ISSN 1992-2426

Osmani Medical Teachers Association Journal

Volume 4: Number 2
July 2005

Original Articles

Conservative Injection Treatment of Tennis Elbow.....3
SHOME PS, UDDIN MH, BARBHUIYAN MMA

Study of 50 Cases of Corneal Injury In Sylhet Osmani MAG Medical Collage Hospital.....6
CHOUDHURY SA, CHANDHURY SH, DUTTA C

Report on Investigation of Antibiotic Resistant Klebsiella pneumoniae at Elsewhere Hospital in the United Kingdom 9
HAQUE MM

Outcome of Rubber Band Ligation in the Treatment of Haemorrhoids Using Suction Ligator 13
DAUD AKM, KHAN AJ

Study on Prevalence and Pattern of Visceral Injury Following Blunt Trauma in a Peripheral Teaching Hospital 17
MAMMUD MK, AHMED MM, SINGHA PR, ALAM MM

Case Reports

Maternal Anaphylactic Reaction During Termination of Pregnancy by Induction of Labor 27
MAHAL MF, UDDIN MH

Leech in the Peritoneum Cavity: An Unusual Casue of Intestinal Obstruction 30
RAHMAN MS

Review Article

Conservative Management of Tennis Elbow: An Overview 33
ISLAM MH

Occasional Note

Prevention of Noise-Induced Hearing Loss 37
ABDULLAH M, OSMANY HQ, CHOWDHURY MR

Editorial

Bird Flu: An Emerging Threat to Mankind 41
PATWARY MI

Information for the Contributors.....44

Teachers Association, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh

Osmani Medical Teachers Association Journal (OMTAJ)

Official Organ of the Teachers Association, Sylhet MAG Osmani Medical College

EDITORIAL OFFICE:

Department of Anesthesiology, Sylhet MAG Osmani Medical College

Telephone : 812121, Extension: 505, 717199

E-mail: shanewaz@btsnet.net

Teachers Association, Sylhet MAG Osmani Medical College

Executive Committee

President
Professor Md. Rezaul Karim

Vice Presidents
Dr. MA Matin
Professor Osul A Chowdhury

General Secretary
Dr. Md. Mustaque Ahmed Barbhuiyan

Treasurer
Dr. Kazi Akter Uddin

Joint Secretary
Dr. Md. Siraj Uddin

Organizing Secretary
Dr. Fazlur Rahim Kaiser

Members
Professor Md. Arif Mian
Dr. Md. Ismail Patwary
Dr. Shibbir Ahmed
Dr. Promode Ranjan Singh
Dr. Helal Uddin
Dr. Shamimur Raman
Dr. Abu Ahmed Adiluzzaman
Dr. Rafiques Salehin
Dr. Md. Shahnewaz Choudhury
Dr. Shamim Akhter Mimi

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Conservative Injection Treatment of Tennis Elbow

*PARTHA SARATHI SHOME[a] , MD. HELAL UDDIN[b], MD. MUSTAQUE AHMED BARBHUIYAN[c]

Abstract

From June 1995 to June 2000, 50 patients having Tennis elbow underwent treatment by 40 mg methyl-prednisolone with 1.5 cc 2% lignocaine. The patients were non-diabetic and having no other rheumatic disorder. The mean follow up period was 15 month (6 months to 24 months). In final follow up, 10 patients (20%) responded well with single injection, 25 patients (50%) with double and 10 patients (20%) with triple injection. Only 5 patients (10%) did not respond at all with injection treatment. Intralesional injection treatment in Tennis elbow causes perfect recovery in most of the cases and the result is statistically significant.

[OMTAJ 2005; 4(2)]

Introduction

The Tennis elbow or lateral epicondylitis is the commonest lesion of the insertion of muscle or tendon onto bone. A tear occurs in or near the insertion of the common extensor tendon on the lateral condyle and humerus. The injury is caused by sharp flexion of the wrist while the extensors are contracted, often by hitting a tennis ball awkwardly during a backhand stroke. The injury also occurs in everyday activities such as gardening and lifting. [1]

On examination, the lateral epicondyle of humerus is tender and stressing the extensor origin, by forcing the wrist into flexion with the extensors contracted, reproduces the symptoms. The treatment basically is of two types, i.e., conservative and surgery. Conservative treatment include intralesional injection of methyl-prednisolone, rest, ultrasound, laser, massage, manipulation, etc.[2] This study has been designed to evaluate the effectiveness of intralesional injection of 1 cc of methyl-prednisolone (Depomedrol 40 mg) with 1.5 cc of 2% Lignocaine.

Methods

Between June 1995 to June 2000, a total of 50 patients with Tennis elbow, underwent treatment with local infiltration of methyl prednisolone 40 mg (Inj. Depomedrol) mixed with 1.5 ml of 2% lignocaine (Lignocaine). Clinical assessment was done with history of pain, which aggravates on griping and resistive palmar flexion in fully extended elbow and pronated forearm, duration of pain, and localized tenderness over the origin of common extensors. The patients belonging to diabetic mellitus and other rheumatic disorders were excluded from the study. After confirmation of the diagnosis clinically, under all aseptic precautions, 1 ml (40 mg) injection methyl-prednisolone, mixed with 1.5 ml 2% lignocaine, was infiltrated under the common extensors origin at the lateral epicondyle, especially under the origin of extensor carpiradialis brevis. Care taken so that drug was not infiltrated into the muscle. Non-steroid acute inflammatory drugs (NSAIDs) and ranitidine were given orally for 7 to 10 days with 12 hour interval.

Results

The results was analyzed on the basis of patients statements regarding the relief of pain and clinical judgment by Resistive Tennis Elbow test (Cozen's Test) and Passive Tennis Elbow Test.[3]

In this study 10 patients 20% responded very well (Get rid of by single injection). In 25 patients (50%) second injection was installed 6 weeks after first injection due to in adequate response and another 10 patients 20% needed third injection infiltration for

a. Professor, Department of Orthopaedic Surgery, Sylhet MAG Osmani Medical College, Sylhet.

b. Assistant Professor, Department of Anesthesiology, Sylhet MAG Osmani Medical College, Sylhet.

c. Assistant Professor, Department of Surgery, Sylhet MAG Osmani Medical College, Sylhet.

* Corresponding author

perfect recovery after 6 weeks interval from second injection. Only 5 (10%) patients in this study did not

Table I : Age distribution of the patients

Age Group	No of patients	Percent
30-40 years	35	70%
40-50 years	15	30%

Table II : Sex incidence

Patients	No of patients	Percent
Male	20	40%
Female	30	60%

Table III : Occupation distribution of the patients

Occupation	No of patients	Percent
House wife	25	50%
Manual worker, Carpenters	10	20%
Service man	5	10%
Tennis player	5	10%
Business man	3	6%
others	2	4%
Total	50	100%

Table IV : Over all results in final follow up

No of injection	No of patients	Percent
Single	25	50%
Double	10	20%
Triple	5	10%
No response	5	10%

Prevalence of the disease was more common among house wives and manual workers. (Table III) The highest recovery (50%) was found in 25 patients by double injection. (Table IV)

The disease shows the highest incidence (70%) among age group between 30-40 years. (Table I). Prevalence of the disease was found high (60%) among the female. (Table II) House wives and manual workers were the highly (70%) vulnerable groups. (Table III) Tennis player were not found highly prone (10%) to the disease. The highest recovery rates (50%) were found in patients receiving double injection and 5 patients (10%) did not show any positive response. (Table -IV)

Discussion

In this study, a high incidence of the disease was found among the age group between 30-40 years and prevalence of the disease was higher among the female sex (60%), which is similar with the study by Talukder in RIHD. [4]

Coonrad and Hooper described that Tennis elbow is a chronic disabling disorder of unknown cause, seen commonly in patients who have occupation requiring rotary motion at the elbow, such as pipe fitters, tennis players, and carpenters.[5] This study is in consistent with the previous one but here the prevalence of the disease is very common among the housewives and manual workers which slightly differ from Coonrad's study. In this study, the vulnerability of the Tennis players was low (10%) which slightly differs from Boyd's study where it was in the moderate profile. [6]

Corticosteroids injections are useful in the treatment of Tennis elbow. Up to three injections is considered appropriate. If three injections are unsuccessful in relieving the symptoms, a release of the extensor origin from humerus must be considered.[8] Andrew described the untoward effect of repeated local injection of hydrocortisone and advocated to avoid repeated injections.[9] In this study, this is quite consistent with the previous studies. Here, 10 patients (20%) were cured by single injection, 25 patients (50%) with double injections and 10 patients (20%) responded well after triple injections. Only in 5 patients (10%), who did not respond well after repeated injections, no further injection was instituted.

Tennis elbow or lateral epicondylitis is a common problem which can be diagnosed easily from history of pain on griping and resisted palmar flexion of the wrist in fully extended elbow and pronated forearm. There is localized tenderness over lateral epicondyle. It can be easily treated with intralesional injection of long acting,

one to three corticosteroid injections (methyl prednisolone) 40 mg with 1.5 cc of (2%) lignocaine.

Reference

1. Dandy DJ. Essential Orthopaedics and Trauma, 2nd edition. London: Churchill Livingstone 1993: p. 361.
2. Skinner HB. Current diagnosis and Treatment in Orthopedics, 1st edition. UK: Hall International Inc. 1995: p. 161.
3. Konin, Wiksten, Isear. Special Tests for Orthopedic Examination, 1st edition. New Jersey: Slack 1997: pp. 85-6.
4. Talukder DN, Roy NK, Bairagi G. Results of the intralesional injection of methyl Prednisolone with local anaesthetic in Tennis elbow. J Bangladesh Orthopaed Soc 1999; 14 (1): 19-20.
5. Coonrad RW, Hooper WR. Tennis elbow: Its course, natural history conservative and surgical management. J Bone Joint Surg 1973; 55 (6): 1177.
6. Boyd HB, McLeod AD. Tennis elbow. J Bone Joint Surg 1973; 55 (6): 1183.
7. Curl WW. Office treatment of elbow injuries in the athlete: Sports, Exercise, Overuse Injuries. Instr Course Lect 1995; 43: 55-61.
8. Doran A, Gresham GA, Rushton N, *et al.* Tennis elbow. Acta Orthop Scand 1990; 61 (6): 535.
9. Russell RCG, Williams NS, Bulstrode CJK. Bailey & Love's Short Practice of Surgery, 23rd edition. USA: Arnold publishers 2000: pp. 345-6.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Study of 50 Cases of Corneal Injury In Sylhet MAG Medical Collage Hospital

*SHAMSUL ALAM CHOUDHURY[a], SHAKWAT HOSSAIN CHANDHURY[b], CHANDRA DUTTA[c]

Abstract

Corneal injuries are important causes of visual impairment and blindness in Bangladesh, affecting both the children and the adults. In this study, 50 cases of corneal injuries were studied in Sylhet MAG Osmani Medical Collage Hospital in the indoor department from July, 2001 to July, 2002. Most of the cases were full thickness corneal injuries with iris prolapses (%) and others such as corneal injury with anterior dislocations, blunt corneal abrasion with hyphaema, etc. Age incidence was from 11 to 30 years predominantly (64%), and sex incidence was male predominant (84%). Full thickness corneal injuries were more common than partial thickness. To prevent these injuries, eradication of poverty, illiteracy, prejudices are all important. Surgical management by skilled surgeons with good microsurgical facilities on an emergency basis, plays a vital role in preventing blindness and early restoration of vision and cosmetic benefits.

[OMTAJ 2005; 4(2)]

Introduction

Occurrence of corneal injuries is very common in children and adults in our country, causing unilateral impairment of vision and blindness. Injuries occur by sharp cutting weapons, blunt injuries predominately. Chemical injuries are, however, not uncommon. Earliest surgical management by skilled surgeon with meticulous care is the pre-requisite for the best visual prognosis and cosmetic results. Latest microsurgical instruments with good operating microscopes and appropriate surgical stitches and timely stitch-removal are also important factors for getting the best surgical outcome. Both sharp pointed and blunt objects, such as pencil, pen, needles, bamboo sticks, glass, stones, and toys, etc. are liable.

a. Assistant Professor (cc),

b. Associate Professor (cc),

Department of Ophthalmology, Sylhet MAG Osmani Medical College

c. Assistant Registrar, Department of Ophthalmology, Sylhet MAG Osmani Medical College Hospital

* Corresponding author

Methods

A prospective study, made on 50 cases of corneal injuries in MAG Osmani Medical College in indoor eye patient department, were studied. Patients up to 60 years of age with only conical injuries of were included. Exclusion criteria were badly injured eyes, ruptured globes.

