	Percentage (%)	Total number of cases (n-66)	Percentage (%)
Acute myeloid leukaemia	16/29	55.17	16/66	24.24%
Acute lymphoblastic leukaemia	5/29	17.24	5/66	7.58%
Multiple myeloma	4/29	13.79	4/66	6.06%
Myelodysplastic syndrome	3/29	10.34	3/66	4.55%
Chronic myelogenous leukaemia in blastic crisis	1/29	3.45	1/66	1.52%

Discussion:

Pancytopenia is quite common in our day today clinical practice and challenging problem to both physicians and haematologists. Patients commonly presents with features of varying degrees of anaemia followed by infections and bleeding manifestations. A wide variety of disorder can causes pancytopenia demanding thorough evaluation. Diagnostic tools of pancytopenic patients are peripheral blood smear examination, bone marrow aspirations and trephine biopsy.

Total 66 patients with pancytopenia were included in our study with male preponderance, 36 (54.55%) patients were male, 30 (45.45%) were female and male: female ratio was 1.2:1. This is in agreement with Parmar JK et al.(1.6: 1), Nigam RK et al. (1.12: 1), Thakkar BB et al. (1.08:1), Para R & Para S. (1.1: 1), Goyal H et al, (1.37:1) studies[9,10,11,12,13]. The exact cause of male preponderance is unknown, but may be partly explained by increased exposure of male to environmental agents like agricultural pesticide. Few studies showed female preponderance[14,15]. Age range was 9 years to 80 years in our study. Same age group also seen by Tilak V and Jain R[8], Tariq M et al.,[16] Mussarrat N et al.,[17] Qamar U and Aijaz J.[18] Khodke K et al.,[19] and Gayathri BN and Rao KS.[20] of their studies.

Generalized weakness 47(71.21%) and fever 23(34.85%) were the most common presenting symptoms. Other presenting symptoms were bleeding 17 (25.76%), weight loss 6 (12.12%), body ache 6(9.09%). Generalized weakness and fever also a most common presenting symptoms were seen in Niazi M and Raziq F.[21] Pathak R et al.[22] Kumar DB, Raghupathi AR[14]. Most common clinical findings were pallor 57(86.36%) and bone tenderness 22(33.33%). Pallor was the common clinical sign seen by Parmar JK et al (100%), Thakkar B B et al. (100%), Goyal H et al. (97.8%), Chhabra A et al.(64.8%) [9,11, 13,23]. The 2nd most common clinical finding was bony tenderness (33.33%) which was near similar to Hayat AS et al. study (44.70%)[24] but lower than Anita PJ et al. study(4.71%)[25]. Other findings in our study were purpura/brusing 13(19.70%), splenomegaly 10(15.15%), lymphadenopathy 4(6.06%) and hepatomegaly 3(4.55%). Common Physical findings included hepatomegaly (24.52%), Splenomegaly (17.92%) and lymphadenopathy (5.66%) in Anita PJ et al. study[25], and hepatomegaly (24.32%), splenomegaly (44.14%) and lymphadenopathy (6.31%) in Santra G et al. study[26]. The frequencies of other clinical features were variable and different from these studies probably due to broad spectrum of etiologies behind pancytopenia.

Hematological malignancy 29 (43.94%) was the most common bone marrow finding of pancytopenic patients in our study which was similar to Imbert M et al.[29] study. The commonest cause of pancytopenia was aplastic anemia reported by Mussarrat N et al. (38.3%)[17],Qamar U and Aijaz J. (50.67%)[18], Khodke K et al. (29.5%)[19], Lakhey A et al (29.6%)[27], Hossain MA et al.[28], whereas in our study it is second common cause accounting for 39.39% (n-26).

The commonest cause of pancytopenia was megaloblastic anaemia reported by Rahim F et al.(24.92%)[7] , Javalgi AP and Dombale VD.(72.6%)[30], Rangaswamy M et al.(49%)[31], whereas in our study it was 6.06% (n-4). We avoided bone marrow examination in suspected cases of megaloblastic anaemia because it is not an essential test for diagnosis.

In our study leishmaniasis seen in 7.58% (n-5) and erythroid hyperplasia seen in 3.03% (n-2) cases. Leishmaniasis 2.1%, 6.9%, 0.6% was seen in Goyal H et al.[13], Mallik M et al.[32], Ojah S et al.[33] study. Erythroid hyperplasia 11.11%, 3%, 11.3% was seen in, Lakhey A et al.[27] , Sakunthala and Subitha S.34 Makheja KD et al.[35] study.

In our study, 43.94% (n-29) of patients presented with haematological malignancy with pancytopenia and was the most common cause of pancytopenia. In present study acute myeloid leukaemia was 24.24% (n-16) of our total cases whereas Jha A et al.[36] found acute myeloid leukaemia to be 19.59% of total cases. Lakhey a et al.[27] found 12.96% of total cases in their study. Savage DG et al.[37] in Zimbabwe and Varma N et al.[38] also described acute myeloid leukaemia as the third most common cause of pancytopenia.

In this study acute lymphoblastic leukaemia comprised of 7.58% (n-5) of our pancytopenic patients. In other studies showed 3.70% and 10% of their total cases[27,39].

Pancytopenia with multiple myeloma as diagnosis was noted in 6.06% (n-4) of our total patient compared to Khodke K et al. where it was reported to be 4%.

Out of 66 cases the incidence of myelodysplastic syndrome in our study constituted 4.55% (n-4). Qamar U and Aijaz J.[18] reported 5.3% which is comparable to our study. Other study were showed 8.3%, 2.4%, 7.4%[14,17,27].

We observed that causes of pancytopenia were variable and different from study to study. This is due to the differences in methodology and stringency of diagnostic criteria, geographic area, period of observation, genetic differences and varying exposure to toxic agents etc.

Conclusion:

Pnacytopenia is a common entity in our clinical practice. Bone marrow aspiration and biopsy is an important diagnostic test for patient management. Hypoplastic bone marrow, haematological malignancy and megaloblastic anaemia are the most common cause's pancytopenia. In our study common causes are haematological malignancy and hypoplastic marrow. However, uncommon and rare causes such as myelofibrosis, hairy cell leukaemia, storage disease and infection etc should be kept in mind during complete work up.

Conflict of Interest: None.

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References:

1.Williams MD. Pancytopenia, aplastic anemia and pure red cell aplasia. In: Lee RG, Foerster J, Lukens J, Paraskevas F, Greer JP, Rodgers GM, (eds). Wintrobe's Clinical Haematology. 10th edn. Williams and Wilkins ;1997: 1449-1476.

2. International agranulocytosis and aplastic anemia study. Incidence of Aplastic anemia, relevance of diagnostic criteria. Blood. 1987; 70: 1718-1721.

https://doi.org/10.1182/blood.V70.6.1718.bloodjournal7061718

https://doi.org/10.1182/blood.V70.6.1718.1718

3. Wintrobe MM (ed). Clinical Hematology. 8th edition. Philadelphia: Lea and Febiger; 1981: 699-915.

4. Keiser M, Ost A. Diagnosis in patients with severe pancytopenia suspected of having aplastic anemia. EurJ Haematol. 1990; 45:11-14.

https://doi.org/10.1111/j.1600-0609.1990.tb00407.x

PMid:2379559

5. Firkin F, Chesterman C, Pennigton D, Rush B. De Gruchy's Clinical Hematology in Medical Practice. 5th ed. London: Blackwell Science Ltd; 1989: 119-136.

6. Iqbal W, Hassan K, Ikram N, Nur S. Aetiological Breakup in 208 Cases of Pancytopenia. J Rawal Med Coll. 2001; 5(1): 7-10.

7. Rahim F, Ahmad I, Islam S, Hussain M, Khattak TAK, Bano Q. Spectrum of hematological disorders in children observed in 424 consecutive bone marrow aspirations/biopsies. Pak J Med Sci. 2005; 21: 433-436.

8.Tilak V, Jain R. Pancytopenia - A Clinico hematologic analysis of 77 cases. Indian J Pathol Microbiol. 1999; 42(4): 399-404.

9. Parmar JK , Sheikh S, Vidja P. Etiological evaluation of Pancytopenia with special emphasis on megaloblastic anemia, Paripex, Indian journal of

Research. 2013; 3(4): 263-264.

10. Nigam RK, Chaudhary R, Malik R, Gour D, Shrivastava A, Tripathi A, et al. Pancytopenia- clinico-haematological studies of bone marrow examination. Journal of Evolution of Medical and Dental Sciences. 2013; 2(7): 9213-9219.

https://doi.org/10.14260/jemds/1598

11. Thakkar B B, Bhavsar N Ukti, Trivedi N J, Agnihotri AS. A study of pancytopenia in adult patients more than 12 years of age in north west region of saurashtra. National Journal of medical research. 2013; 3(1): 48-52.

12. Para R, Para S. Pancytopenia - a study of 58 cases. Journal of Evolution of Medical and Dental Sciences. 2013; 2(45): 8724-8728.

https://doi.org/10.14260/jemds/1513

13. Goyal H ,Tilak V & Singhal A. Clinico-Hematological Study of Pancytopenia with Special Reference to Idiopathic Pancytopenia. Global Journal of Medical Research. 2017; 17(1): 8-17.

14. Kumar DB, Raghupathi AR. Clinicohematologic analysis of pancytopenia study in a tertiary care centre. Basic and Applied Pathology. 2012; 5: 19-21.

https://doi.org/10.1111/j.1755-9294.2011.01121.x

15. Yadav S, Kushwaha R, Aggrawal K,Tripathi AK, Singh US, Kumar AK. A Clinico- hematological study in cases of pancytopenia: correlation of automated cell counter parameters in various etiologies. Journal of Evolution of Medical and Dental Sciences. 2013; 2(22): 4013-4023.

https://doi.org/10.14260/jemds/795

16.Tariq M, Khan N, Basri R, Amin S. Aetiology of pancytopenia. Prof Med J. 2010; 17: 252-256.