Results

A total of 50 patients with corneal injury was included in this study. Age incidence of majority of the patients (36%) was from 21 to 30 years (Table I). Results showed that males (86%) were affected more than the females (16%) (Table II). Full thickness corneal injuries with prolapses of the Iris were greater in occurrence (20%), followed by corneal injuries with dislocation of the lens (16%) predominating nextly. Foreign body trauma and acid and alkali burns were less common findings of corneal injury (6%). (Table IV).

Table I: Age groups of the patients (n=50)

Age groups in years	Number of cases	Percent
0-10	05	10
11-20	14	28
21-30	18	36
31-40	04	08
41-50	06	12
51-60	02	04
61-70	01	02

Table II: Distribution of sex of the patients (n=50)

Sex	Number of cases	Percent
Male	43	86
Female	07	14

Table III: Types of Corneal Injury (n=50)

Corrected Visual Activity	Number of cases	Percent
NPL (Blind)	05	10
Hand movement	03	06
1/60	02	04
3/60	03	06
6/60	07	14
6/36	08	16
6/24	05	10
6/18	10	20
6/12	04	08
6/9	02	04
6/6	01	02

Table IV: Final Visual Outcome and Prognosis (n=50)

Sl no	Nature of the Injury	Number of Cases	Percent
01	Simple Corneal Abrasions and Foreign Body	04	08
02	Partial Thickness Corneal Injury with Desmetocoele	03	06
03	Full Thickness Corneal Injury with Iris incarceration	05	10
04	Full Thickness Corneal Injury with Iris Prolapses	10	20
05	Full Thickness Corneal Injury with Iridodialysis	02	04
06	Full Thickness Corneal Injury with anterior dislocation of Lens and Capsule Rupture	08	16
07	Chemical Injury (Acid or Alkali Burns)	03	06
08	Full Thickness Corneal Injury with Vitreous Prolapse	05	10
09	Blunt Trauma with Hyphaema	06	12
10	Blunt Trauma with Vitreous Haemorrhage	02	04
11	Blunt Trauma with Retinal Detachment	01	02
11	Blunt Trauma with Optic Atrophy	01	02
	Total	50	100

Discussion

The nature of the injuries ranged from both sharp cutting wounds to ragged lacerated wounds. Both full thickness and partial thickness wounds were found. The sites of the injuries were in central and peripheral areas of the cornea. Peripheral cornea! injuries involved the Sclera, Ciliary body as well as Lens capsule. Rupture, with or without lens dislocation, both anteriorly and posteriorly were also, however, not uncommon. Various prolapses, loss or haemorrhage, and retinal detachments were less common.

Some cases were simple abrasions, rupture of descemets membrane, corneal endothelial damages, blood staining of the cornea due to hyphaema were also reported. Since the central corneal injuries involved the nodal point, there was gross impairment of vision. Injuries involving the periphery of the cornea leads to minimal impairment of vision. Even vision of up to 6/6 could be attained (2%) in these instances (Table III).

Occurrence of corneal injuries were found very common in our country (57%) as shown in a study by Manzur *et al* and as 54% by Islam *et al*.[3] Peak age group of corneal injuries were found in interval of 11-30 years, nearly similar to the study by Islam *et el.* Male incidence were more (84%) versus female which is also similar to work of the previous authors. Age predominance from 11-30 years was due to major working groups in this range. More male predominance is attributable to the same reason.

For a successful outcome, a very skilled and meticulous surgery is needed by senior skilled ophthalmic surgeons. Timely repair by senior skilled ophthalmic surgeons, with thorough asepsis, and proper post-operative cares, are all important preconditions in attaining a good visual outcome and a better cosmetic result. Lack of initiatives by the attendants of the patients owing to poverty and ignorance and lack of microsurgical facilities in the district hospital as well as trained surgical personnel are the additional causes of failure of successful surgical repairs. Shortage of anaesthetic personnel is also another important cause, because corneal repair needs GA facilities for better surgical outcome.

References

1. Roper Hall MJ. Immediate management of iris and lens in perforations of the eye. Trans. Ophthalmol Soc 1980; 102: 221-4.
2. Lamkin JC, Azar DT, Mead MD. Simultaneous corneal lacerations repair, cataract removal and posterior chamber intraocular lens implantation, chamber intraocular lens implantation. Am J Ophthalmol 1992; 113 (6): 326-31.
3. Chowdhury ZA. Types and management of ocular injuries in Dhaka Medical College Hospital -A study of 50 cases. 1139, 1992.
4. Hossain MM, Hadi MA, Perforating corneal injury .....& its management. Trans ophth. December 2001, 28 (2): 20-23
5. Islam MN, Hossain A, Ocular Ocular injuries Prospective study & Prevention Trans ophth. December 2001: 28(2): 42-44.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Report on Investigation of Antibiotic Resistant *Klebsiella pneumoniae* at Elsewhere Hospital in the United Kingdom

MD. MOYNUL HAQUE[a]

Abstract

Eight isolates (A-H) of multi-drug-resistant *Klebsiella pneumoniae* causing cluster of infections at Elsewhere Hospital Intensive Care Unit were investigated. Control organisms P4291, 50192, RP4+ and JM83 were also used for the experiments. Four experiments were carried out to investigate these isolates, which included strain typing, plasmid analysis, PCR, and colony blot. In the experiment of strain typing, two types of *Klebsiella pneumoniae* causing cluster of infections in the Hospital Intensive Care unit were found. Six among the isolates (A, B, C, F, G, and H) of the organism were similar and they were found to be of one type of strains, but two others (D and E) were not similar and they were found to be another type of strains. In plasmid analysis, clearly visible bands were found in four of the isolates (C, F, G and H), and they were found carrying different types of plasmids. In two of the isolates (C and F), the size of plasmid was 150 ±10 kb and, in other two (G and H), size of the plasmids was 150 ±15 kb. But no clear bands could be found in five among the isolates (A, B, E, D and E). In PCR, all of the isolates showed TEM gene in gel photographs. Sequencing of DNA of one of the isolates (A) had been done and compared with known TEM sequence found in the Web site. It was found to be TEM-10 family-beta-lactamase. No result could be found from colony blot experiment, and it was probably due to experimental error as positive control did not give any result.

[OMTAJ 4 (2): July 2005:]

Introduction

The significance of antibiotic resistant organism is very diverse. It is one of the most important causes of hospital-acquired infections. It brings a major limitation on clinical use of antibiotics. It is also involved in many community-acquired infections. Now-a-days, the clinical problem in the hospitals caused by antibiotic-resistant-organisms have been recognized in an increasing frequency. Hospital infections caused by nosocomially acquired Gram-negative bacteria, especially *Klebsiella pneumoniae*, produce extended spectrum beta-lactamase (ESBL) as an antibiotic resistance mechanism. The production of ESBL by *Klebsiella pneumoniae* causes a widespread nosocomial problem. These types of multi-drug-resistant organisms are now spreading in the hospital settings and have become important nosocomial pathogens. They produce difficult problem, particularly for intensive care units, cystic fibrosis centres, burn units, oncology and neonatal wards where clusters of patients are highly susceptible to infections. In Intensive Care Units, antibiotic use is the heaviest and have the greatest potential for patient-to-patient transmission of diseases. On the other hand, hospital strains are often resistant to multiple antibiotics, mainly due to acquisition of plasmids carrying several genes that encode the enzyme to mediate resistance. So, infections with resistant organisms result in avoidable failure of treatment and increased cost in patients receiving inappropriate antibiotic treatment. As a result, it causes prolonged hospital stay as well as devastating or even fatal consequences.

Methods

Four procedures were carried out to investigate the isolates A, B, C, D, E, F, G and H of *Klebsiella pneumoniae* found to cause clusters of infections in the Elsewhere Hospital in the UK. The investigations were as follows:

A. Strain typing by: a) DNA extraction: From an over night broth culture of each isolate, extraction of DNA done. b) Restriction digest DNA: Restriction

a Assistant Professor,
Department of Microbiology, Sylhet MAG Osmani Medical College, Sylhet-3100

digestion of DNA was done by restriction enzymes. For the restriction digestion, *Sma*I enzyme plus buffer mix were added. c) Set up gel (electrophoresis of restriction digest): Each digest mixed with loaded buffer and loaded each sample on the maxi-gel. The gel ran, stained and photographed. d) The gel were viewed and analyzed by looking at photographs from different isolates and comparing the bands patterns of the isolates with the control ones. [1,2]

B. Plasmid analysis: done by: a) Extraction of plasmid DNA: The rapid method of plasmid extraction by Kado and Liu was used and extraction of plasmid DNA was done. b) Plasmid electrophoresis: done by loading of plasmid preparation in mini-gel system. The gel comprises 0.7% w/v agarose in TBE (0.089 M Tris.0.089 boric acid and 2 mM EDTA). b) Analysis of results: Looking at the pictures and comparing with the positive and negative controls bands corresponding to the plasmids were worked out.[3]

C. PCR: was done by the following steps: a) Set up PCR reaction: Part of DNA extracted from the isolates for strain typing was used for PCR, and water used for negative control. We used A pair of primers were used and a Reddy mix (PCR master mix, AB gene, including nucleotides, buffer, polymerase, and a dye for easy visualization) was used. b) Electrophoresis of the PCR products: loading of each sample on mini-gel done. The gel ran, stained and photographed. c) Analysis of results: Looked at gel photographs, compared with the positive and negative controls. d) DNA sequence: PCR product of the isolate A was sequenced by one of the teachers and supplied to find out which TEM gene it was.[4]

D. Colony blot: In this experiment, hybridization instead of PCR was used to detect the presence or absence of TEM gene. a) Inoculate filters: A nutrient agar plate was used and a piece of "Hybond-N" filter' was placed on the nutrient agar plate and transferred a small quantity (one colony) of each isolate on to the filter in the marked position. b) Colony lysis and hybridization: lysis of the cells grown on filter was done and setting up hybridization with the TEM probe. c) Probe detection: The labeled probe was detected that has hybridized to the filter, i.e., how many isolates can hybridize with the TEM probe?

Results

Strain typing:

The photographs of gel analysis of all the isolates showed clearly visible bands in different strains in comparison with the photographs of the control strains. There was a marker in the photographs, the sizes of which was shown in kilobases (kbs) to compare the sizes also. From the photographs, it was clearly visible that the strains A, B, C, F, G and H have got similar type of bands, so, they were of same type of strains. The isolates D and E showed similar bands, so, they were another type of strains. Now, we conclude that there were two types of multi-drug-resistant *Klebsiella pneumoniae* causing cluster of infections in Hospital Intensive Care Units showing resistance to third generation cephalosporins.

Plasmid analysis:

Photographs of gel electrophoresis of the isolates C, F, G, and H showed clear bands in comparison with the control organism 50192. But the isolates A, B, D, and E did not show clearly visible bands. But the sizes of the plasmids were different in different isolates, which was compared with the marker. The sizes of the plasmids in the isolates C, and F were 150 ±10 kbs each, but the sizes of the plasmids in G and H were 150 ±15 kbs. So, different sizes of plasmids were present in C, F, G and H, i.e., the isolates C, F, G and H contained different types of plasmids.

PCR analysis:

Photographs of the gel analysis of the PCR showed all the isolates A, B, C, D, E, F, G, and H have got TEM gene. It was compared with the control organism RP4+ and also compared with the marker. Sequencing of the DNA from isolate A was done and compared with the published TEM sequences from website kept by Bush and Jacoby at "http//www.lahey.org/studies/webt.stm and found that it was TEM 10 family-beta-lactamase.

Table I: Results of Plasmid analysis, Strain typing, PCR, and Colony blot

Isolate	Plasmid detection	Plasmid size (kb)	Strain typing
A	-	-	i
B	-	-	t
C	+	150 ± 10	t
D	-	-	2
E	-	-	2
F	+	150 ± 10	1
G	+	150 ± 15	1
H	+	150 ± 15	1

Colony blot:

From this experiment, we could not get any results and it was probably due to experimental error, because positive control also did not give any result.

Discussion

Four experiments were done on the multi-drug-resistant *Klebsiella pneumoniae* isolates A, B, C, D, E, F, G and H. These were strain typing, plasmid analysis, PCR, and Colony blot. From the strain typing, we found that two different strains *of Klebsiella pneumoniae* caused cluster of infections at Elsewhere Hospital Intensive Care Unit. From the plasmid analysis, we found that different type of plasmids were present in different strains *of Klebsiella pneumoniae.* From the PCR results, we found that all the isolates contained TEM gene. Sequencing of DNA from isolate A was done and compared with the published TEM sequences from website and found that it was TEM-10 family-beta-lactamase. So, from the above mentioned results of the different experiments, it can be concluded that two types of *Klebsiella pneumoniae* carrying different types of plasmids, and each of them containing TEM-10 family-beta-lactamase gene caused the cluster of infections in Intensive Care Units.

But the experiments, which were carried out, got some limitations also. In case of PCR, as it id known to be about 10-fold more sensitive, and extreme sensitivity may cause problem, can potentially detect dead organisms also. One major problem with PCR is that it may cause false negative reactions due to the introduction of contaminating nucleic acid into the reaction mixture.

In plasmid analysis, the agarose gel method (mini-gel), the larger molecules greater than 40,000 bps were not separated from one another. So, all the plasmids in all of the isolates may not be visualized properly. In colony blot, no result was obtained, so, if it would be done again, it might give a better result.

Now, it can be thought that if other experiments could be performed, which could help to clarify, or extend the results. These experiments include: (i) Pulsed field gel electrophoresis (PFGE): by which the larger molecules, more than 40,000 bps could be identified. It separates the large fragments, involves sending short pulses of electricity to an array of electrodes surrounding the agarose gel. The DNA fragments are clearly separated into resolved bands on electrophoresis. (ii) Restriction fragment length polymorphism: here digestion of bacteria done by restriction endonucleases and cut DNA at specific recognition sequence composed of 4 to 6 basepairs are identified. After gel electrophoresis in agarose gel, visualization is done by staining the gel with ethidium bromide. By using this method, isolates from nosocomial outbreaks or clusters can be analyzed to see if they have same or different restriction fragment length polymorphism. (iii) Ribotyping: ribosomal RNA (rRNA) gene restriction pattern are studied by this method. It is both rapid and specific method for bacterial identification. It is used for identification of the species of the organisms and for differentiation between and among strains within a species.

So, if the above mentioned experiments could be carried out, results of the present study could be further extended and clarified. In order to make the result more acceptable, a recent work on multi-drug-resistant *Klebsiella pneumoniae* in different hospitals is mentioned here, which correlates with this study. [4] They studied 440 patients with 455 consecutive episodes of *Klebsiella pneumoniae* bacteremia between January, 1996 and December, 1997, of which 253 episodes were nosocomially acquired. They found that overall 30.8% episodes of nosocomial bacteremia, and 43.5% episodes acquired in ICU were due to ESBL-producing organism where antibiotic use is the heaviest and potential for patient-to-patient transmission of the organism is the greatest.

Klebsiella pneumoniae examined for presence of ESBLs by PFGE was used by a group of investigators to analyze the molecular epidemiology of nosocomial bacteremia with the organism.[5] They found more than one ESBL-producing isolate in 7 out of 10 hospitals with multiple strains with same genotypic pattern, which indicate patient-to-patient spread of organism. [6]

Now, from the above-mentioned recent studies and from the experiments done in this study, it can be concluded that two types of multi-drug-resistant ESBL-producing *Klebsiella pneumoniae,* carrying different types of plasmids, along with TEM-10 family-beta-lactamase showing resistance to third generation cephalosporins caused cluster of infections at Elsewhere Hospital Intensive Care Unit, where patient-to-patient transmission of organism occurs.

References

1. Madigan MT, Martinko JM, Parker J. Technique and Applications of Working with nucleic acids: In: Broock Biology of Microorganism. UK Blackwell Publications 2000: pp. 172-3.
2. Madigan MT, Martinko JM, Parker J. The Tools, Conventional and Molecular Taxonomy, Nucleic Acid Probes in Clinical Diagnosis. In: Broock Biology of Microorganism. UK: Blackwell Publications 2000: pp. 360-1.
3. Madigan MT, Martinko JM, Parker J. Amplification of DNA: The Polymerase Chain Reaction. In: Broock Biology of Microorganism. UK: Blackwell Publications 2000: p.882.
4. Koneman EW, Alien SD, Janda WM, Schreckenberger PC, Winn WC, Jr. Molecular Methods in Clinical Microbiology, technical Problem with PCR. In: Color Atlas and Text Book of Diagnostic Microbiology. UK: Blackwell Publicatioons 1997: pp. 50-1.
3. Paterson DL, Ko WC, Gottberg AV, Mohapatra S, Casellas JM, Goossens H, *et al.* Annals of Internal Medicine, 6th ed: pp. 26-32.
4. Nsamaha-Kfoury J, Araj GF. Clinical review: Recent Development in 3-Lactamases and Extended Spectrum beta-Lactamases. BMJ 2003; 327: 1209-13.
5. Sanders CC, Sanders WE Jr. Clinical importance of inducible beta - lactamases in gram- negative bacteria. Eur J Clin Microbiol 1987; 6(4): 435-8.

6. Institute of Cell and Molecular Science. The Bards and The London, Queen Mary School of Medicine and Dentistry: Centre for Infectious Diseases. Basic Biology of Bacteria, Practical and Lecture Classes: M.Sc and Diploma in Clinical Microbiology. UK: 2003-2004. [Handout]

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Outcome of Rubber Band Ligation in the Treatment of Haemorrhoids Using Suction Ligator

A.K.M DAUD[a] ANWAR JAHAN KHANB[b]

Abstract:

Background: Hemorrhoids are a common surgical problem seen in the outpatient department. Rubber band ligation using suction is an effective treatment for symptomatic hemorrhoids but associated with significant morbidity. This prospective observational study was earned out to assess the efficacy, effectiveness and complications of rubber band ligation performed in outpatient for symptomatic hemorrhoids using suction ligator.

Methods: 50 consecutive patients with symptomatic hemorrhoids underwent rubber band ligation in the outpatient clinic. Outcome measures were symptomatic cure i.e. stoppage of bleeding and reduced mucosal prolapse. Complications were categorized as immediate, early (within one week) and late (within 3 months). Results: Symptomatic cure was achieved in 45(89.20%) patients (stoppage of bleeding and reduced mucosal prolapse). 5 (10.72%) patients required further banding. Pain (mild to severe) was most common immediate and intermediate complication (14 and 20 patient's respectively). Conclusion: Rubber band ligation is an effective, outdoor procedure for symptomatic hemorrhoids, but associated with certain degree of morbidity.

[OMTAJ 2005;4 (2)]

Introduction

Hemorrhoids are among the commonest surgical ailments of anorectal region seen in the outpatient department1. These are usually associated with distressing symptoms. The incidence of hemorrhoids increases with age. At least 50% of patients over the age of 50 years have some degree of discomfort from them. Shyness and fear are the main reasons of avoiding medical treatment2. Treatment of hemorrhoids has remained controversial among surgeons all over the world. For the last many decades' surgeons have treatment, which would give good results with minimum complications3.

Most patients in initial stages are treated with conservative or minimally invasive approaches. Injection sclerotherapy and rubber band ligation(RBL) are two common non-surgical interventional procedures to treat first (Tf) and second (2nd) degree hemorrhoids. These procedures can be performed in the outpatient clinics, with minimum resources and are cost effective. Rubber band ligation has been shown to be superior to the injection sclerotherapy; however it is also associated with certain complications. Introduction of suction ligator has almost completely replaced the conventional Barren's apparatus. In this article I will present the outcome of rubber band ligation using suction ligator in patient who presented or were referred for the treatment of hemorrhoids

Material and Methods

This study was conducted at Private Chamber colorectal centre Karim Monjil manik pir road Noyasarak road, Sylhet From February 2005 to December 2005 Fifty consecutive patients with different degree of hemorrhoids were included as shown in Table 1.

After complete history and examination (both digital rectal examination and proctoscopy) diagnosis and degree of hemorrhoids was confirmed. Rigid sigmoidoscopy was performed in selected cases. The indications for sigmoidoscopy were other symptoms like change in bowel habit, diarrhea and to rule out any malignant pathology in the distal colon. All patients were explained about the procedure and its complications and other treatment options available. *An* informed consent was obtained.

a. Assistant professor, Department of Surgery MAG Osmani Medical College Sylhet
b. Anaesthesiologist, Jalalabad Ragib Rabeya Medical College, Sylhet.

Patient was placed in left lateral position. Routine rectal examination and proctoscopy was performed to ascertain the degree of hemorrhoids. The base of hemorrhoids was clearly identified and bands were applied above the dentate line using suction band ligator (Karlstorz Germany) (Figure-1). Two hemorrhoids were banded at single sitting. Repeat banding was done after 4 weeks when needed.

The patients were asked to report any pain on applying suction. If they had any pain, suction was released and a higher spot was chosen and bands were applied only when patient had no complaints of pain. Following the procedure, patients were asked to rate the discomfort / pain on a scale of 1-10. The pain was graded as mild (1-3), moderate (4-6) and severe (7-10). The patients were observed for immediate complications for 30 minutes and were followed up for intermediate complications (within 1 weeks) and late (within 2 months) intheOPD.

Data was analyzed by using SPSS version 10 and p-value was calculated using *chi* square test.

Results

In this study there were 50 patients out of which 36 (85.6%) were male and 14 (14.3%) were female. All the patients had the symptoms of bleeding per rectum while *29* (51.7%) patients had complaints of prolapse and 23 (41%) had complaints of itching / pruritus ani and 16 (28.2%) had complaints of discharge per rectal. Total 100 bands were applied (two per single sitting) and repeat banding was done in 6 (10.7%) cases.. The comparative outcome and complications are given in the tables 2 and 3.

Twenty-four (42.8%) patients had immediate complications (Table 2) while 27 (48.21%) patients experienced intermediate complications (Table 3). There were no late and serious complications like sepsis, massive bleeding necrosis, ulceration and stenosis. There was no mortality. After two months follow up 50 (89.3%) patients had symptomatic cure and six patients had complaints of bleeding. On proctoscopy there were complete recovery in 41 (73.21%) cases, 9 (16.07%) had residual bulge, 3 (5.3%) had complete one pile left and (5.3%) had two complete piles left. (Table 4).

Table-1: Degree of Hemorrhoids

Degree	Number of Cases	Percentage
1S1 (1°)	05	19.04 %
2nd (Ho)	41	73.22 %
•ird (11 lo)	04	07.14%

Table-2: Immediate Complications of Suction Band Ligatkm

Problem	Total
Mild Pain	3(5.3%)
Moderate Pain	3(5.3%) . .
Severe Pain	2(3.5%)
Vasovagal episode	1(1.7%)
Bleeding	2(3.5%)
Total	11(19.64%)

Table-3: Intermediate Complications of Suction Band Ligation

Complication	
Discomfort/mild Pain	6(10.71 %)
Moderate Pain	4(7.14%)
Severe Pain	2(3.5%)

Discussion

Various techniques have been instituted for the treatment of hemorrhoids. Ligation and excision is the conventional treatment for prolapsing hemorrhoids. This procedure can be an unpleasant experience for some patients. Complications such as per-operative and post-operative hemorrhage, urinary retention, post-operative pain and anal stenosis are well documented. To avoid these complications various alternatives such as selerotherapy, rubber band ligation (RBL) and variety of other techniques requiring costly equipment include cryosurgery, photocoagulation. laser surgery, radio frequency coagulation and direct current coagulation, infra red coagulations and bipolar diathermy have been devised. However, these procedures are also not devoid of complications [1>4-6'7].

Surgical procedures include manual dilation of anus, internal sphincterotomy and various modifications in techniques of hemorrhoidectomy.[8"14]

In the recent past there has been a strong trend in favour of day care surgery for the treatment of internal hemorrhoids. Lord's procedure of maximum anal dilatation, introduced in 1968, is the most controversial of the newer methods of treating hemorrhoids [15>16].

Sclerotherapy is simpler than other outpatient procedures, which require costly instruments. Patients treated by sclerotherapy and RBL experience dull aching anal pain lasting from 2 hours to a couple of days[6].

Fever with Rigors	**1(1.78%)**
Burning Micturation	0
Slippage of Band	0
Total	13(23.21 %)

Table-4: Result after two **months of banding**

Parameter	No of Cases
Symptoms	
Parameter	No or Cases
Symptoms	
No bleeding	50(89.28%)
Some bleeding	6 (10 112%)
Signs (Pro cW's copy)	
Complete Recovery	41(7321%)
Residual Bulge	09(115.07%)
Complete one oile left	03 (03 35 %)
Complete two pile left	03(0335%)
No bleeding	50(89.28 %)
Some bleeding	6(10.62%)
Signs (Proctoscopy)	
Complete Recovery	41 (73.21 %)
Residual Bulge	09 (16.07 %)
Complete one pile left	03 (03.35 %)
Complete two pile left	03 (03.35 %)

Fig-1: **Suction band ligator (Karl** Storz **Germany)**

Rubber Band Ligation was introduced by Blaisdell in 1958 and refined by Barren in 1963. The cost effectiveness, safety, ease of treatment for both patients and doctor combined with good clinical results has increased the popularity of RBL [17].