17. Mussarrat N, Fazal-e-R M, Mohammad TK. Clinical and hematological features of megaloblastic anemia alone or in combination with iron deficiency anemia - An analysis of 349 patients. J Med Sci. 2009; 17: 81-84.

18. Qamar U, Aijaz J. Results of bone marrow examination in patients presenting with pancytopenia and high mean corpuscular volume. Gomal J Med Sci. 2012;10:133-136.

19. Khodke K, Marwah S, Buxi G, Yadav RB, Chaturvedi NK. Bone marrow examination in cases of pancytopenia. J Indian Acad Clin Med. 2001; 2:56-59.

20. Gayathri BN, Rao KS. Pancytopenia: A clinico hematological study. J Lab Physicians. 2011; 3:15-20.

https://doi.org/10.4103/0974-2727.78555

PMid:21701657 PMCid:PMC3118050

21. Niazi M, Raziq F. The incidence of underlying pathology in pancytopenia: an experience of 89 cases. J Postgrad Med Inst. 2004; 18:76-79.

22. Pathak R, Jha A, Sayami G. Evaluation of bone marrow in patients with pancytopenia.J Pathology Nepal. 2012; 2: 265-271.

https://doi.org/10.3126/jpn.v2i4.6875

23. Chhabra A, Chandar V, Patel A, Chandra H. Clinicoaetiological profile of pancytopenia in paediatric practice Journal. Indian Academy of Clinical Medicine. 2012;13(4): 282-285.

24. Hayat AS, Khan AH, Baloch GH, Shaikh N. Pancytopenia; study for clinical features and etiological pattern of at tertiary care settings in Abbottabad. Professional Med J. 2014; 21(1): 60-65.

25. Anita PJ, Vijay DD. Clinico - Hematological Analysis of Pancytopenia: A Bone Marrow Study. National Journal of Laboratory Medicine. 2013; 2(4): 12-17.

26. Santra G, Das BK. A cross sectional study of the clinical profile and aetiological spectrum of pancytopenia in a tertiary care center. Singapore Med J. 2010; 51(10): 806-812.

27. Lakhey A, Talwar OP, Singh VK, Raj SK. Clinico-hematological study of pancytopenia. J Pathol Nepal. 2012; 2:207-210.

https://doi.org/10.3126/jpn.v2i3.6023

28. Hossain MA, Akond AK, Chowdhary MK, Singh KJ, Ahluwalia G, Sharma SK, et al. Pancytopenia - A study of 50 cases. Bangladesh J Pathol.1992; 1: 9-12.

29. Imbert M, Scoazec JY, Mary JY, Jouzult H, Rochant H, Sultan C. Adult patients presenting with pancytopenia: A reappraisal of underlying pathology and diagnostic procedures in 213 cases. Hematol Pathol.1989; 3: 159-167.

30. Javalgi AP, Dombale VD. Bone marrow study in pancytopenia. Natl J Lab Med. 2013; 2: 12-17.

31. Rangaswamy M, Prabhu, Nandini NM, Manjunath GV. Bone marrow examination in pancytopenia. J Indian Med Assoc. 2012; 110: 560-566.

32. Mallik M, Bhartiya R, Mallick S, Singh R, Kumar M, Singh RVN. Pancytopenia - A study of Clinico-Haematological Profile in Adults with its Bone-Marrow Co-Relation in a Tertiary Hospital of Bihar. International Journal of Contemporary Medical Research. 2016; 3(6): 1689-1691.

33. Ojha S, Haritwal A, Meenai FJ, Gupta S. Bone marrow examination findings in cases of pancytopenia- a study from central India. Indian Journal of Pathology and Oncology. 2016; 3(3): 479-484

https://doi.org/10.5958/2394-6792.2016.00089.2

34. Sakunthala and Subitha S. Bone Marrow Examination In Cases Of Pancytopenia. IOSR-JDMS. 2018; 17(5): 75-78.

35. Makheja KD, Maheshwari BK, Arain S, Kumar S, Kumari S, Vikash. The common causes leading to pancytopenia in patients presenting to tertiary care hospital.Pak J Med Sci. 2013; 29(5): 1108-1111.

https://doi.org/10.12669/pjms.295.3458

36. Jha A, Sayami G, Adhikari RC, Patna AD, Jha R. Bone marrow examination in cases of pancytopenia. J Nepal Med Assoc. 2008; 47: 12-17.

https://doi.org/10.31729/jnma.209

37. Savage DG, Allen RH, Gangaidzo IT. Pancytopenia in Zimbabwe. Am J Med Sci.1999; 317: 22-32.

https://doi.org/10.1016/S0002-9629(15)40466-5

https://doi.org/10.1097/00000441-199901000-00004

PMid:9892268

38. Varma N, Dash S. A reappraisal of underlying pathology in adult patients presenting with pancytopenia. Trop GeogrMed. 1992; 44: 322-327.

39. Sreeramulu D, Laxmibai S, Srinivasulu D, Malik M S. Pancytopenia-An Evaluation. International Journal of Applied Research. 2015; 1(10): 744-746.

DOI: https://doi.org/10.3329/medtoday.v32i1.44835

ORIGINAL ARTICLE OPEN ACCSESS

The Association between Glucose Levels and Hospital Outcomes in Patients with Acute Exacerbations of Chronic Obstructive Pulmonary Disease

Saleh Ahmed*[1], Pritam Bordhan[2], Hasan Imam[3], Umme Salma[4]

Abstract

Introduction: *Chronic obstructive pulmonary disease (COPD) is the fourth most common cause of death in the world and will become the third most common in a few years. Identification of prognostic factors may lead to improved treatment strategies and clinical outcomes for COPD. To find out the association between glucose levels and hospital outcomes in patients with acute exacerbations of chronic obstructive pulmonary disease.* ***Materials and Methods:*** *This retrospective study was carried out different privet and public hospital in Chandpur. The information reported in this manuscript was collected during three separate projects in the same hospital. The first cohort included 35 COPD patients admitted for an acute exacerbation to a non-ICU bed to determine if there was a relationship between corticosteroid dosage and hyperglycemia. The second cohort included 210 COPD patients with acute exacerbations who were admitted to the MICU to determine factors associated with mortality and LOS. The third cohort included 105 COPD patients who were monitored for re-admission within 30 days after discharge for an acute exacerbation.* ***Results:*** *Mean arterial pressure, hemoglobin, blood urea nitrogen, albumin, nursing home, pleural effusion and intubation were statistically significant ($p<0.05$) between two group. Intubation had 5.318 (95% CI 2.578 to 11.721) times increase in odds having length of stay. Intubation was significantly associated with length of stay.* ***Conclusion:*** *Mean arterial pressure, hemoglobin, blood urea nitrogen, albumin, nursing home, pleural effusion and intubation were statistically significant between in hospital deaths group and survivors group.*

Keywords: *Acute exacerbation, Adverse effects, COPD, Corticosteroids.*

Number of Tables: 05; Number of References: 23; Number of Correspondence: 03.

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Introduction:

Chronic obstructive pulmonary disease (COPD) is the fourth most common cause of death in the world and will become the third most common in a few years. Acute exacerbation is a frequent event in the natural course of COPD (AECOPD) and is characterized by the acute aggravation of respiratory symptoms such as dyspnea and coughing with or without sputum that require specific treatment regimens[1].

Identification of prognostic factors may lead to improved treatment strategies and clinical outcomes for COPD. Among acute exacerbation of COPD (AECOPD) patients, adverse outcomes are associated with lower arterial pH, older age, 2-5 male gender, underlying comorbidities, higher income, [2-4] disease severity, and in-hospital complications[4].

Effective management of an acute exacerbation of COPD (AECOPD) requires symptom relief and reducing the risk for subsequent exacerbations. Identification of patients at risk for more complicated hospital courses should facilitate in-patient management, and risk factors for adverse outcomes include lower arterial pHs, older age, male gender, underlying comorbidities, disease severity, and in-hospital complications[2]. Hyperglycemia is associated with poor outcomes in patients with pneumonia,[5] myocardial infarction,[6] and stroke but the effect of hyperglycemia on outcomes during AECOPD has not been definitely established.

The outcomes from these episodes depend on the severity of the underlying chronic lung disease, the degree of acute respiratory failure superimposed on the chronic lung disease, comorbidity, and possibly hospital related complications. Hyperglycemia represents an independent risk factor for hospital associated complications and/or mortality in other medical diagnoses, such as stroke and acute myocardial infarction. Recent studies in patients with acute exa-cerbations of COPD demonstrate that hyperglycemia is associated with an increased length of hospital stay, failure of noninvasive ventilation, and/or mortality. Acute stress and medications used with an acute flare, such as glucocorticoids and beta agonists, increase blood glucose levels. The explanation for poor outcomes likely involves an increase in colonization with pathogenic bacteria, acute changes in host defenses, and possibly metabolic disorders related to hyperglycemia and glycosuria.

Materials and Methods:

This retrospective longitudinal study was carried out different privet and public hospital in Chandpur. The information reported in this manuscript

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was collected during three separate projects in the same hospital. The first cohort included 35 COPD patients admitted for an acute exacerbation to a non-ICU bed to determine if there was a relationship between corticosteroid dosage and hyperglycemia. The second cohort included 210 COPD patients with acute exacerbations who were admitted to the MICU to determine factors associated with mortality and LOS. The third cohort included 105 COPD patients who were monitored for re-admission within 30 days after discharge for an acute exacerbation. This study we included all adult patients with a discharge diagnosis of COPD exacerbation identified by the coders in the Department of Internal Medicine; we excluded patients younger than 18 years and those admitted to an ICU. Data collected included forced expiratory volume in 1 second (FEV1), history of diabetes, blood glucose at admission, peak glucose, and maximum daily dose of corticosteroid. Data collected included the patients' ages, sex, body mass indices (BMI), baseline pulmonary function tests, comorbidities, complete blood counts (CBCs), complete metabolic profiles, albumin levels, initial arterial blood gases, sputum cultures, blood cultures, chest X-rays, Acute Physiology and Chronic Health Evaluation II (APACHE II) scores, confusion, urea, respiratory rate, systolic blood pressure (CURB65) scores, final diagnoses, total duration of hospital stay, and in-hospital mortality. We separated patients into two groups: Group One died during hospitalization and Group Two survived to hospital discharge. The primary outcome in this study was the identification factors associated with increased in-hospital mortality; the secondary outcome was the identification of the factors associated with increased LOS. We separated patients by LOS into four quartiles to identify parameters associated with increased LOS. Descriptive statistics to summarize base line characteristics of patients in all three cohorts. T-tests and Chi square tests were used to analyze differences between patients who died and patients who survived the acute flare. Multivariable logistic regression models were used to determine factors that predicted increased mortality and LOS. T-tests were used to compare highest blood glucose levels in the early re-hospitalization group with the non-early rehospitalization group.

Results:

Table-I: Clinical characteristics of three cohorts.

Clinical characteristics	Cohort I (n=35)	Cohort II (n=210)	Cohort III (n=105)
Male	26	98	52
Age (years)	69.7	66.9	73.6
Diabetes	13	82	-
FEV1 (%)	48.1	43.2	27.9
Mortality	-	11	4
LOS (days)	-	8	4
Re-admission	-	-	14

Cohort I: Glucose levels and corticosteroid dosage

Cohort II: Glucose levels and mortality and LOS

Cohort III: Peak glucose levels and re-admission

The basic clinical characteristics for the three cohorts are summarized

Table-II: Glucose levels in patients with chronic obstructive pulmonary disease often have acute exacerbations.

	Non diabetes (n=22)	Diabetes (n=13)	p value
	Mean±SD	Mean±SD	
Initial glucose (mg/dL)	122.3±32.1	171.9±109.7	0.054^{ns}
Peak glucose (mg/dL)	183.2±50.3	303.1±111.7	0.001^{s}
Change in glucose (mg/dL)	61.1±56.8	129.2±115.5	0.001^{s}

s= significant, ns= not significant
p value reached from unpaired t-test

Mean peak glucose was found 183.2±50.3 mg/dl in non-diabetic and 303.1±111.7 mg/dl in diabetic group. The mean change in glucose was found 61.1±56.8 mg/dl in non-diabetic and 129.2±115.5 mg/dl in diabetic group. Which were statistically significant ($p<0.05$) between two group.

Table-III: Factors associated with mortality in patients admitted to an intensive care unit.

	In hospital deaths (n=24)	Survivors (n=186)	P value
	Mean±SD	Mean±SD	
Mean arterial pressure (mmHg)	64.2±13.8	84.7±17.9	^{a}0.001^{s}
Initial PO2 sat (mmHg)	88.6±5.7	89.9±4.8	^{a}0.223^{ns}
Initial blood sugar (mg/dl)	162.4±59.3	142.7±49.7	^{a}0.076^{ns}
Hemoglobin (g/dl)	11.8±2.7	13.4±2.1	^{a}0.001^{s}
Blood urea nitrogen (mg/dl)	32.6±22.2	18.5±9.8	^{a}0.001^{s}
Albumin (gm/dl)	3.0±0.7	3.9±0.5	^{a}0.001^{s}
APACHE II score	23.1±7.6	11.6±4.4	^{a}0.001^{s}
Nursing home	13(54.2%)	16(8.6%)	^{b}0.001^{s}
Pleural effusion	6(25.0%)	15(8.1%)	^{a}0.009^{s}
Intubation	19(79.2%)	72(38.7%)	^{a}0.001^{s}

s= significant, ns= not significant
^{a}p value reached from unpaired t-test
^{b}p value reached from chi square test

Mean arterial pressure, hemoglobin, blood urea nitrogen, albumin, nursing home, pleural effusion and intubation were statistically significant ($p<0.05$) between two group.

Table-IV: Factors associated with length of stay in patients admitted to an intensive care unit (ICU).

	P value	OR (95%CI)
Nursing home	0.701	1.212 (0.434-3.352)
Initial PO2 sat	0.063	0.903 (0.874-1.132)
Albumin	0.274	0.689 (0.326-1.334)
Intubation	0.001	5.318 (2.578-11.721)
Pleural effusion	0.080	2.628 (0.789-7.704)
APACHE II score	0.953	1.00 (0.949-1.073)

Intubation had 5.318 (95% CI 2.578 to 11.721) times increase in odds having length of stay. Intubation was significantly associated with length of stay.

Table-V: Comparison of patients with chronic obstructive pulmonary disease (COPD) exacerbations according to early re-hospitalization status.

	Early re-hospitalizations		P value
	Yes (n=14)	No (n=91)	
	n(%)	n(%)	
Ejection fraction			
<55%	5(35.7%)	16(17.6%)	[a]0.114[ns]
≥55%	9(64.3%)	75(82.4%)	
Diastolic dysfunction			
Yes	6(42.9%)	46(50.5%)	[a]0.592[ns]
No	8(57.1%)	45(49.5%)	
Pulmonary artery pressure			
Normal	10(71.4%)	69(75.8%)	
Mild PAH	1(7.1%)	9(9.9%)	[a]0.903[ns]
Moderate PAH	2(14.3%)	8(8.8%)	
Severe PAH	1(7.1%)	5(5.5%)	
COPD severity			
Mild	1(7.1%)	8(8.8%)	
Moderate	1(7.1%)	9(9.9%)	
Severe	6(42.9%)	24(26.4%)	[a]0.666[ns]
Very severe	4(28.6%)	23(25.3%)	
Not done	2(14.3%)	27(29.7%)	
ProBNP			
<900	7(50.0%)	40(44.0%)	
901-1800	2(14.3%)	8(8.8%)	
>1800	1(7.1%)	12(13.2%)	[a]0.813[ns]
Not done	4(28.6%)	31(34.1%)	
Cor pulmonale	1(7.1%)	12(13.2%)	[a]0.523[ns]
Beta-blocker use	3(21.4%)	10(11.0%)	[a]0.270[ns]
Long-acting bronchodilators and/or inhaled corticosteroids	9(64.3%)	56(61.5%)	[a]0.844[ns]
Coronary artery disease	5(35.7%)	25(27.5%)	[a]0.525[ns]
Atrial fibrillation	0(0.0%)	5(5.5%)	[a]0.400[ns]
Abnormal WBC (<4.0 or >12.0 × 10^3 /μL)	2(14.3%)	19(20.9%)	[a]0.445[ns]
Intubation	1(7.1%)	7(7.7%)	[a]0.943[ns]
Mean blood glucose (mg/dl)	186.2±38.7	209.8±42.3	[b]0.052[ns]
Mean length of stay (days)	4.2±0.8	4.5±0.9	[b]0.242[ns]
Mean follow-up (days)	20.2±3.4	18.4±4.6	[b]0.121[ns]

ns= not significant

[a]p value reached from chi square test

[b]p value reached from unpaired t-test

These patients had 105 admissions, including 14 early rehospitalizations (13.3%), defined as a re-hospitalization within 30 days after discharge. The difference were not statistically significant (p>0.05) between two group.

Discussion:

In this study observed the mean peak glucose was found 183.2±50.3 mg/dl in non- diabetic and 303.1±111.7 mg/dl in diabetic group. The mean change in glucose was found 61.1±56.8 mg/dl in non- diabetic and 129.2±115.5 mg/dl in diabetic group. Which were statistically significant (p<0.05) between two group. Islam et al.[8] reported that Twenty non-diabetic patients had a mean initial glucose of 122±33 mg/dl with a range of 83 to 238 mg/dl. Six patients had maximum glucose levels above 200 mg/dl. Ten patients with diabetes (DM) had mean initial glucose levels of 196±110 mg/dl with a range of 77 to 446 mg/dl, and nine had maximum glucose levels above 200 mg/dl. We stratified the patients into tertiles based on the maximum daily dose of corticosteroids received during hospitalization. There were no differences in the initial or maximum glucose levels in these three groups (P > 0.05) Parappil reviewed 246 episodes of AECOPD[9]. Forty-two percent of the admissions had a random glucose greater than 180 mg/dl, but mean values were not provided. Fifty-three patients (22%) had DM. Burt measured glucose values in 47 patients using a continuous monitoring system; the mean level was 137±34 mg/dl[10]. The peak levels in their cohort were 220±61 mg/dl. Seven patients (15%) had DM. Chakrabarti studied 88 patients with decompensated COPD (mean pH = 7.25) who required non-invasive ventilation[11]. Jafar et al.[7] reported only the highest blood glucose recorded for the patients during their hospital stay was used for analysis, and patients were grouped according to their blood glucose quartile (group 1, <108 mg/dl, n = 69; group 2, 108–125 mg/dl, n = 69; group 3, 126–160 mg/dl, n = 75; and group 4, >160 mg/dl, n = 71). The relative risk (RR) of death or a longer inpatient stay was significantly increased in group 3 (RR 1.46, 95% CI 1.05 to 2.02, p = 0.02) and group 4 (RR 1.97, 95% CI 1.33 to 2.92, p<0.0001) compared to group 1. For each 18 mg/dl increase in blood glucose the absolute risk of an adverse outcome increased by 15% (95% CI 4% to 27%, p = 0.006).