RBL using suction is a recent modification. It is performed by a simple disposable plastic apparatus which has both band applicator and suction device in one instrument, the haemmorhoidal mass is sucked and band is applied.

It is best suited to the most of second -degree hemorrhoids and patient friend]}' procedure even for pregnant, elderly and those unfit for surgery. RBL should be attempted only on second degree or early third degree hemorrhoids. Since with first degree, especially in early cases, there is insufficient tissue available to pull in to the ligature drum to make the method worthwhile, and in any event such small piles can be as effectively managed by injection sclerotherapy. Two or three hemorrhoids dealt with at the same session. Over 90% of symptomatic hemorrhoids can be treated conservatively or with RBL. The main criticism of RBL is that it does nothing to remove the skin-covered component of the pile or an associated skin covered component of the pile or an associated skin tag. The bothering skin tags may be removed under local anesthesia as an out door procedure later on[18].

Secondary hemorrhage which may be life threatening is another problem which may occur at home as the patient is treated on out patients' basis. For the third degree hemorrhoids with large skin covered component, RBL has very limited and temporary value and is no substitute for surgical treatment. As far as complications of this method are concerned, delayed massive rectal bleeding, urinary retention, pain and fever, perianal abscess& perianal fistula, band related muscosal ulcer and priapism lias been reported in the literature[19]. i20

Studies of Murie et al and Poen at al[21] have shown RBL as effective as haemorrhoidectomy.The study also confirms that RBL is an effective treatment for symptomatic hemorrhoids. Kurnar et al[22] described cure rate up to 71%, whereas in our study cure has been 89% and the incidence of immediate and intermediate complications was 67.3% and 74.4 respectively in their study whereas in our series it was 42.85% and 48.21 % respectively which as quite less as compared to Kumar et al series. All these patients required observation for 1 hour and later discharged, no patient was admitted to in-patient. This is comparable to other international studies [5'8]. There were no late complications. Though very rare, but serious complications, like bleeding, sepsis and mortality have been reported. Though the incidence of complications in our series is less but it is significant. In

a series of 100 patient's ninety one percent (91%) patients were symptoms free after six weeks of treatment[19].

In a study carried out by Murie in 1980 "in which he compared rubber band ligation and hemorrhoidectomy for second degree hemorrhoids concluded that rubber band ligation should be considered as the first line of treatment for second-degree hemorrhoids[20].

Some surgeons have performed rubber band ligation at only one site at each outpatient visit while others have applied two bands, and some have band ligation of all three hemorrhoids at single visit.

In a series of 200 patients out-come in 89% of patients with prolapse; banding was effective in relieving anal pain, pruritus ani and soiling, which are regarded as secondary symptoms of the disease[17].

Conclusion

There is remarkable individual variation in the management of hemorrhoids. Day care and less invasive procedures are more acceptable to the patients.

RBL is an efficient, safe and acceptable modality. Addition of suction gun to banding apparatus adds to the convenience of surgeon and the procedure becomes quick and effective. Selection criteria of patients of RBL have important impact in the outcome of management of hemorrhoids.

References

1. Bhatti A A, Ahmed R, Butt MA. Comparative study between sclerotherapy and manual anal dilatation in the management of second-degree hemorrhoids. Pakistan Postgrad Med J 1993; 4 (4): 267-76.
2. Sheikh AR, Ahmed 1.Comparative study of haeniorrhoidectomy with rubber band ligation for second and third degree hemorrhoids Specialist 1995; 12(1): 39-45.
3. Malik AM, Zafar MR, Abbasi SA. Submucosal closed and open (ligation Excision) hemorrhoidectomy. Pak Armed Forces Med Jour 1947; 02(6-7): 20-2.
4. Quraishy MS, Idris F, Sultan N. Out Patient treatment of hemorrhoids with electronic coagulator-initial experience with 103 patients. Med Spectrum 1999; 02(6-7): 20-2.
5. Aftab ML. A comparative analysis of infrared coagulation and injection sclerotherapy as non-operative treatment for the management of hemorrhoids. Biomedica 1997; 13: 70-2.
6. Aftab ML. Role of photocoagulation in the management of non-prolapsing and early prolapsing hemorrhoids. Annals 1999; 5(3): 310-1.
7. Muslim A. Non-surgical treatment of hemorrhoids. Specialist 1993; 9(3): 269-72.
8. Khan AZ, Naqi SA, Gondal KM, Butt HA, Ahmed M. Comparison of band ligation with hemorrhoidectomy in second and early third degree hemorrhoids. Annals 1998; 4(4): 59-61.
9. Malik SN. Management of prolapsed hemorrhoids. Ann KE Med Coll 1999; 5(2): 114-6.
10. Chaudhry MR, Ahmed MS, Abbas T. Management of fourth degree hemorrhoids by lateral internal sphinceterotomy, a safe alternative to emergency haemorrhoidectomy. Pakistan J Surg 1996; 12(4): 184-5.
11. Ervosji SO. Immediate hemorrhoidectomy for thrombosed fourth degree hemorrhoids. JPMA 1994; 44(11): 264-5.
12. Ho YH, Seow Choen F, Tan M, Leong AFPK. Randomized controlled trial of open and closed haemorrhoidectomy. Br J Surg. 1997; 84:1729-30.
13. Seow-Choen F. Stapled hemorrhoidectomy. Pain or gain. Br J Surg 2001; (88): 1-3.
14. Ganio E, Altmare F, Gabrielli F, Milito G, Canuti S. Br J Surg. 2001; (88) 669-74.
15. Hancock BD. How do surgeons treat hemorrhoids? Ann R Coll of Surg Engl 1982; 64:397-400.
16. Snook S, Henry MM. Swash M.Faecal incontinence after anal dilation Br.J Surg. 1984; 71:617-8.
17. Ahmed R, Khan MR Rashid A, Management of haemorrhoids by rubber band-Ugation. Specialist 1995; 12(1): 79-81.
18. Aftab ML. Rubber band ligation in the management of haemorrhoids. Pakistan J Surg 1995; 11(4)219-20.
19. Khan SA, Kazmi SAR, Qureshi MI Chaudhry MR, Shah S TA. Experience of rubber band ligation with suction gun in the management of haemorrhoids at Mayo Hospital, Lahore. Annals of KEMC 1998; 4(4): 70-1.
20. Murie JA Machkenzia J, Sim AJW. Comparison of rubber band ligation and hemorrhoidectomy for second and third degree hemorrhoids: a prospective clinical trial. Br J Surg 67:786-8.
21. Poen AC, Felt BRJ Cuesta M, Deville W, Meuwissen SG. A randomized controlled trial of RBL Vs infrared coagulation in the treatment of internal hemorrhoid Euro J of Gastroenterology 2000, 12: 535-9.
22. Kumar N, Paulvanan S, Billings PJ. Rubber band ligation of hemorrhoids in the out patient clinic. Ann R Coll Surg Engl2002; 84: 172-74

ORIGINAL ARTICLE

The Osmani Medical
Teachers Association Journal
Volume 4 Number 2

Study on Prevalence and Pattern of Visceral Injury Following Blunt Trauma in a Peripheral Teaching Hospital

*MUHAMMAD KHALED MAHMUD[a], MD. MUSTAQUE AHMED BARBHUIYAN[b], PROMODE RANJAN SINGH[c], MEER MAHBUBUL ALAM[d]

Abstract

This study includes 60 cases of blunt abdominal trauma (BAT) admitted in different Surgical and Paediatric Surgical wards of Sylhet MAG Osmani Medical College Hospital between the period of July, 1999 to June, 2001, showing prevalence of BAT 9.3 cases/1000 admissions. The majority of the patients (50%) were injured in RTA followed by blows, kicks, falls, machinery injuries. The mean age of the patients was 32.50 yr. with male preponderance (87%). Major presenting features were pain abdomen (51), vomiting (19), shock (7), abdominal distensions (33), tenderness (47), rigidity (42), hypotension (20) and absent bowel sound (25). The injury was limited only to abdomen in 40 cases (66.67%). The associated injuries were pelvic fracture (10), rib fracture (8), head injury (5) and long bone fracture (7), Multiple organ injury was found in 17 cases. The common viscera injured in BAT in this study were liver (14), spleen (9), small gut (17), kidney (5), stomach (4), large gut (4), mesentery (4), etc. Plain x-ray abdomen, USG, paracentesis can diagnose evidence of visceral injury of 43.6%, 50%, and 63.5%, respectively. The initial assessment and resuscitation are continued simultaneously. The treatment of fracture was deferred until abdominal operation in most cases. 44 patients were operated upon and others were managed conservatively. The common post-operative complications were: wound infections (17), respiratory tract infections (16), and urinary tract infections (70). Total 09 patients died in this series during resucitation period (04), 1 after delayed laparotomy, and another 04 after immediate laparotomy. The cases of deaths included haemodynamic instability, septicaemia and electrolyte imbalance. Though mortality rate in this study is low (15%), but the real picture would be expected to be different because a number of victims of BAT with solid organ injury or multiple organ injury died on the way to hospital. Most of the patients (50%), left hospital within 8-14 days and only 10 (6%) patients stayed for more than 21 days. This study showed commonest causes of blunt trauma abdomen causing solid visceral injury particularly liver is RTA, and fall from height cases hollow viscus injury, particularly small gut and stomach.

[OMTAJ 2005; 4(2)

Introduction

Injury is the leading cause of death and disability in the first four decades of life and is the 3rd most common cause of death overall[1]. The abdomen is the third most commonly injured region, with injuries requiring operation in about 20 percent of caviling trauma victims[2]. The common cause of blunt abdominal trauma include (a) motor vehicle accidents, (b) falls from height, (c) assaults, (d) industrial accidents,(e) climbing high trees, (f) trawler injury, etc. There is frequently a poor correlation between the physical examination and presence or absence of significant intra-abdominal injuries in patients who have suffered from blunt abdominal trauma[3]. Open wounds are sometimes easier than non-penetrating blunt trauma and this is particularly true regarding abdomen and so is the

a. Assistant Registrar, Surgery Unit.
Sylhet MAG Osmani Medical College Hospital
b. Assistant Professor of Surgery
c. Assistant Professor of Urology
d. Professor and Head of the Department of Surgery,
Sylhet M.A.G. Osmani Medical College
* Corresponding author

doctrine-"0bscure abdominal injury is better treated with exploration."

It is often difficult to evaluate patients with blunt trauma to the abdomen as so many have other multiple injuries to the head, other areas of the trunk and extremities. Many have confounding problems such as acute alcohol intoxication or symptoms from drug abuse. The other major problem in dealing with patients with blunt trauma is that the most commonly injured organs are solid organs that may bleed slowly thus peritoneal signs may be lacking in the first several hours after admission to the hospital.

Blunt abdominal trauma constitutes one percent of all trauma admission in the United States of America, but it is associated with significant mortality (20-30%), much of which are attributable to the associated injuring of head, chest and/or pelvis. A significant percent of victims die before any treatment could be instituted. A staggering 12,00,000 people die from trauma each year in the USA, and more than 18,000 in the UK.

The human body is Subjected to an ever-increasing number and variety of external forces including falls, blows, knives and bullets as well as those imposed by the modern automobile and other transport vehicles. These wounding agents seldom are limited to anatomical boundaries and so injuries to several body areas are common. Blunt Abdominal Trauma (BAT) presents a challenge to the clinician, because of diagnostic difficulties in some cases. Many cases of blunt abdominal injuries get admitted in all hospitals of our country. Some cases present only with blunt abdominal injury and others are associated with other injures like chest injury, head injury, limb injury, etc.

Though fatalities of only the blunt abdominal injuries are largely correctable, but their recognition greatly impair the smooth recovery including death[1]. But all cases of blunt abdominal trauma do not require operative treatment. Some cases well responds to conservative treatment. Many of BAT patient initially do not show any sign-symptom of visceral injury both clinically and after routine investigation. But latter they develop sign-symptom of peritonitis. If this patient is not observed continuously there is chance of missing of diagnosis resulting fatality. Pattern of visceral injury is related with the type or cause of blunt trauma. But there is no such study in our country to show the pattern of visceral injury in relation to the type of blunt trauma.

The purpose of the study is to find out the prevalence of blunt abdominal trauma, the correlation of cause of blunt trauma and types of visceral injury.

This study was designed to realize the importance of early diagnosis, continuous follow up, and nature of the injuries to reduce the morbidity and mortality of the blunt abdominal trauma.

Methods

This prospective study was carried out with all patients admitted in Surgical, and Paediatric Surgical units of Sylhet MAG Osmani Medical Collage Hospital (SOMCH) following blunt trauma of abdomen, during the period of July, 1999 to June, 2001. Patients of all ages having blunt abdominal trauma, were included in the study, and patients with the history of penetrating abdominal injury were excluded from the study.

The diagnosis of visceral injury in the patients of blunt abdominal trauma was done by clinical history, physical examination, relevant investigations, and recorded in the protocol devised for the study. (Table I). The mode of injury and its relation to different types of visceral injuries, types of treatment, mortality and morbidity patterns were also recorded. (Table II).

After initial resuscitation, the patients were divided into four groups for the purpose of management: i. Group-A: Clinically and investigation suggests visceral injury (Immediately laparotomy), ii. Group B: No suspicion of visceral injury clinically and by investigation within 24 hours, but sign-symptoms of peritonitis develops within 48-72 hours (delayed laparotomy), iii. Group C: No sign-symptom of visceral injury and/or peritonitis develops during hospital stay (conservation), and iv. Group D: Not recovered from initial shock and succumbed (failed to resuscitate).

Table I: Protocol 1 to diagnose visceral injury of the patients

I. Particulars of Patient:

1. Name..... Age..... Sex.....
2. Regn no..... Ward..... Bed .....
3. Date of Admission .....
   Date of Discharge/ Death.....
4. Lose of Consciousness ..... Present/Absent
5. Time of Trauma.....

6. Time lapse between trauma and presentation: 0-12 hr/12-24 hr /1-2day />2day
7. Mode of injury- RTA (Passenger/ pedestrian/Motor cycle accident)

II: diagnosis:
Assault (Brow/Kicks) Fall (fall from height /fall on object)
Trawler injury
Machinery injury
Other

IV: Presentation:
Pain in abdomen- Yes/No
Shock- Yes/No
Dyspnoea- Yes/No
Vomiting- Yes/No
Urinary Retention- Yes/No
Haematuria- Yes/No Other

V. Physical Signs:

a. General:

Pulse...../min	Pallor.....
BP..... .....mn Hg	Cyanosis.....
Temp.....°C	Dehydration.....
Heart.....	Lungs .....

Associated injury .....

b. Abdomen:
(i) Inspection:
Abdominal distension Yes/No
Alteration of abdominothoracic Rhythm-Yes/No
Fullness of flant Yes/No
Abrasion, Bruise laceration Yes/No
(i i) Palpation:
Muscle guard and rigidly: Yes/No
Localized deep tenderness Yes/No
Rebound tenderness Yes/No
(iii) Percussion:
Percussion note: Dull/ Resonant
Obliteration of liver dullness: Yes/No
(iv) Auscultation-Bowel sound: Present / Absent
(v) Digital Rectal Examination: Tenderness: Yes/No
Rectovesical /Rectouterine Pouch: Empty / cystic

VI: Investigations:
a. Routine: Blood:
TC..... ....
DC-N-
L-
M-
HB%
Random Blood Sugar
Blood Urea
Blood Grouping

Serum Electrolytes

b. Special:
i. Plain X-ray Abdomen (in erect posture):
Free gas shadow/ Ground glass appearance / Distended small bowel
ii. Chest X-ray PA view: Rib fracture/ Haemothorax
iii. X-ray Pelvis and limbs
iv. USG of Whole Abdomen:
Visceral injury (Liver/ Spleen/Kidney)
Free fluid /Pelvic Collection
v. Four-quadrant aspiration test-
Haemorrhagic/Bile-stained/ others
vi. Others:

Table II: Protocol 2 to record the mode of injuries and their relation with visceral injuries

1. Clinical Diagnosis:
Visceral injury - Certain /Suspected / not suspected

2. Final Diagnosis (after laparotomy):
a) Visceral injury - present/ Absent.
b) If Present: (i) Gut, (ii) Other organ (Stomach/ Duodenum/ Small gut/ Large gut)

Liver	Spleen	Pancreas
Kidney	Diaphragm	Mesentery
Urinary bladder		Others

3. Treatment:
Repair
Excision
Resection and anastomosis
Others

4. Postoperative period:
a) Uneventful
b) Complication-

Wound infection- Yes/No
Chest infection- Yes/No
Urinary infection- Yes/No
Prolonged Paralytic ileus- Yes/No
Wound dehiscence- Yes/No
Fistula- Yes/No
Septicaemia- Yes/No
Others-

5. Mortality- Yes/No

Diagnosis of visceral injury was confirmed in all cases by laparotomy (except group-C and D). Statistical analysis was done by mean ± SD and the data are presented in tabular from.

Results

Table I: Age distribution of the patients (n-60)

Age (in years)	Number of patients	Percentage
0-10	5	8.33 %
11-20	6	10%
21-30	22	36.67 %
31-40	10	16.67%
41-50	7	11.67%
51-60	4	6.67 %
61-70	5	8.33 %
>70	1	1.67%

Table II: Sex distribution of the patients (n-60)

Sex	Number of patients	Percentage
Male	52	86.67 %
Female	8	13.33%

The age of the patients in this study series ranged from 5 years to 71 years with the highest incidence (36.67 %) in persons between the ages of 21-30 years. The mean age was 32.50 years. Five Patients were at or below ten years of age and one patent was over seventy years of age. There were 52 male Patents (86.67%) and 8 female Patents (13.33%) in the series. So the Male: Female ratio is 6. 5:1.

Table III: Mode of injury and its distribution (n=60)

Injury type	Number of patients	Percentage
RTA (passenger, pedestrian, motor cycle accident)	30 (12+15+3)	50%
Assaults (blow, kicks)	13(7+6)	21.67%
Fall from height	8	13.33 %
Fall on object	2	3.33 %
Trawler/machinery injury	3	5%
Miscellaneous	4	6.67 %

In this study series of 60 cases with BAT, majority of the cases were due Road Traffic (RTA) which include motor vehicle accident (Passenger-12), Pedestrian struck -15 and motor cycle accident- 3 and resulted in a total of 30 casualty occupying 50 % of entire series. Assaults like blows or kicks over the abdomen resulted BAT in 13 cases, which is 21.67%. Injury resulting from falls include both fall upon blunt object-2 < and fall from height-8 comprised 16.67%.

Table IV: Presentation of the patients (n=60)

Presentation	Number of patients	Percentage
Pain abdomen	51	85%
Shock	7	11.67%
Dyspnoea	6	10%
Vomiting	19	31.67%
Urinary retention	5	8.33 %
Haematuria	4	6.67 %
Unconsciousness	3	5%

Table V: Clinical findings and its distribution in the patients (n=60)

Clinical findings	Number of patients	Percentage
General		
Pallor	24	40%
Hypotension	20	33.33 %
Tachypnoea	15	25%
Tachycardia	19	31.67%
Abdominal		
Abdominal distention	33	55%
Abrasion, Bruise, Lacaration	19	31.67%
Alteration of abdominothoracic rhyth	15	25%
Fullness of flanks	21	35%
Localized deep tenderness	47	78.33 %
Muscle guard and rigidity	42	70%
Obliteration of liver dullness	24	40%
Absent bowel sound	25	41.67%

Table VI: Clinical findings and its distribution in the patients (n=60)

Associated injury	Number of patients	Percentage
None	40	66.67 %
Pelvic fracture	10	16.67%
Rib fracture	8	13.33%
Head injury	5	8.33 %
Long bone fracture	7	11.67%
Soft tissue injury	5	8.33 %

The commonest mode of presentation were pain abdomen 51 (85 %), vomiting 19 (31.67 %) and retention of urine 5 (8.33 %) (Table-4). Major clinical sings included sings of peritonitis- abdominal tenderness 47(78.33 %), rigidity 42 (70 %), pallor 24(40 %) hypotension 20 33 (3 %)and absent bowel sound 25 (41.67 %)(Table-5). Among the 60 cases of BAT,20 was associated with extra-abdominal injuries, pelvic fracture (16.67 %) followed by rib fracture (13.33%)and long bone fracture (11.67 %) were the associated injuries.

Table VII: Management group

Group	Type of management	No. of patients
Group A	Immediate laparotomy	40 (66.67 %)
Group B	Delayed laparotomy	4(6.67 %
Group C	Conservative	12(20%)
Group D	Failed to resuscitate	4 (6.67 %)

Table VIII: Results of plain X- Ray abdomen (n=55)

Findings	Number of Patents			
	Group-A	Group-B	Group-C	Group-D
Free gas shadow	20	1	0	3
Ground glass appearance	30	2	1	3
Distended small bowel	4	1	1	3
Non- specific	4	0	0	0

Table IX: Results of USG of abdomen (n=16)

Findings	Number of Patents		
	Group-A	Group-B	Group-C
Liver injury	Not	2	2
Spleen injury	done	1	0
Kidney injury		0	1
Free fluid/pelvic collection		1	1
NAD		0	8

Table X: Results of Peritoneal tap or Paracentesis (n-30)

Findings	Number of Patents		
	Group-A	Group-B	Group-C
Haemoperitoneum	13	2	1
Bile- Stained	3	0	0
Negative aspiration	0	2	5
Others	4	0	0

Table XI: Change of decision from conservative to operative management (n=4)

Time Factor	No. of patents	Percentage
12-24 hr	-	-
24-48 hr	3	18.75%
>48 hr	1	6.25 %

This shows that RTA & Blow abdomen cause more injury to solid organs (35 %) and Fall from height & Machinery injury cause more injury to hollow viscera (30%). Besides this, RTA is associated with multiple injuries (50%).

TabIe XII: Mode of injury and types of visceral injury (n=44)

Mode of injury	No. of patient	Isolated visceral injury							Multiple injury
		Solid				Hollow			
		Liver	Spleen	Kidney	Pancreas	Mesentery	S. gut	Stomach	
RTA	30	8	2	1	1	0	2	1	15
Blow and Kicks	13	2	1	1	2	0	5	1	3
Fall	10	1	1	1	0	1	4	1	1
Machinery injury	7	0	0	0	0	1	2	0	4
Total	60	1	4	3	3	2	13	3	23

Table XIII: Operative finding of visceral injury in different management Group (n=44)

Group	Liver	Spleen	Kidney	Small gut	mesentery	Diaphragm	Large gut	pancreas	Stomach	UB
Group A (40)	10	8	4	16	4	1	4	3	4	2
Group B (4)	2	1	0	1	1	0	0		0	0

Its shows that in group A, liver is the most common solid organ to be injured in BAT followed by spleen. On the other hand small gut is the commonest hollow organ to be injured. Bat in group B, liver injury is more common.

Table XIV: Overall Pattern of visceral injury in BAT victims (n=60)

Sl no.	Organs involved	No. of patents group-A+B+C=Total	Percentage
	Liver	10+2+2+= 14	23.33%
	Spleen	8+1+0=9	15%
	Pancreas	3+0+0-3	5%
	Kidney	4+0+1=5	8.33 %
	Stomach	4+0+0-4	6.67 %
	Duodenum	2+0+0-2	3.33%
	Small gut	16+1+0=17	28.33 %
	Large gut	4+0+0-4	6.67 %
	Diphragm	1+0+0=1	1.67%
	Mesentery	4+0+0=4	6.67 %
	Urinary bladder	2+0+0=2	3.33 %
	Urethra	1+0+0-1	1.67%
11.	Visceral injury not confirmed	10+0+1+4-15	25%

In this series, the most common solid organ injury was liver in 14 cases (23.33%) followed by spleen in 9 cases (15 %) and kidney is 5 cases (8.33%). Among the hollow viscera, small gut was mostly involved in BAT in 17 cases (28.33 %) followed by Stomach and Colon each in 4 cases (6.67 %). Bat the urinary bladder was injured only in 2 cases (3.33 %).