In this study observed that the mean arterial pressure, hemoglobin, blood urea nitrogen, albumin, nursing home, pleural effusion and intubation were statistically significant (p<0.05) between two group. Islam et al.[8] reported that the majority of excluded patients had congestive heart failure with exacerbation or acute asthma and did not have an AECOPD as the primary diagnosis. Therefore, this study enrolled 217 cases with AECOPD who needed ICU admission; 26 died during hospitalization and 191 survived to hospital discharge. Eighty-nine percent of the patients who died in the hospital had a FEV1 less than 50% of predicted; 68% of the survivors had a FEV1 less than 50 % of predicted. The mean initial blood glucose on admission in this cohort was 156±74.3 mg/dl (range 60 to 485 mg/dL). The overall LOS was 9.01±6.00 days. It was 8.37±4.99 days in survivors and 13.69±9.78 days in non-survivors. Initial mean blood glucose was 156±74.3 mg/dl and did not predict a statistically significant increased LOS in univariate logistic regression analysis (OR 1.00; 95% CI: 0.99-1.01, P = 0.59). Baker et al. measured glucose levels in 284 patients with acute exacerbations of COPD requiring hospitalization and reported the highest value measured either at admission or during the admission[12]. Glucose levels have a circadian pattern with higher values in the late afternoon in corticosteroid treated patients with AECOPD, and routine morning lab tests may not detect the range of glucose elevations[13,14]. In addition, glycemic variability is associated with increased length of stay and mortality in

hospitalized patients and needs to be considered in these studies[15]. In-hospital management of hyperglycemia is relatively easy and prevents complications[16,17]. Moretti and colleagues studied 186 patients admitted to a respiratory intensive care unit with respiratory failure characterized by a mean pH of 7.23± 0.07 and a mean PaCO2 of 85.3 ±15.8 mm Hg[18]. Krinsley studied the relationship between hyperglycemia and hospital mortality in a heterogeneous group of critically ill patients and concluded that even modest hyperglycemia after intensive care unit admission was associated with a substantial increase in hospital mortality in patients with a wide range of medical and surgical diagnoses[19].

In current study reported intubation had 5.318 (95% CI 2.578 to 11.721) times increase in odds having length of stay. Intubation was significantly associated with length of stay. Islam et al.[8] observed multivariable logistic regression including statistically significant mortality risk factors from univariate logistic regression showed that patients who lived in a nursing home (OR 50.02; 95% CI: 2.7-923.19), had a low hemoglobin (OR 0.40; 95% CI: 0.17-0.94), were intubated (OR 27.54; 95%1.78-425.39), and had a high APACHE II score (OR 1.55; 95% CI: 1.07-2.23) were at increased risk for mortality, but the initial glucose levels were not associated with increased risk. Univariate analysis did demonstrate that nursing home status, low albumin, the presence of a pleural effusion, intubation, and high APACHE II scores were associated with increased LOS ($P < 0.05$ for each factor). A multivariable logistic regression model demonstrated that only intubation (OR 5.93; 95%CI 2.78-12.65) predicted a statistically significant increase in hospital LOS. High intracellular glucose concentrations increase the production of reactive oxygen species, such as superoxide, and of peroxynitrite which can impair mitochondrial function and damage proteins, cellular membranes, and nucleic acids[20,21]. Hyperglycemia causes glycosylation of proteins, impaired leukocyte function, and activation of pro-inflammatory genes through transcription factors[20,22]. Glycosuria potentially causes an osmotic diuresis with a loss of electrolytes and volume contraction which could adversely affect the clinical course. Occasionally, patients on corticosteroids develop hyperglycemic hyperosmolar syndrome or diabetic ketoacidosis. Other studies have reported that hyperglycemia increases the mortality in patients with acute myocardial infarction, with ischemic or hemorrhagic stroke, and with community-acquired pneumonia[6,23]. Parappil et al.[9] retrospectively analyzed 172 patients admitted with AECOPD, including 39 patients with comorbid diabetes mellitus. In their study the presence of DM was associated with increased length of stay (mean 7.8 days) and mortality (8%) in comparison with patients without DM (6.5 days and 4% mortality), but these differences were not statistically significant.

In present study observed that patients had 105 admissions, including 14 early rehospitalizations (13.3%), defined as a re-hospitalization within 30 days after discharge. The difference were not statistically significant ($p>0.05$) between two group. Islam et al.[8] reported these patients had 103 admissions, including 13 early rehospitalizations (12.6 %), defined as a re-hospitalization within 30 days after discharge. The mean peak blood glucose levels were 212±105 mg/dl with a range from 94 mg/dl to 600 mg/dl. There were no differences in mean peak glucose levels between early re-hospitalization and non-early re-hospitalization patients ($P = 0.318$).

Conclusion:

Mean arterial pressure, hemoglobin, blood urea nitrogen, albumin, nursing home, pleural effusion and intubation were statistically significant between in hospital deaths group and survivors group. On the frequency and severity of hyperglycemia in these patients, the factors associated with hyperglycemia, the effects of hyperglycemia on short term outcomes.

Conflict of Interest:

The developments of hyperglycemia introduce practical problems in the management of the patients both in the hospital and after discharge. A number of information was not consistently available in the hospital records. Different physicians were managed the patients and different approaches would influence the choice of medication and dose of medication.

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References:

1. Global Initiative for Chronic Obstructive Lung Disease (GOLD). 2011. Global Strategy for the Diagnosis, Management and Prevention of COPD. http://www.goldcopd.org/. Accessed:1 May 2012.

2. Patil SP, Krishnan JA, Lechtzin N, Diette GB. In-hospital mortality following acute exacerbations of chronic obstructive pulmonary disease. Arch Intern Med. 2003; 163(10): 1180-1186.

https://doi.org/10.1001/archinte.163.10.1180

PMid:12767954

3. Groenewegen KH, Schols AM, Wouters EF. Mortality and mortality-related factors after hospitalization for acute exacerbation of COPD. Chest. 2003; 124(2): 459-467.

https://doi.org/10.1378/chest.124.2.459

PMid:12907529

4. Bustamante-Fermosel A, De Miguel-Yanes JM, Duffort-Falco M, Muñoz J. Mortality related factors after hospitalization for acute exacerbation of chronic obstructive pulmonary disease: the burden of clinical features. Am J Emerg Med. 2007; 25(5): 515-522.

https://doi.org/10.1016/j.ajem.2006.09.014

PMid:17543654

5. McAlister FA, Majumdar SR, Blitz S. The relation between hyperglycemia and outcomes in 2,471 patients admitted to the hospital with community-acquired pneumonia. Diabetes Care. 2005; 28(4):810-5.

https://doi.org/10.2337/diacare.28.4.810

PMid:15793178

6. Capes SE, Hunt D, Malmberg K. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. Lancet. 2000; 355(9206): 773-8.

https://doi.org/10.1016/S0140-6736(99)08415-9

7. Jafar N, Edriss H, Islam E, Nugent K. The association between blood glucose levels and hospital outcomes in patients admitted with acute exacerbations of chronic obstructive pulmonary disease. The Southwest Respiratory and Critical Care Chronicles. 2014; 2(7): 1-11.

https://doi.org/10.12746/swrccc2014.0207.082

8. Islam EA, Limsuwat C, Nantsupawat T, Berdine GG, Nugent KM. The association between glucose levels and hospital outcomes in patients with acute exacerbations of chronic obstructive pulmonary disease. Ann Thorac Med. 2015; 10: 94-9.

https://doi.org/10.4103/1817-1737.151439

PMid:25829959 PMCid:PMC4375748

9. Parappil A, Depxynski B, Collett P, Marks GB. Effect of comorbid diabetes on length of stay and risk of death in patients admitted with acute exacerbations of COPD. Respirology. 2010; 15: 918-22.

https://doi.org/10.1111/j.1440-1843.2010.01781.x

PMid:20546185

10. Burt MG, Roberts GW, Aguilar-Loza NR, Quinn SJ, Frith PA, Stranks SN. Relationship between glycaemia and length of hospital stay during acute exacerbation of chronic obstructive pulmonary disease. Intern Med J. 2013; 43: 721-4.

https://doi.org/10.1111/imj.12157

PMid:23745995

11. Chakrabarti B, Angus RM, Agarwal S, Lane S, Calverley PM. Hyperglycaemia as a predictor of outcome during non-invasive ventilation in decompensated COPD. Thorax. 2009; 64: 857-62.

https://doi.org/10.1136/thx.2008.106989

PMid:19454410

12. Baker EH, Janaway CH, Philips BJ, Brennan AL, Baines DL, Wood DM, et al. Hyperglycaemia is associated with poor outcomes in patients admitted to hospital with acute exacerbations of chronic obstructive pulmonary disease. Thorax. 2006; 61: 284-9.

https://doi.org/10.1136/thx.2005.051029

PMid:16449265 PMCid:PMC2104606

13. Burt MG, Roberts GW, Aguilar-Loza NR, Frith P, Stranks SN. Continuous monitoring of circadian glycemic patterns in patients receiving prednisone for COPD. J Clin Endocrinol Metab. 2011; 96: 1789-96.

https://doi.org/10.1210/jc.2010-2729

PMid:21411550

14. Roberts GW, Monteiro VE. Pattern of high-dose prednisoloneinduced hyperglycaemia in COPD exacerbations. J Pharm Pract Res. 2009; 39: 50-4.