Table XV: Types of Operative procedures for different types of visceral injuries

Sl no	Visceral Injury		
	Type (number)	Procedures adopted	Number
01	Liver (12)	Simple repairs Buttress suture	04
02	Spleen	Splenectomy	07
		Splenorrhaphy	02
03	Pancreas (03)	Peritoneal toileting and drain	03
04	Small gut (17)	Simple repair	11
		Resection & anastomosis	6
05	Stomach (04)	Repair with omental patch	03
		Simple repair	01
06	Duodenum (02)	Repair with bypass	02
07	Large gut (04)	Resection and anastomosis	01
		Repair & proximal colostomy	03
08	Diaphargm (01)	Repair and water seal chest drains	01
09	Mesentery (04)	Mesenteric repair	02
10	Kidney (4)	Nephrectomy	01
		Repair	03
11	Urinary bladder and urethra (2)	Repair and suprapubic cystostomy	02
12	No visceral injury seen (1)	Peritoneal toileting	01

Table XVI: Pattern of complication related to management group

Complication	Number of Patents into each group			Total (%)
	A	B	C	
Wound infection	13	2	0	1-5 (25%)
Chest infection	14	1	0	15 (25%)
Paralytic ileus	7	2	1	10 (16.67%)
Wound dehiscence	2	1	0	03 (5%)
Fistula	2	1	1	04 (6.67%)
Septicaemia	3	1	0	04 (6.67%)
Total	46	8	3	

The common complications were wound infections (17), respiratory infections (16), urinary tract infections (7) and paralytic ileus (10). (Table XVI)

Table XVII: Mortality chart in relation to group

Period	No. of patients died				Percent
	A	B	C	D	
Preoperative period	00	00	00	04	6.67 %
Peroperative period	00	00	00	00	00
Post-operative period	04	01	00	00	8.33 %
Total	04	01	00	04	15.0%

A total 09 patients died of whcih o4 (6.67%) died during resuscitation period, which was categorized as group D. Some 05 patients died due to failed resuscitation in the post-operative period, one of them grouped into B died due to electrolyte imbalance, following ileal fistula on 25^{n} day. The other 04cases of deaths were among group A. (Table XVII)

Table XVIII: Cases and mode of deaths of group A

Number of deaths	POD	Injuries	Mode of Deaths
01	2nd	Spleen + Stomach + Diaphragm	Hypovolaemic shock
01	3rd	Colon + Spleen	Shock + Electrolyte imbalance
01	4th	Colon + Spleen	Septicaemia + ? ARDS
01	5th	Small gut (multiple)	Electrolyte imbalance

Table XIX: Duration of hospital stay.

Duration (in days)	No. of Patients				Total (%)
	A	B	C	D	
00-07	00	00	03	04	7 (11.67%)
08- 14	23	02	05	00	30 (50.0 %)
15- 21	08	01	04	00	13 (7.8%)
>21	09	01	00	00	10 (06.0%)

In this study, most of the patients (30, 50.0 %) left hospital within 8-14 days, followed by 13 (7.8 %) patients leaving within 15-21 days. Only 10 (6.0%) patients developed burst abdomen, requiring secondary suture, and was discharged on the 50th day. (Table XIX)

Discussion

Abdominal trauma is a very common day to day incidence in our society occurring both accidentally (RTA) and incidentally (assault). Today trauma is the leading cause of death and disability in the first 4th decades of life and is the third most common cause of death overall[1]. In our country, inspite of the recorded high incidence of deaths due to this cause, there is no definite statistics.

In broad term abdominal trauma can be classified into penetrating and non-penetrating trauma. A more appropriate term for non-penetrating trauma would be BLUNT or CLOSED trauma because some surgeons limit the use of the term non-penetrating injuring to penetrating wounds restricted to the abdominal wall in which the peritoneum has not been involved.

In this study period (July 1999 to June 2001), 14, 200 patients were admitted in all surgical units of SOMCH of which 6450 had traumatic injury. Among the 60 patients having Blunt Abdominal Trauma giving the prevalence of 9.3/1000 admission of traumatic patient were included in the study.

Injury is produced by forces that deform tissues beyond their failure limit. Blunt trauma distributed energy over a larger area. Such an injury may be associated with rapid acceleration (pedestrian struck) or rapid deceleration (high speed motor vehicle accident and fall). Due to differential inertia such an injury produces extreme degree of strain at point of anatomical fixation. Severe direct deformation of tissues also occur at point of direct contact.

The ages of the patients in this study ranged from 5 years to 71 years with the highest incidence (36.67 %) in persons between 21-30 yrs followed by the age group 31-40 yrs (16.67 %). the mean age was 32.50 year. Five patients were at or below ten years of age one patient was over seventy. The above figure indicates that affected people are those who are most mobile and active in their daily life. They are the earning members of the family and as such subjected more to the various traumatic agents, these results were almost similar to that of S. Gupta et al. in a study of 63 cases of BAT. But in a study of 100 cases of Bat the highest age group was 31-40 years which was conducted by Quammzzaman .

Only 8 (13.33%) cases were female in this study, reflecting the less out going people of the community in the context of RTA, but not applicable to falls and assault as the causative agents in producing BAT. The sex ration in this study compares favourably with that of Quamruzzaman study
TQ
which documented only 10% female in this study .

Mode of injury

Majority of the documented cases of BAT were due to RTA (50%) which included 12 passenger, 15 pedestrian, struck down by vehicles and 3 were victims of motor cycle accidents. It seems that pedestrians are more affected in RTA than passengers. Injuries in 13 cases were produced by direct force or blunt weapons including kicks or blows with fists, clubs and other

instrument (like hockey sticks) Injuries resulting from falls included 8 persons with the history of the remaining 17 cases, trawler and machinery injury was the cases in 3 cases and miscellaneous cases (struck by domestic cows) in rest 4 cases.

In a study by Robbs *et al* of South Africa, the causes of BAT was as follows: RTA 23.2%, industrial accidents 8.9%, and physical assaults 67.9%.[30] In another study of 200cases of BAT by Fitzerald *et a*l of Texas showed the cases of BAT as follows: RTA 83.5 %, assault 6.5 %, and fall 5%.[4] In comparing the findings of this study to that of Robb's study, the main difference lies in the large percentage of cases of BAT due to physical assaults. Probable explanation can be the effective utilization of health care facilities available in the country by the health conscious, people for even the minor injury. In the latter study by Fitzerald *et al* RTA was the major cases and in this era of high speed transportation it is the usual finding in almost all countries of the world. It not only matches with our study but also with the study Gupta *et al.* There are many cases of higher incidence of RTA in our country, such as, poor traffic control by the inexperienced drivers. Defective motor vehicles, defying the traffic rules and regulations both by pedestrians and drivers, not implementing the laws against the defaulters, bad and narrow highways admixtures of high speed (car, bus, truck, etc) and slow speed vehicles (Rickshaw, baby taxi)in the same congested roads, etc. But in the Western countries, alcoholism and super high speed vehicles are the common causes.

The commonest mode of presentation were pain abdomen (51), vomiting (19), shock (7) and retention of urine (5). Major clinical sings include abdominal distention (33), tenderness (47), alteration of abdomino-thoracic respiratory rhythm (15), rigidity (42) hypotension (20) and absent bowel sound (25). Obliteration of liver dullness was found in 24 cases and all of them had gut injury as substantiated on laparotomy. So, it is a good parameter for diagnosis of hollow viscus injury. Abdominal injury was evident by local signs (abrasion, bruise, etc) on abdominal wall also. The high incidence of vomiting might be related to coeliac plexus irritation in upper abdominal trauma.

n a study series by Mackersie *et al* it was shown that hypotension on hospital arrival, presence of major chest injury and arterial base deficit were <3 mEq/L on arrival are the most powerful indicators of intra abdominal injury .But in our setup, we mainly depend on history of trauma, clinical presentation and findings, and simple investigation like X-rays, USG, etc. The clinical features of the cases in this study are almost similar to that of F. Ahmed[29]and Quamruzzaman[28].

In the unconscious patients (3), intraabdominal injury was suspected from signs of continued haemorrahge not evident externally, signs of injury on the abdominal wall, ribs fracture and peritoneal tap.

This study of small series of 60 cases of Blunt Abdominal trauma was an enthusiastic approach towards realization of the importance of early detection of abdominal organ injury and immediate management. In this dissertation, an attempt has been made to evaluate the clinical pattern of visceral injury following blunt abdominal trauma .BAT is no longer an academic curiosity.

In this study patients often present deceptively with normal appearance leading the physicians to a false sense of security. So it is vital to harbour a high index of suspicion for BAT patients with multiple injuries rather than investigation. Continued follow-up is mandatory to reduce mortality and morbidity of patients. The pattern of visceral injury, in general, is related to the mode of injury. Direct blunt trauma is related to more visceral injury than indirect trauma, specially for solid organ injury.

This study shows the commonest cause of blunt abdominal trauma is RTA &blow abdomen causing injury to solid organ particularly liver. But fall from height & machinery injury cases hollow viscus injury. Presence of blood and bile following abdominal paracentesis is valuable diagnostic investigation for visceral injury. Continued follow up is needed to diagnose visceral injury for late presentation. Complication is more common in those who have shown definite sign-symptom of visceral injury following blunt trauma. A high index of suspicion, immediate and repeated clinical examination for any case of BAT leads to early diagnosis of visceral injury. Prompt management and early laparotomy should be the rule rather than exception.

In conclusion, it can be highlighted that we cannot prevent all deaths of the patients of blunts abdominal trauma, but at least we can try with our limited resources and facilities to reduce the morbidity and mortality of patients bases on our knowledge, keen observations honest intentions and since efforts.

References

1. Russell RCG Williams NS, Blustrode CJK. editors. Bailey and love's shorts practice of surgery,23rd Ed. London: Aenlod;2000:170-7.
2. Cox EF. Blunt abdominal trauma. Annals of surgery 1984, Aprile:467.
3. Wu,CL Chou MC. Surgical management of blunt abdominal trauma. Department of surgery.Kuang Tien General Hospital, 1993, Sept 9(9):5-52.
4. Fizerald JB, Crowford EC. Surgical Consideration of Non-penetrating abdominal injuries. Am. JSurg 1960 July; 100.
5. Sabiston david C. Jr. MD. Gert H Brieger, MD. The Development of surgery: Text Book of Surgery; sabiston : 4111 edition.
6. Krik RM Manafield AO, Cochrance JPS. editors.Clinical Surgery in General, 3rd Ed. 1999:32.
7. Oslen WR, Rednan HC, Quantitative prritioneal lavage in blunt abdominal trauma. Arch surg 1972;104:536
8. Rossi D, Goyet J,Were F,clerment S. Moulin D. Management of intraabdominal organ injury following blunt abdominal trauma in children. Intensive Care Med. 1993; 19 (7): 415-9.
9. Goletti O et al. The role of ultrasonography in blunt abdominal trauma. Department of Emergency Surgery , Uurgery, University of Pisa. Italy (J Trauma, 1994 Feb;36(2): 178-8.
10. Sation M, Matsuda H,et al. Early diagnosis of small intestine rupture from blunt abdominal trauma using computed tomography.J Trauma 1995 Apr.
11. Shires GT,Thal FR,et al abdominal Trauma: principles of Surgery, Vol-16th edition, by Seymour I Schwarts MD,Prof.&Chair, Deptt.of Surgery, University of Rochester USA.
12. Dudley HAF. editors. Hamilton bailey's Emergency surgery.l 1th ed. Bristol, 1985:477-82.
13. Verdechia GM, et al. Indicationa and results of conservative treatment of closed trauma of the liver. Division Di Chirurgia General e Toracica, Ospedale G.B.Morgagni,Forli (G-Chir,1993,Dec;14(9):472-4.
14. Meredith JW, Young JS,Bowling J. Nonoperative Management of blunt hepatic trauma:the exception or the role? J Trauma 1994 Apr;36(4):529-35.
15. Shires T, Poules E, Mechlelland R, Hepatic resection for massive trauma. J Trauma 1964;4:282.
16. David V Feliciano MD. Abdominal Trauma In: Maingot's Abdominal Operrations:9th ed.Vol 1.
17. Northrup WF, Simmons RL.Pancreatic trauma -A review. Surgery 1979;27.
18. Paul C. peters MD, Arthur I Sagalowsky MD: Genitourinary trauma :Camball's Urology: 6th ed;3:257-8.
19. Chirstopher Fowler. Bailey and love's short practice of surgery, 23rd Ed. London: Arnold: 2000:1179.
20. Michael S. Hickey MD. Surgical Matabolism; Current Surgical Diagnosis and Treatment. 10th ed. USA: prentice Hall int.inc; 1994:143-7.
21. Craig MH, Talton DS Major gastroenteric injuries from blunt trauma . Am. J Surg. 1995 Jan.
22. Ledgerwood AM. Lucas CE. Factors influencing outcome after blunt duodenal injury. J Trauma 1975;15:839.
23. Cerise EJ, Sculy JH. Blunt trauma to small intestine. J Trauma 1970; 1:46.
24. Lindenmann JM. Schurid D. Jejunum perforation following blunt abdominal trauma .Chirurgisehe abteihng Scheiz, Pflegerinnenschub Zurich (Schweiz- Rundesh-Med- Prax; Aug.1994,83 (31): 87560).
25. Flint LM, et al. The injured colon. Annalsof Surgery 1981; 193:619
26. Rintoul RF. editor. Farquharson's Text Book of Operative surgery, 8th ed. Churchill livingstone; 1995:621-2.
27. Gupta S. Talwar S. Sharma Rk. Indian Journal of Medical science!996 Aug;50(8):272-6.
28. Quamruzzaman M Blunt Abdminal Trauma a Study of loo cases (Dissertation) Dhaka IPGMR 1996
29. F. Ahmed ; Blunt Abdominal Trauma- clinical evaluation of the cases admitted in SBMC Barisal during the period 1994-95

Case Report

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Maternal Anaphylactic Reaction During Termination of Pregnancy by Induction of Labor

*MRS. FORHAT MAHAL[a], MD. HELAL UDDIN[b]

The Case Report

A 32 years, fourth gravid lady, unsure of her date (estimated at 38 weeks by ultrasonography), suffering from severe pregnancy induced hypertension (PIH) was admitted in a maternity clinic for termination of pregnancy.