https://doi.org/10.1002/j.2055-2335.2009.tb00704.x

15. Mendez CE, Mok KT, Ata A, Tanenberg RJ, Calles-Escandon J, Umpierrez GE. Increased glycemic variability is independently associated with length of stay and mortality in non-critically ill hospitalized patients. Diabetes Care. 2013; 36: 4091-7.

https://doi.org/10.2337/dc12-2430

PMid:24170754 PMCid:PMC3836112

16. Kavanagh BP, McCowen MB, McCowen KC. Clinical practice. Glycemic control in the ICU. N Eng J Med 2010; 363: 2540-6.

https://doi.org/10.1056/NEJMcp1001115

PMid:21175316

17. Moghissi ES. Addressing hyperglycemia from hospital admission to discharge. Curr Med Res Opin. 2010; 26: 589-98.

https://doi.org/10.1185/03007990903566822

PMid:20078323

18. Moretti M, Cilione C, Tampieri A, Fracchia C, Marchioni A, Nava S. Incidence and causes of non-invasive mechanical ventilation failure after initial success. Thorax. 2000; 55(10): 819-825.

https://doi.org/10.1136/thorax.55.10.819

PMid:10992532 PMCid:PMC1745609

19. Krinsley JS. Association between hyperglycemia and increased hospital mortality in a heterogeneous population of critically ill patients. Mayo Clin Proc. 2003; 78(12): 1471-148.

https://doi.org/10.4065/78.12.1471

PMid:14661676

20. Clement S, Braithwaite SS, Magee MF, Ahmann A, Smith EP, Schafer RG, et al. American Diabetes Association Diabetes in Hospitals Writing Committee. Management of diabetes and hyperglycemia in hospitals. Diabetes Care. 2004; 27: 553-91.

https://doi.org/10.2337/diacare.27.2.553

PMid:14747243

21. Chaudhuri A, Umpierrez GE. Oxidative stress and inflammation in hyperglycemic crises and resolution with insulin: Implications for the acute and chronic complications of hyperglycemia. J Diabetes Complications. 2012; 26: 257-8.

https://doi.org/10.1016/j.jdiacomp.2012.04.016

PMid:22658407 PMCid:PMC3718464

22. Nielson CP, Hindson DA. Inhibition of polymorphonuclear leukocyte respiratory burst by elevated glucose concentrations in vitro. Diabetes. 1989; 38: 031-5.

https://doi.org/10.2337/diabetes.38.8.1031

PMid:2753233

23. Umpierrez GE, Isaacs SD, Bazargan N, You X, Thaler LM, Kitabchi AE. Hyperglycemia: An independent marker of in-hospital mortality in patients with undiagnosed diagnosis. J Clin Endocrinol Metab. 2002; 87: 978-82.

https://doi.org/10.1210/jcem.87.3.8341

PMid:11889147

DOI: https://doi.org/10.3329/medtoday.v32i1.44836

ORIGINAL ARTICLE OPEN ACCSESS

Adhesive Capsulitis of Shoulder (Frozen Shoulder) among the Diabetic Patients with Rheumatic Complaints

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Abstract

Introduction: *Adhesive capsulitis of shoulder is the most common soft tissue rheumatism among the diabetes patients with rheumatic complaints but the etiology is still unknown. To detect the incidence of adhesive capsulitis (frozen shoulder) among diabetic patients with rheumatic complaints.* ***Materials and Methods:*** *All patients having diabetes with rheumatic complaints attended in the department of Physical Medicine and Rehabilitation, BIRDEM and BSMMU, Dhaka during the period June 2003 to November 2003 were included in this study.* ***Results:*** *After discarding unsatisfactory samples, 273 samples were assayed for study. Out of 1665 patients 273 (16.40%) had adhesive capsulitis. Out of 273 patients 145 (53.12%) were female and 141 (51.65%) were housewives and of 206 (75.45%) patients was in age group 41-60 years.* ***Conclusion:*** *The incidence of adhesive capsulits of shoulder among diabetic patients female is more than male. It is found that housewives and elderly people are mostly sufferer with this disease.*

Key words: *Adhesive capsulitis, Diabetes, Rheumatic disease.*

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Introduction:

Frozen shoulder is a general term used to describe any shoulder that is stiff. Adhesive capsulitis of shoulder is a common and disabling condition is characterized by pain and stiffness in the absence of any recognized intrinsic abnormality in the shoulder joint[1]. This condition involves the spontanesous, gradual onset of shoulder stiffness and pain caused by tightening of the joint capsule. Adhesive capsulitis is a condition in which there is pain and stiffness or motion loss in the shoulder[2]. Adhesive capsulitis is a painful and disabling condition that often causes great frustration for patients and caregivers due to slow recovery. Movement of the shoulder is severely restricted. Pain is usually constant, worse at night, and when the weather is colder; and along with the restricted movement can make even small talks impossible. Certain movement or bumps can cause sudden onset of tremendous pain cramping that can last several mintus[3].

The shoulder is a unique anatomical structure with a an extraordinary range of motion (ROM) that allows us io interact with our environment. A loss of mobility if this joint will cause significant morbidity. Adhesive capsulitis is a poorly understood musculoskeletal condition that can be disabling. Adhesive capsulitis is diagnosed by numerous physical characteristics including a thickening of the synovial capsule, adhesions within the subacromial or subdeltoid bursa, adhesions to the biceps tendon, and/or obliteration of the auxillary fold secondary to adhesions[4-12]. Since Duplay initially described a case report of adhesive capsulitis almost 130 years ago, this condition remains an enigmatic shoulder disorder that causes pain and restricted ROM at the glenohumeral joint[13].

Stages of adhesive capsulitis progression:

First, Early freezing stage (last 2-9 months, gradual reduction ROM, Increase in pain-principally at night, achy at rest, aggravated by arm movement.)

Second, Frozen stage(last 4-12 months, pain reduces, ROM greatly reduce or lost entirely, compensatory movements)

Third, Thawing stage (last 4-14 months, gradual restoration of mobility, reduction in pain, in 10-20% of cases the second shoulder will be affected within 5 years)

Patients with different rheumatic complaints were referred from orthopedic, neuromedicine and other outpatient department to the of Physical Medicine & Rehabitation, BIRDEM and BSMMU for proper treatment and rehabilitation. They used to treat by drugs, physical therapies, exercises, braces and activities of daily of living instructions.

An attempt has been taken to find out the incidence and etiological pattern of adhesive capsulitis of shoulder among the diabetes patients. Data collected from this study may be helpful for proper management of the diabetes patients with adhesive capsulitis and suffering of the patients can be reduced.

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Materials and Methods:

It was a prospective study carried out in the department of Physical Medicine & Rehabilitation, BIRDEM Hospital collaborated with the department of Physical Medicine, BSMMU, Dhaka during June 2003 to November 2003. Two hundred seventy three patients were selected following the inclusion criteria (suffering from adhesive capsulitis, having diabetes mellitus with rheumatic complaints, aged more than 20 years, both male and female).

Proper consent was taken from the patients. All patients were examined thoroughly and clinical and demographic history was taken in a pre-designed form. Relevant investigation were done. All data were compiled and edited meticulously. The data were screened and were checked for any discrepancy. All omissions and inconsistencies were corrected methodically. The numerical data were analyzed statistically by using SPSS 15.0 statistical package. The results were expressed as frequency and percentage.

Results:

Adhesive capsulitis of shoulder was 16.40% among 1665 patients with rheumatic disorders.

Table-I: Distribution of adhesive capsulitis of shoulder among diabetic patients.

Total number of patients	Number of patients with adhesive capsulitis	Percentage
1665	273	16.40

Table-II: Distribution of adhesive Capsulitis of shoulder among diabetic patients by sex (n=273).

Sex	Number of patients	Percentage
Male	128	46.88
Female	145	53.12
Total	**273**	**100.00**

Table-III: Age distribution of adhesive capsulitis of shoulder among diabetic patients (n-273).

Age	Number of patients	Percentage
21-30	3	1.10
31-40	21	7.69
41-50	102	37.36
51-60	104	38.09
60-70	31	11.36
Above 70	12	4.40
Total	**273**	**100.00**

Table-IV: Occupational distribution of adhesive capsulitis of shoulder among diabetic patients(n=273).

Occupation	Number of patients	Percentage
House wives	141	51.65
Service	56	20.51
Retd. Serviceman	30	10.99
Business	27	9.89
Cultivators	4	1.47
Driver	5	1.83
Other	10	3.66
Total	**273**	**100.00**

Discussion:

Adhesive capsulitis of shoulder is comprises the most common disease among diabetes patients it appears to increase with age and most common in women than men. A study done by J.F. BRIDGMAN revealed that 10.16% of the study population presented frozen shoulder[14].

In our study, 1665 patients attended in the department of Physical Medicine and Rehabilitation, BIRDEM and BSMMU, Dhaka. Among them 273 (16.50%) patients were found with adhesive capsulitis.

In our study most of the patients 145 (53.12%) were female. Female male ratio was 1.13:1. Frozen Shoulder affects women more frequently than men, with a female-to-male ratio of about 1.4:1[15].

In our study most of the patients were of 41-60 years age group constituted 75-40% of the total study population followed by 31 (11.36%), 21 (7.69%), 12 (4.40%) and 31 (1.10%) were in age group 60-70 years, 31-40 years, above 70 years and 21-30 years respectively. Adhesive capsulitis is rare in children,[16] and peaks between 40 and 70 years of age[4].

In this study most of the patients 141 (51.65%) were housewives followed by serviceman, retd, serviceman, business, other profession, driver and cultivator were 56 (20.51%), 30 (10.99%), 27 (9.89%), 10 (3.66%), 5 (1.83%) and 4 (1.47%) respectively.

Conclusion:

Considering the information gathered from this study, adhesive capsulitis of shoulder is the common form of soft itssue rheumatism of non articular rheumatism among diabetic patients with rhemetic complaints. From the present study it may be concluded that female patients are more sufferer than male patients and housewives elderly people are mostly affected with this diseases.

Conflict of Interest: None.

Acknowledgement:

This is my great pleasure to all patients having diabetes with rheumatic complaints attended in the department of Physical Medicine and Rehabilitation, BIRDEM and BSMMU, Dhaka during the period June 2003 to November 2003.

References:

1. Nukii G. Diseases of the connective tissue, joints and bones. In: Edwards CRW, Bouchier IAD, Haslett C, editors. Davidson's principles and practice of medicine. 17th ed. Edinburgh: Churchill Livingstone; 1995: 865-77.

2.http://my.clevelandclinic.org/orhtopaedics-rheumatology/diseases-conditions/frozen-shoulder-vs-adhesive-capsulitis.aspx (Viewed on 1/10/2013)

3. http://en.wikipedia.org/wiki/Adhesive_capsulitis_of_shoulder (Viewed on 1/10/2013)

4. Anton HA. Frozen shoulder. Can Fam Physician. 1993; 39: 1772-8.

5. Fareed DO, Gallivan WR. Office managment of frozen shoulder syndrome. Clin Orthop. 1989; 242: 177-833.

https://doi.org/10.1097/00003086-198905000-00017

6. Loyd JA, Loyd HM. Adhesive capsulitis of the shoulder: arthrographic diagnosis and treatment. South Med J. 1983; 76: 879-83.

https://doi.org/10.1097/00007611-198307000-00016

PMid:6867799

7. McClure PW, Flowers KR. Treatment of limited shoulder motion: a case study based on biomechanical consideration. Phys Ther. 1992; 72(12): 929-36.

https://doi.org/10.1093/ptj/72.12.929

PMid:1454869

8. Murnaghan JP. Adhesive capsulitis of the shoulder: current concepts and treatment. Orthopdics. 1988; 11(1): 153-8.

9. Neviaser JS. Adhesive capsulitis and the stiff and painful shoulder. Orthop Clin North Am. 1980; 11: 327-31.

10. Parker RD, Rromison AL, Arsham NZ. Frozen shoulder. Part II: treatment by manipulation under anesthesia. Orthopedics. 1989; 12: 989-90.

https://doi.org/10.3928/0147-7447-19890701-12

PMid:2771824

11. Rizk TE, Christopher RP. Adhesive capsulitis (frozen shoulder): a new approach to its management. Arch Phys Med Rehabil. 1983; 64: 29-33.

12. Rizk TE, Pinals RS. Frozen shoulder. Semin Arthritis Rheum.

1982; 11: 440-51.

13. Melzer C, Wallny T, Wirth CH, Hoffman S. Frozen shoulder: treatment and results. Arch Orthop Trauma Surg. 1995; 114: 87-91.

https://doi.org/10.1007/BF00422832

PMid:7734240

14. Bridgman JF. Periarthritis of the shoulder and diabetes mellitus. Ann Rheum Dis. 1972; 31(1): 69-71.

https://doi.org/10.1136/ard.31.1.69

PMid:5008469 PMCid:PMC1005864

15. Lundberg BJ. The frozen shoulder. Clinical and radio graphical observations. The effect of manipulation under general anesthesia. Structure and glycosaminogly can content of the joint capsule. Local bone metabolism. Acta Orthop Scand Supp. 1969; 119: 1-59.

https://doi.org/10.3109/ort.1969.40.suppl-119.01

16. Modesto C, Crespo E, Villas C, Aquerreta D. Adhesive capsulitis. Is it possible in chilhood? Scand J Rheumatol. 1995; 2424(4): 255-256.

https://doi.org/10.3109/03009749509100885

PMid:7481593

DOI: https://doi.org/10.3329/medtoday.v32i1.44837

CASE REPORT OPEN ACCSESS

Lichen Planus in Down Syndrome: Treatment with Systemic Retinoid - an Observation

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Abstract

Introduction: *Down syndrome (DS) is the most common congenital abnormality affecting numerous organs and is associated with an increased incidence of many cutaneous diseases. Lichen Planus (LP) is a quite uncommon disease and associated with many immunologically altered condition. DS is also a medical condition with altered immune function. So, a higher research of LP associated with DS is very important for better understanding of both the diseases and also for comprehensive medical care.* ***Case Report:*** *A 33 years old known patient of Down Syndrome presented with signs and symptoms of LP is reported in this study. The diagnosis of LP was confirmed by histopathological study. She revealed a treatment history of several course of topical and systemic steroid since last 2 years for treatment of LP without any satisfactory outcome. After proper counseling we prescribed her systemic Acitretin (25 mg) daily for 3 months. She was examined every month for subsequent 3 months and was observed very satisfactory result. There was no relapse within one year after discontinuation of treatment.* ***Discussion:*** *We describe an adult patient with Down syndrome, associated with lichen planus (LP). LP is an inflammatory muco-cutaneous disease and benign in nature. However, some varieties of LP may cause considerable discomfort and need various systemic treatment. We treated the patient with systemic Retinoid with satisfactory result.* ***Conclusion:*** *LP associated with DS is a rare condition. It may be treated with systemic Retinoid.*

Keywords: *Lichen planus, Acitretin, Down syndrome.*

Number of Figures: 04; Number of References: 13; Number of Correspondence:03.

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Introduction:

Down syndrome (DS), also known as **trisomy 21**, is an autosomal chromosomal disorder caused by the presence of all or part of a third copy of chromosome 21. It is typically associated with physical growth delays, characteristic facial features and mild to moderate intellectual disability. Down syndrome is one of the most common chromosomal abnormalities in humans with a birth incidence of approximately 1 in 1000 live birth[1]. The cutaneous manifestations of DS are numerous[2,3,4,5,6]. Common associated condition are: xerosis, atopic dermatitis, seborrheic dermatitis, cutaneous infections, alopecia areata, vitiligo, syringoma, elastoses, keratoderma palmaris et plantaris, pityriasis rubra pilaris etc. We describe an adult patient with Down syndrome, associated with lichen planus (LP). LP is an inflammatory muco-cutaneous disease characterized by shiny, flat-topped papules and plaques. It affects around 1% of the population. The disease is usually self-limiting and benign in nature. However, some varieties of LP may cause considerable discomfort and recalcitrant in nature. Owing to advances in medical care and changes in attitude, the life expectancy of the DS patients is improving. A good number of research papers is published in medical literature regarding physical and psychological aspects of this disease. But papers related to LP and other cutaneous aspects of DS are not enough in number. Still, there is no published report on LP associated with DS. But LP is a disease which significantly affect quality of life of patient. The disease is also associated with many immunologically altered condition[7]. DS is also a medical condition with altered immune function[4,8]. So, an appreciation and higher research of LP associated with DS is very important for comprehensive medical care as well as quality of life of these patients.

Case Report:

A 33 years old Bangladeshi female presented with flat topped, violaceous papules and plaques in flexor surface of wrist, trunk and foot for last 2 (two) years. Plaques in foot were polymorphic in shape, 1.5 cm to 2cm in diameter with wickham's striae in surface [Fig:1].

The lesions were slightly hypertrophic and signs of scratching were seen. However, lesions in wrist [Fig-2] and trunk were smaller in size. She complained of severe itching. Her treatment history revealed that she was prescribed both topical and systemic steroid several times with partial outcomes and recurrences.

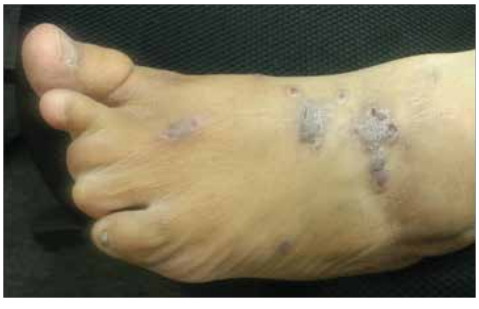

Figure-1: LP lesions in feet.

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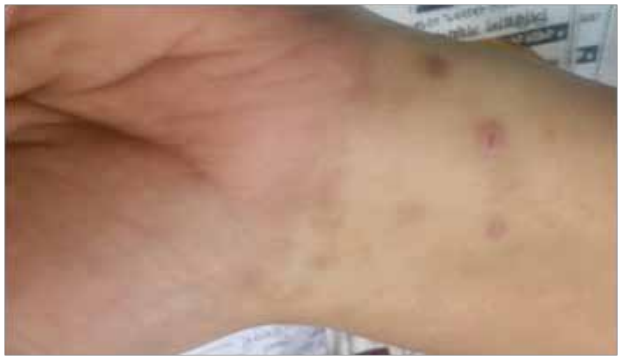

Figure-2: LP lesions in wrist and forearm.

Her physical appearance was compatible with that of DS and following features were observed: epicanthic fold, brachicephaly and depressed nasal bridge, upward angle of eyes, short broad neck and widened hands and foot and shortening of 4 (four) lateral toes. Routine laboratory data were between normal ranges. Her liver and kidney function parameter were within normal range. Histopatholgical study revealed hyperkertatosis, hypergranulosis, vacuolar degeneration of basal layer of epidermis and inflammatory cell infiltrate in upper dermis [Fig:3,4]. These findings are compatible with lichen planus. Her lipid profile was within normal limit. An interview with her family revealed that she had learning disability otherwise she was psychologically sound; She had only one brother and nobody of her family members had any relevant clinical conditions.