She had three previous normal deliveries at home. Age of last child was six years. During this pregnancy, she has had irregular antenatal checkup during 1st and 2nd trimesters. During last trimester, she developed leg oedema, which was increasing day by day and sought specialist's supervision as she had also raised blood pressure. Her urine was free from albumin and reducing substances. Her kidney- and liver-function tests as well as coagulation profiles were normal.

At first, oral anti-hypertensive was given but she was a defaulter and did not take the medicine regularly. She also came for antenatal checkup irregularly. When lastly she attended the specialist's chamber, her blood pressure (BP) was found 180/110 mm. of Hg with marked leg oedema (+++), and no others symptoms. She was advised to get admission because of uncontrolled hypertension. After admission, bed-side urine test was found albumin- and sugar-free. Her BP, and foetal heart rate (FHR) were monitored carefully.

a. Associate Professor
Department of Obstetrics and Gynaecology, North East Medical College, Sylhet.

b. Assistant Professor, Department of Anesthesiology, Sylhet MAG Osmani Medical College, Sylhet.

* Corresponding author

Tab. Dopezyt (Methyldopa) 250 mg, 2 tablets 8-hourly had been given. Next day, the dose of Methyldopa was increased to 3 tablets 8 hourly, and 1 tablet thrice daily of Nefidipine 10 mg was also added. Her BP came down ranging from 135/85 to 145/100 mm of Hg. The FHS was also monitored and was found within normal range.

In view of her previous good obstetrical records, satisfactory present foetal condition, and controlled BP with Methyldopa and Nefidipine, it was decided to terminate the pregnancy by induction of labour. The situation was explained to the patient and her husband. They were also willing to have a vaginal delivery.

Induction of labour was done by application of a commercial preparation of prostaglandin PGE_2 (Serviprime gel) to the intra-cervix by an applicator as instructed. It was applied in the morning at 10.30 am. The patient was closely monitored of her BP, FHS and progress of labour. At 1.30 pm, the patient experienced mild abdominal pain with backache. The patient was examined vaginally at 6.40 pm as the pain was increasing and cervix was dilated up to 5-6 cm with the membrane bulging. Artificial membrane rupture was done and liquor was found mildly meconium-stained. The BP of the patient was raised to 180/110 mm Hg, FHR was around 145/minute.

As the labour progressed, an infusion of 5% Dextrose in Aqua 1000 cc was started and a prophylactic antibiotic (Inj. Cefradin, 500 mg I/V) given side by side. To control her raised BP, 2 drops of sublingual Nefidipine (Neficap) was also given.

All on a sudden, just after 3 to 5 minutes of starting of all of these measures, the patient went into shock. Her face had undergone oedematous, lips and eyelids were swollen and the sclera was congested. She had severe respiratory distress, BP shut down to 80/40 and the pulse was very feeble and rate was slow. Per vaginal

examination was carried out and cervix was found almost fully dilated but the station was high up. The foetal bradycardia was started. The infusion was stopped and changed. The woman was transferred to the operation theatre and an Anesthesiologist and a Neonatologist were called as she might need intubations and operative delivery.

In the operation theatre, the patient was put under oxygen mask with 4 litre/minute flow to ensure 100% oxygen. The patient was conscious and auscultation of the chest revealed inspiratory and expiratory rhonchi. There had been development of angioedema in the face that was congested along with swelling of the lips and eyelids and congestion of the sclera. These features gave the impression of development of anaphylactic reaction either with the fluid (dextrose in aqua), or the antibiotics, Cephradine or Nefidipine.

The FHR was markedly bradycardic and dropped below 100 bpm persistently. Inj. Hydrocortisone 300 mg was given intravenously; Inj. Phenergan (Chlorpheniramine) was already given before in the cabin. An indwelling Foley's catheter was inserted and urine output was measured. At first, the urine was high coloured and scanty in amount. With the increase of infusion (with Hartsmann's solution) the BP was started to rise. Urine became clear and output was good. During this time of 20-30 minutes of resuscitation, the cervix was found fully dilated and foetal head was found low down. Forceps delivery was performed and a severely asphyxiated male baby of 3 kg weight was delivered. APGAR score was 3 in 1st minute, and 6 in 10th minute, and the baby was transferred to the Neonatologist's care.

After delivery of the baby, the placenta was examined and found intact. There was no retro-placental clot and lower segment was found intact. The BP started to improve gradually and rose again, which was controlled by Tab. Meltyldopa, 1 tablet thrice daily.

The patient was discharged on 3rd post natal day. But the baby died due to severe birth asphyxia even after prompt resuscitation as the interval between the developments of severe bradycardia and delivery, was about 20-30 minutes.

Discussion

Anaphylaxis is an allergic condition due to antigen and antibody reaction in which cardiac output and BP often decrease drastically. The full-blown syndrome includes urticaria and angioedema with profound hypotension and brochospasm.[1]

The anaphylactic shock is becoming more common and is frightening to treat because of its severity. The classic form was described in 1902 as involvement of prior sensitization to an allergen with later re-exposure, producing symptoms via an immunologic mechanism.[2] After exposure to inciting substances, there occurs rapid onset of increased secretion from mucous membranes, bronchial spasm, and vasodilatation with increased capillary permeability. These effects are produced by the release of mediators which include histamine, leukotriene, complements, prostagladin D2 and tryptase. In classic form, mediator release occurs when the antigen, usually protein, binds to antigen-specific immunoglobulin E (IgE) attached to previously sensitized basophils and mast cells.[3] The mediators are released almost immediately when the antigen binds. An anaphylactoid reaction, exposure to an inciting substance cause direct release of mediators, a process that is not mediated by IgE. Clinically it is not possible to distinguish between the two forms, and fortunately, treatment for both the mechanisms are same.

There are many records of severe allergic reaction to the following classes of substances like food, drugs and insect bites in liberating allergic mediators. Drugs which are notorious to produce serious reactions are antibiotics like penicillin, cephalosporin, immunizing agents, horse sera, dextran, haemacil, morphine, pethidine, thiopental, latex, and the list is expanding. Atopic patients are more liable to allergic reaction those who have history of bronchial asthma, eczema, hay fever and food allergy.

The only available diagnostic test of anaphylaxis is serum tryptase, which is an indicator of activation of mast cells.[4]

Administration of injection adrenaline is the most important treatment of anaphylaxis, though we had given the women Inj. Ephedrine intravenously. Oxygen should be administered at once to ensure 100% saturation even by positive pressure ventilation. Intramuscular or slow intravenous Chlorpheniramine 10-20 mg, and Hydrocortisone 200-400 mg should be given.

Successful and sophisticated management of a patient in shock requires an integrated team approach. In

our case, the diagnosis of anaphylactic shock was done clinically by appearance of angioedema in face, eyelids and sclera congestion, and appearance of bronchospasm and hypotension. After delivery of the baby, uterus was examined and no retroperitoneal clot was found. The lower segment of uterus was found intact. These findings excluded the possibilities of abruption placenta, and rupture uterus, respectively.[5-8]

Fluid 5% D/A sometimes cause pyrogenic reaction due to presence of pyrogen in the fluid, but the anaphylactic like reaction is difficult to establish, as not been yet reported. The patient got Nefidipine tablet from last two days of this reaction. The antibiotic which was given along with fluid was Cephradine, but the patient received injectable Cephradine after the catastrophe without any reaction. So, we suspected the intravenous fluid that might contain some impurities that acted as antigen and caused such adverse effects.

Acknowledgement

The author would like to thank Dr. Monajjir Ali (Neonatologist), Sylhet MAG Osmani Medical College; Dr. Abdur Rashid (Anaesthesiologist), Sylhet MAG Osmani Medical College Hospital, Dr. Ibrahim, Dr. Saiful Islam Khan and the nursing stuff for their support.

Reference

1. Guyton AC, Hall JE. Circulatory shock and physiology of its treatment. In: Text Book of Medical Physiology, 10th edn. India: Harcourt Asia Pte Ltd 2000: pp. 259.
2. Shqartz LB. Systemic anaphylaxis. In: Diseases of systemic anaphylaxis, allergy and clinical immunology, 22nd ed. USA: Elsevier 1999: pp. 201-7.
3. Mitchell RN, Cotran RS. Hemodynamic Disorders, Thrombosis, and Shock. In: Cotran RS, Kumar V, Collins T. Robbin's Pathologic Basis of Disease, 6th edn. India: Harcourt Asia Pte Ltd 1999: pp. 134-7.
4. Schwarz LB. Tryptase, a mediator of human mast cells. J Allergy Cell Immunol 1990; 86: 594-8.
5. Myerscough PR. Murokerrs operative obstetrics, 10th edn. London: Bailliere Tindall 1996: pp. 473-5.
6. Stannard L, Bellis A. Maternal anaphylactic reaction to a general anaesthesia during emergency Caesarean section for fetal bradycardia. Br J Obstet Gynaecol 2001; 108: 539-40.
7. The Association of Anaesthetist of Great Britain and Ireland and British Society of Allergy and Clinical Immunology. Suspected anaphylactic reaction to Anaesthesia. London 2003.
8. Andrew PC, Tooke M, Bethune CA, Fay AC, Spickell GP. Adrenalin in the treatment of anaphylaxis, what is the evidence? A clinical review. BMJ 2003; 327: 1332-5.

Case Report

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Leech in the peritoneal Cavity- An unusual cause of intestinal obstruction-A case report.

MD. SHAMSUR RAHMAN

Abstract:

A 3 years old girl admitted in the Paediatric Surgery department of Sylhet Medical College Hospital with the complaints of persistent pain in whole abdomen associated with fever, vomiting and constipation for 3 days. Patient's mother gave a history of bleeding per vagina and suspected leech bite 5 days back, but she was confused whether leech entered inside the vagina or not. Clinically it was a case of Acute Intestinal Obstruction. Plain radiography and Ultrasonography of whole abdomen revealed intestinal obstruction. Emergency laparotomy was done and a dead leech measuring 3 inches in length in stretched condition was found in the left iliac fossa which was attached from the mid ileum to the descending colon causing band effect. No perforation or gangrene found in the intestine or any collection in the peritoneal cavity. No other port of entry could be detected on external or internal surface. Post operative recovery was uneventful. The patient was discharged from the hospital on the 10th post operative day. Route of entry of a leech in the peritoneal cavity through genital tract is a rarity and presentation as acute intestinal obstruction yet not reported.

Asstt. Professor (CC)
Dept. of Paediatric surgery
SOMC

Introduction

The common causes of acute intestinal obstruction in children are:

-Congenital (Atresia, HPD, ARM etc.)

- Helminthiasis
- Postoperative bands and adhesions
- Burst appendix
- Intussusception
- Tuberculosis
- Trauma
- Tumour

The Specific symptoms always may not be expressed by children properly. Leech bite is common in the rural areas of Bangladesh in rainy season (Ashar & Srabon). Leech bite is commonly found in the external body surfaces. Leech bite in nose, pharynx, larynx, oesophagus, genitalia, urethra and rectum are also reported[1]. Vaginal bleeding in children as the result of a leech bite is very rare and there are only three case reports available in the Medline indexed literature[2]. The entry of a leech into the peritoneal cavity through the genital tract is a rare incident and acute intestinal obstruction caused by leech is still not reported.

Case Report

A 3 years-old girl hailing from Nobigonj, Hobigonj, was admitted in our word with a history of persistent abdominal pain, vomiting & constipation for 3 days. She first went to the nearest primary health care centre from where she was referred to the Paediatric Surgery department of SOMCH after 72 hours.

The patient's mother gave the history of vaginal bleeding following a bath in a pond and a suspected leech bite 5 days back as it was common in their area. There was no history of trauma, nor was there any evidence of abuse.