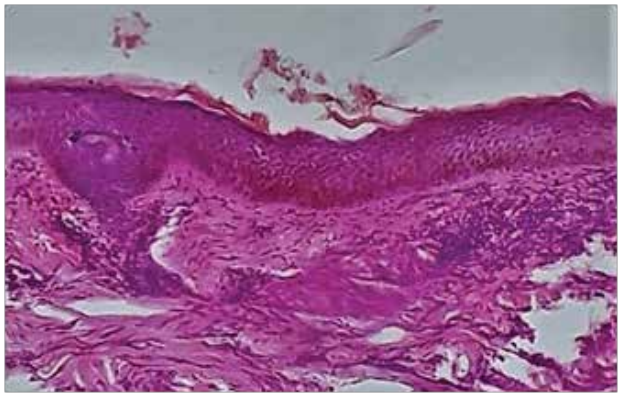

Figure-3: Histopathological feature (H&E staining 100 xs).

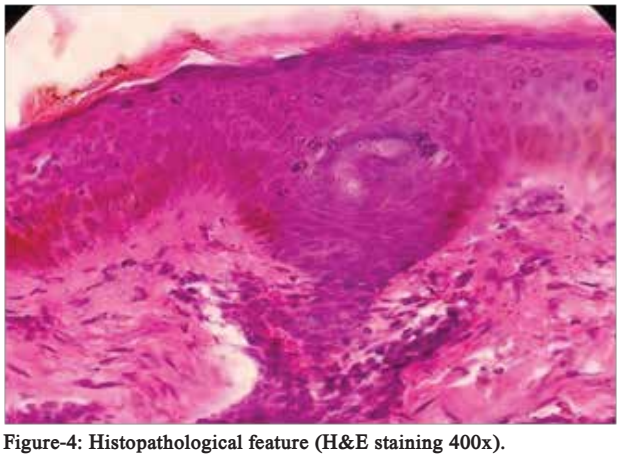

Figure-4: Histopathological feature (H&E staining 400x).

She was a diagnosed case of DS and received several therapies in different institution by both internist and dermatologist. Her past history revealed that she had frequent episodes of bacterial and fungal infection since childhood. She revealed a treatment history of several course of topical and systemic steroid since last 2 yrs without any satisfactory outcome.

After proper counseling we prescribed her systemic Acitretin (25 mg) daily for 1month. She was examined after 1 month and we found her lesions were significantly cured with less intensity of itching. She was prescribed again for Acitretin (25mg) for consecutive 2 month after evaluation of her relevant laboratory parameters. She attended after 1month with 90% clearance of lesions. In her follow up visit after I month, she was found fully cured. Patient did not complain of any significant side effects of the drugs. There was no relapse within one year after discontinuation of treatment.

Discussion:

Few case reports on lichen nitidus with DS were reported in literature but cutaneous LP associated with DS is not reported until now.

LP is one of the common disease in dermatology. The overall prevalence of lichen planus in the general population is about 0.1–4.0% [9]. It generally occurs more commonly in females, in a ratio of 3:2, and most cases are diagnosed between the ages of 30 and 60, but it can occur at any age[9].

Pathogenesis of LP is not fully understood. However, there is a general consensus that LP is an immunologcally-mediated disease, where basal cell damage occurs due to a complex interaction between keratinocytes, Langerhan's cells and T cells[7].

The LP with DS is not a recognized association. However, it is shown in different studies that cutaneous diseases are more frequent in patients of DS than normal population. More ever, it is reported that immune-mediated diseases like atopic dermatitis, psoriasis, seborrheic dermatitis etc are more frequent in patients of DS[4].

LP is usually a self-limiting benign disease some form of the disease may persist for a long duration and may cause a range of morbidity.

Without treatment, most lesions will spontaneously resolve within 6–9 months for cutaneous lesions, and longer for mucosal lesions[10].

More aggressive forms of treatment are needed for the few recalcitrant variants of LP including hypertrophic type. Standard therapy for LP includes topical and systemic steroids, retinoids and PUVA. Acitretin has shown a relatively good efficacy in the treatment of Hypertrophic LP[11,12,13].

In our case, patient was symptomatic, and she was treated with systemic steroid without satisfactory outcome. So, patient had a choice for systemic retinoid and we observed a very good result with these drugs.

Conclusion:

In the medical literature, there is no research report on LP associated with DS. We presented the case to draw attention of the concerned health professional for more research in this arena. At present, the patients of Down syndrome are enjoying a longer life span than before; they deserve the best medical care and quality of life in this 21st century.

Conflict of Interest: None.

Acknowledgement:

Prof Dr. Mohammad Kamal, Ex-Professsor of BSSMU for his kind support and for histopathology.

References:

1. Weijerman, ME; de Winter JP. The care of children with Down syndrome. European journal of pediatrics. 2010; 169 (12): 1445-52.

https://doi.org/10.1007/s00431-010-1253-0

PMid:20632187 PMCid:PMC2962780

2. Carmelo Schepis, Concetta Barone, Maddalone Sirgnsa. An Updated Survey in Skin Conditions in Down Syndrome. Dermatology. 2002; 205: 234-8.

https://doi.org/10.1159/000065859

PMid:12399669

3. Dourmishev A. Mitev V, Pramatarov K, Schartz RA. Cutaneous aspect of Down Sundrome. Cutis. 2000; 66: 420-4.

4. Barankin B, Guenther L. Dermatological manifestations of Down syndrome. J Cutan Med Surg. 2001; 5: 289-93.

https://doi.org/10.1177/120347540100500401

https://doi.org/10.1007/s102270000021

PMid:11907837

5. Brown AC, Olkowaski ZL, Mklaren JR, Kutner MH. Alopecia areata and vitiligo associated with Down nsyndrome. Arch Dermatol. 1977; 113: 1296.

https://doi.org/10.1001/archderm.1977.01640090144032

https://doi.org/10.1001/archderm.113.9.1296b

https://doi.org/10.1001/archderm.113.9.1296

PMid:143244

6. Hazini AR, Rongioletti F, Rebora A. Pityriasis rubra pilaris and vitiligo in Down syndrome. Clin Exp Dermatol. 1988; 13: 334-5.

https://doi.org/10.1111/j.1365-2230.1988.tb00716.x

PMid:2978467

7. Shiohara T, Mariya N, Tanaka Y. Immunopathological study of lichenoid skin diseases. Correlation between HLA-DR positive keratinocytes or langerhans cells and epidermotropic T cells. J Am Acad Dermatol. 1988; 18: 67-74

https://doi.org/10.1016/S0190-9622(88)70011-0

8. MAA Kuters, RH Verstegen, EFA Gemen, E de Vries. Intrinsic defect of the immune system in children in Down syndrome: a review. Clin Exp Immunol. 2009 May;(2): 189-193.

https://doi.org/10.1111/j.1365-2249.2009.03890.x

PMid:19250275 PMCid:PMC2759463

9. Le Cleach, Laurence; Chosidow, Olivier "Lichen Planus". New Eng Jourof Med. 2013 Feb; 366 (8): 723-32.

https://doi.org/10.1056/NEJMcp1103641

PMid:22356325

10. Scully, C.; El-Kom, M. (1 July 1985). Lichen planus: review and update on pathogenesis. Journal of Oral Pathology and Medicine. 1985 Jul. 14 (6): 431-58.

https://doi.org/10.1111/j.1600-0714.1985.tb00516.x

11. Oliver GF, Winklemann RK. Treatment of lichen planus. Drugs. 1993; 45:56-65.

https://doi.org/10.2165/00003495-199345010-00006

PMid:7680985

12. Alamri A, Alsenaid A, Ruzicka T, Wolf R. Hypertrophic Lichen Planus- Successful treatment with acitretin. Dematol Ther. 2016 May; 29(3): 173-6.

https://doi.org/10.1111/dth.12337

PMid:26809380

13. Vazirnia A, Cohen P. Acitretin for the management of genealized cutaneous lichen planus. Dermatol Online J. 2014 Sep; 20(9): 25244164.

DOI: https://doi.org/10.3329/medtoday.v32i1.44838

CASE REPORT OPEN ACCSESS

Management of Pancreatic Calculi – an Experience of Ten Cases in a District Hospital

Md. Rafiqul Islam*[1], Kazi Moinur Rahman[2], Gazi Shafiqur Rahman[3]

Abstract

Introduction: *Pancreatic calculi (PC) are not an uncommon surgical condition in daily practice.Alcohol, smoking, genetic factors, metabolic disturbances and defects in immunity are some of the known etiological agents. The common presentations are severe abdominal pain, vomiting, malabsorbtion, weight loss, diabetes mellitus etc.* ***Case Report:*** *We have managed ten cases in our hospital,Initial medical treatment was given to all of the patients, followed by open surgical procedure.* ***Discussion:*** *Endoscopic stone removal is the best procedure in a higher center. Open surgical procedure is needed in some cases.* ***Conclusion:*** *We have done ten cases of open pancreatolithotomy in our district hospital. The outcome of the procedure is satisfactory.*

Keywords: *Pancreatic calculi (PC), Chronic pancreatitis (CP), Pancreaticojejunosetomy.*

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Introduction:

Chronic pancreatitis (CP) is a disease of diverse etiology characterized by progressive and irreversible changes in the pancreas, resulting in loss of exocrine and endocrine functions. Pain, either continuous or episodic, is the dominant and distressing feature of this illness and significantly worsens the quality of life. Alcohol, smoking, genetic factors, metabolic disturbances and defects in immunity are some of the known etiological agents[1]. While alcohol is the commonest etiological agent in most industrialized countries, the nonalcoholic idiopathic type of CP is more prevalent in some countries[2–4]. Pancreatic calculi (PC) are the sequelae of CP and can occur in about 50% of patients[5]. These calculi aggravate or produce the typical pancreatic pain experienced by patients, by obstructing pancreatic ducts and producing upstream ductal hypertension and subsequent parenchymal hypertension. Therapy, either endoscopic or surgical aims at clearing these calculi and reducing the ductal hypertension, relieving pain and improving quality of life. PC seen in the nonalcoholic, idiopathic variety of CP tend to be large and denser than those seen in the alcoholic variety[6,7].

Case Report: Classification of Pancreatic Calculi

PCs are classified on the basis of type, numbers and location. They may be (1) radio opaque, radiolucent or mixed; (2) single or multiple; (3) located in the main pancreatic duct (MPD), side branches or in the pancreatic parenchyma; and (4) located in head, body or tail regions (Fig. 1). Majority of PCs are radio opaque while a few are radiolucent or mixed[2].

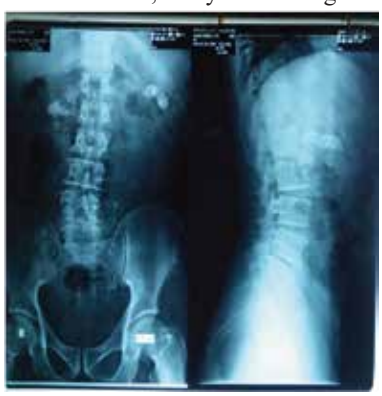

Figure-1: Plain x-ray abdomen in erect posture A/P and Lateral view. Showing radio-opaque shadows of pancreatic stone.

Diagnosis

Patient with pancreatic calculi usually presents with characteristic abdominal pain, associatedwith vomiting. A plain x-ray of abdomen is sometime enough to diagnose the case. There are radiopaque shadows present at the level of body of L1-L2 or on either side. Other investigations are Complete blood count, Blood sugar level, Serum creatinine, Blood grouping, Serum amylase / lipase, Ultrasonography of abdomen, CT Scan, MRI, ERCP etc.

Further investigations are necessary to diagnose the comorbidities and anaesthetic fitness such as x-ray chest, ECG, Echocardiography etc.

Pathogenesis and Composition

Pancreatic stone protein (PSP) plays a key role in the formation of PC[8]. Various factors including gene expression, cause a reduction in PSP. Reduction in PSP results in supersaturation of calcium carbonate in the pancreatic juice. This calcium carbonate is then deposited over an inner nidus. Irrespective of the etiology of CP, the structure and composition of PC are the same suggesting a common pathway for pancreatolithiasis[9].

Scanning electron microscopy and energy dispersive X-ray fluorescence have revealed that all PC have an amorphous nidus, which forms the center of the PC. The nidus contains elements such as nickel, iron and chromium. It is over this nidus that calcium carbonate in the form of

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calcite is deposited in multiple layers and over multiple stages[9].

Modalities of Treatment

As mentioned earlier pain is the dominant symptom in patients with CP and calculi contribute by obstructing the pancreatic ducts and increasing upstream hypertension. A coexisting ductal stricture can exacerbate the preexisting hypertension.

The following are the modalities of therapy for removal of PC.

1. Endoscopic therapy: this includes endoscopic retrograde cholangiopancreatography (ERCP) and stone extraction and ESWL.

2. Surgical therapy: both drainage and resection procedures are widely used in the surgical management of CP with calculi.

3. Dissolution of PC: even though chemical agents such as trimethadione have been earlier shown to dissolve stones, these are seldom used in modern day practice.

Figure-2: Pancreatic calculi.

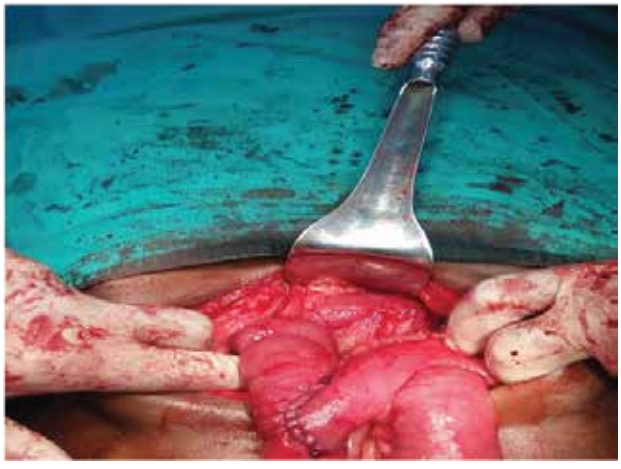

Figure-3: Lateral pancreatojejunosetomy with Roux-en-Y anastomosis.

Discussion:

We have treated ten cases of pancreatic calculi. All the cases were diagnosed by careful history taking, clinical examination with relevant investigations. Out of ten cases one is female patient. Ages are ranging from 22 years to 65 years. Initially conservative medical treatment was given to every patient but no significant improvement was there. Patient with multiple stones (Fig.2) and dilated main pancreatic duct were selected for open surgical procedure. With all aseptic precaution laparotomy was done, lateralpancreaticojejunosetomy with Roux-en-y anastomosis (Fig.3) were done in six cases, lateral pancreaticojejunosetomy with jejunojejunosetomy were done in two cases, Shahid procedure (lateral side to sidepancreaticojejunosetomy) were done in two cases. The outcome of the treatment were satisfactory, four patients need two or three visits postoperatively for abdominal pain, which were improved on conservative treatment.

Conclusion:

PC are the natural sequel of the ongoing process of CP. Pain is the dominant symptom of patients with CP and various endoscopy and surgical therapy aims at reducing this pain by eliminating the calculi. Small PC are cleared by the standard technique of pancreatic sphincterotomy (PS) followed by balloon trawl or basket. Large calculi in uncomplicated patients should be subjected to ESWL for fragmentation prior to a subsequent ERCP. In properly selected patients ESWL is an efficient and useful tool and provides adequate long-term relief[10]. Patients with extensive calculi, multiple strictures, suspicious mass lesions and those who have failed endotherapy are ideal candidates for surgery. The outcome of our cases after long term follow up is satisfactory.

Consent

Prior informed consent was obtained from the patient for publication of this case study and any accompanying images. All images are taken by the authors won cell phone during operation.

Conflict of Interest: None.

Acknowledgement:

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References:

1. Lankisch PG, Banks PA. Pancreatitis. New York: Springer-Verlag; 1998.

2. Tandan M, Reddy DN, Santosh D. Extracorporeal shock wave lithotripsy and endotherapy for pancreatic calculi: a large single center experience. Indian J Gastroenterol. 2010; 29: 143–148. doi: 10.1007/s12664-010-0035-y.

3. Bhasin DK, Singh G, Rana SS. Clinical profile of idiopathic chronic pancreatitis in North India. ClinGastroenterolHepatol. 20097594–599. doi: 10.1016/j.cgh.2009.01.009.

4. Balakrishnan V, Unnikrishnan AG, Thomas V. Chronic pancreatitis: a prospective nationwide study of 1,086 subjects from India. JOP. 2008; 9: 593–600.

5. Rosch T, Daniel S, Scholz M. Endoscopic treatment of chronic pancreatitis: a multicenter study of 1000 patients with long-term follow-up. Endoscopy. 2002; 34: 765–771. doi: 10.1055/s-2002-34256.

6. Ong WC, Tandan M, Reddy V, Rao GV, Reddy N. Multiple main pancreatic duct stones in tropical pancreatitis: safe clearance with extracorporeal shockwave lithotripsy. Gastroenterol Hepatol. 2006; 21:1514–1518. doi: 10.1111/j.1440-1746.2006.04224.x.

7. Chari S, Jayanthi V, Mohan V, Malathi S, Madanagopalan N, Viswanathan M. Radiological appearance of pancreatic calculi in tropical versus alcoholic chronic pancreatitis. J Gastroenterol Hepatol. 1992; 7: 42–44. doi: 10.1111/j.1440-1746.1992.tb00932.x.

8. Jin CX, Naruse S, Kitagawa M. Pancreatic stone protein of pancreatic calculi in chronic calcified pancreatitis in man. JOP. 2002; 3: 54–61.

9. Pitchumoni CS, Viswanathan KV, Gee Varghese PJ, Banks PA. Ultrastructure and elemental composition of human pancreatic calculi. Pancreas. 1987; 2: 152–158. doi: 10.1097/00006676-198703000-00005

10. Tandan M, Nageshwar Reddy D. Endotherapy in chronic pancreatitis. World J Gastroenterol. 2013; 19: 6156–6164. doi: 10.3748/wjg.v19.i37.6156.

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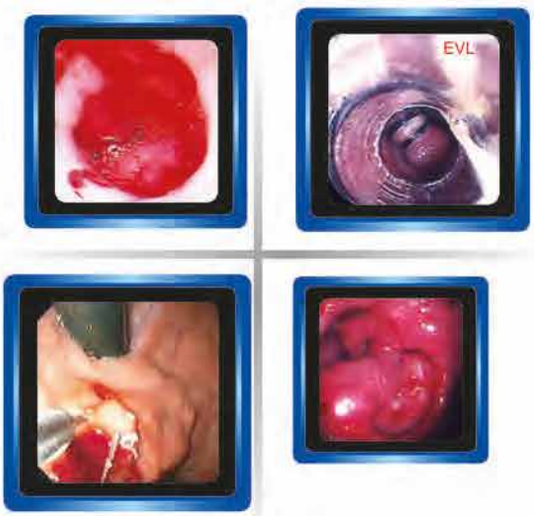

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