Figure –I . The patient on operation table

At the time of admission in our ward, there was no active bleeding from vagina or any foreign body within it. Her physical examination was carried out & our clinical diagnosis was Acute Intestinal Obstruction. Routine blood examination (Hb%- 10.5 gm/dl), serum electrolytes, urine R/M/E & CXR were normal. Diagnosis of Acute Intestinal Obstruction was made by plain radiography of abdomen in erect posture and ultrasonography of whole abdomen.

Figure – 2: Plain X-ray Showing features of inestinal obstruction

Laparotomy was done under general endotracheal anaesthesia through a supraumbilical transverse incision on the 6th day of incident.

There was no haemorrhagic fluid or exudate found in the peritoneal cavity but the small gut was distended and a band like structure was found between a loop of ileum & the descending colon.

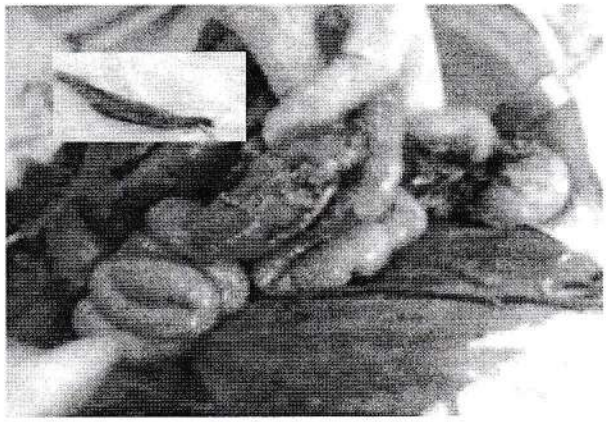


Figure -3: Per operative appearance of the gut where leech was attached

Later the band was identified as a dead leech about 3 inches in length, which was attached by its two ends. The sites of the gut where leech was attached were red in appearance and oedematous but there was no ulceration or perforation.

No other port of entry of the leech was found on external or internal surfaces of the abdomen. Abdomen was closed in layers without keeping any drain.

Naked eye examination confirmed the band like structure as a leech which was about 3 inches in length and was preserved in a formalin containing bottle.

Figure -4: Dead leech 3 inches in length

The postoperative recovery was uneventful and the patient was discharged on 10th POD. There was no complaint when she came back for follow up after 2 weeks of discharge.

Discussion

Leeches are invertebrates of phylum Annelida and class Hirudinea. A leech is usually about 12.5-15.25 cm long. Leeches are classified according to the different ways them feed[3] such as:

a) Jawed leeches or Gnatbobdellida
b) Jawless leeches or Rhyncobdellida
c) Worm leeches or Pharyngobdellida

Leeches can be classified according to their habitat

a) Freshwater leeches
b) Terrestrial leeches
c) Marine leeches

The bodies of all leeches are divided into the same number of segments (34), with a powerful clinging sucker at each end- front sucker & rear sucker[4]. Leeches usually have three jaws and make a Y-shaped incision. The Australian land leeches have only two jaws and makes a V-shaped incision.

The prolonged bleeding after a leech bite is due to the action of factors in the leech saliva left in the bite[5], which include

Histamine-like vasodilator.
Hirudin (a potent antithrombin)
Hyaluronidase
Calin (a platelet aggregation inhibitor)

Bleeding from a leech bite wound can persist for a mean of 10 hours and as long as 7 days.

All leech species are carnivorous. Some are predatory, feeding on a variety of invertebrates such as worms, snails, insect larvae, crustaceans, while a very few are haemophagic parasitic blood-sucking leeches, feeding on blood of vertebrates such as amphibians, water fowl, fish & mammals.

Given the opportunity they will also feed on human blood. The most important predators on leeches are fish, aquatic insects, crayfish and other leeches specialized for predation on leeches.

Haemophagic leeches attach to their hosts and remain there until they become full at which point they fall off to digest.

When a jawed leech bites it holds the sucker in place by making its body rigid. Using its semicircular and many toothed jaws like minute saws, it then makes an incision in the skin and excretes a mucous. This helps the sucker to adhere and anaesthetize the host. A salivary secretion containing the anti coagulant and histamine floods the wound and the leech relaxes its body to allow the blood to be ingested. This mixture allows the blood to flow and prevents clotting once inside the leech.

Conclusion

Entrance of a leech into the peritoneal cavity through a female genital tract of a young girl is possible specially when bathing in a pond or a river. A leech in the peritoneal cavity can be attached to the adjacent loops of intestine and may cause Acute Intestinal Obstruction.

Leech infestation is common in rural areas of Bangladesh specially in the rainy season and the children getting substandard sanitary facilities are at high risk for leech bites in the genital regions; a high index of suspicion is of great help to make an early diagnosis and ensure prompt treatment.

References

1. Richardson, L.R. Observations on the Australian land leech, Chtonobdella Libbata. Aust. Zoology J 1968;XIV (3): 342-45.
2. Sawyer RT. Leech biology. Medscape general medicine. 2005;7(4): 152-53.
3. Mann KH. Leeches (Hirudinea) Their structure, Physiology, Ecology and Embryology. Pregmon press Ltd 1962; 6(2): 46-47.
4. Sawe RT. Leech Biology & Behavior (reprint). In: Rao P, Bailey BN, eds. Neurobiology of the Leech. 6th ed. USA: Churchill Livingstone Inc 1981: p 1015-17.
5. Munro R. Hechtl FOP, Sawyer RT. Sustained bleeding after a leech bite in the apparent absence of hirudin. Thrombosis and haemostasis. The practitioner 1989; 61(3): 366 -369.

REVIEW ARTICLE

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Conservative Management of Tennis Elbow- An Overview

MD. HILALUL ISLAM[a],

Summary

By tennis elbow is meant a painful lesion of the extensor muscles controlling wrist, situated near elbow. The classic tennis elbow, which bears the name after a description by Morris (1882), who referred to some patients with the 'Lawn tennis arm' is lateral epicondylitis or backhand tennis elbow. It is one of the commonest lesions of the arm and was first described by Runge in 1773. Roughly 40% of tennis enthusiasts will suffer from it at least once in their life but tennis players are not only sufferers. It is common in golfers, carpenters, bricklayers, violinists and housewives. The precise aetiology is unknown but the syndrome in often ascribed to a combination of degenerative changes and chronic overuse. Cyriax (1936) listed no less than 26 aetiologies from the literature but subscribed to what is probably the most widely held theory that there is an inflammatory reaction in the common extensor. More than 40 treatments have been suggested indicating that the ideal remedy has not yet been found. Finally, the course, management and aftercare of tennis elbow have been reviewed.

Introduction

The tennis elbow or lateral epicondylitis designates a pattern of pain at the origin of the extensor tendons of the wrist and fingers on the lateral epicondyle of the humerus. The name of this condition is a misnomer because it does occur more commonly in the non-athletes than in tennis players.[1] It is one of the commonest lesion of the arm[2] and is common in the dominant arm.[3] It is an overuse syndrome[4] and is more common in females with a male-female ratio of 1:2.[5] Its incidence is 1-3% in general population and about three times that in manual workers.6 The patient who present with this common and refractory problem is usually middle aged (between 40 and 60 years), and about only one in 20 plays tennis.[7] In general practice, not more than 5 of all cases are related to the game. The syndrome is rarely seen in individuals under the age of 30 and coloured peoples are apparently affected less frequently than whites.[3] Tennis players are not only sufferers. It is common in golfers, carpenters, bricklayers, violinists and housewives especially those between 35 and 55 years of age.[8]

Aetiology

The precise aetiology is unknown but the syndrome is often ascribed to a combination of degenerative changes and chronic overuse ischaemia could be a contributing factor. Muscular work steals blood from the relatively avascular (and thus vulnerable) enthesis. Vibration may play an additional role possibly by their more mechanical irritation or also be impeding blood perfusion.

Pathophysiology

Virtually every anatomical structure of the lateral aspect of the elbow has been suggested to be a morphological substrate of the disease.[3] Nirschle and Pettrone (1979), and Garden (1961) located the lesion more specifically in the extensor carpi radialis brevis.[5] The lesion is characterised by macroscopic and microscopic tears, which may be superficial or deep and situated at the tendonous origin of extensor carpi radialis brevis into the periosteum of the lateral humeral epicondyle. Microavulsion fractures may be seen as well as round cell infiltration, scattered foci of fine calcification, and scar tissue with marginal areas of cystic degeneration and fibrinoid degeneration may be evident in some cases; repair is by immature reparative tissue. These findings indicate rupture of the tendon. Typically,

a. Associate Professor, Department of Physical Medicine, Sylhet MAG Osmani Medical College, Sylhet

* Corresponding author

repeatitive and cumulative injury produces the pathological changes; forced overload may be intrinsic by muscle contraction or extrinsic by traumatic overstretching, occasionally the case is direct trauma.[2]

Dynamics of Injury

Tennis elbow has 2 distinct modes of onset. The first type of onset is gradual, reacting a peak of pain by 24 to 72 hours after activity. The patient may give a history of recent unaccustomed repetitive rotatory movements of the hand and wrist such as laying bricks using a screwdriver or indulging a long periods of craft hobbles. The second type of onset is acute. The player or patient can usually pinpoint the exact time and circumstance at which the pain occured.[6]

Symptoms

The outer bony projection of the elbow (the lateral epicondyl) is painful. For some people the pain is constant and can interfere with sleep. The forearm aches with grasping and lifting movements like pouring tea, turning stiff door-handles, ironing clothes and typing. Even simple things like picking up a glass, shaking hands or brushing teeth can cause pain.[4]

Signs

On examination, the elbow looks normal and flexion and extension is painless. There are three important positive physical signs:

i) Localized tenderness to palpation over the anterior aspect of lateral epicondyle.
ii) Pain on passive stretching at the wrist with elbow held in extension and the forearm prone (Mills maneuver).
iii) Pain on resisted extension of the wrist also with the elbow held in extension and the forearm prone (Cosen's test).[1] Elbow X-rays usually are normal for the patient's age but small areas of calcification may at times be seen in the extensor origin.[7]

Course

Fortunately, most cases undergo natural remission although tennis elbow tends to run a prolonged course with exacerbations and remission, depending usually on the activity for about six months but often as long as two years.[1] There is a spontaneous resolution of symptoms over long time- as many as 40% have persistent (although minor) symptoms five years later. Tennis elbow does have a high recurrence rate (18%-50% recur within one year) particularly in those people who persist in any precipitating activity.[18]

Management

More than 40 treatment modalities have been suggested indicating that the ideal remedy has not yet been found. General practitioners, rheumatologists, orthopedic surgeons and psyiatrists (Physical Medicine specialist) may opt for different, sometimes opposing strategies. Without doubt, however, initial treatment must be conservative. Furthermore, it seems wise to try physical methods first as they rarely harm and often help. They are immobilization plus oral NSAID, ultrasound, phonophoresis, laser, electromagnetism, massage, electrotherapy, manipulation, topical treatment etc.[4]

Immobilization of the forearm and wrist in a cock-up plaster has previously been advocated but is not recommended now. The patient needs to rest from those activities that are associated with this condition.[7] Oral NSAID are useful as first-line anti-inflammatory agents in patients with lateral epieondylifts.[8] Oral naproxen (250 mg twice daily for two weeks) is as effective as a single injection of a corticosleroid into the site of tenderness in the treatment of lateral epicondylitis.[9] Tenoxicam would seem to be beneficial in soft-tissue injuries, tendinitis, fibrositis, and epicondylitis.[10]

Therapeutic ultrasound enhances recovery in most patients with lateral epicondylitis.[11] It is felt to influence the inflammatory process through its effect on intracellalar calcium transport. Ultrasound (US) applied during the proliferative phase of tissue healing can increase the rate of tissue healing. This is presumably accomplished through non-thermal mechanisms. Acoustic streaming reportedly results in increased cellular activity by increasing the rate of ion flux across cellular membranes. This in turn has been postulated to increase cellular metabolism and reduce interstitial oedema. It may also increase the elasticity of tendons. The depth of penetration may be up to 4 cm. Wattage required depends on the size of the transducer head but ranges from 0.5 to 2.0 W/cm. While there are few controlled studies comparing different US regimens for epicondylitis 2 to 4 minutes of US two to three times a week is effective before therapeutic exercise.[8] Steriod

phonophoresis may be helpful in patients with epicondylitis and provides an alternative to injection in some patients. The mechanism of action of phonophoresis is that ultrasound drives intact large macromolecular steroid into the soft tissues. The depth of penetration can be considerable, perhaps up to 10 cm. US causes heat-induced vasodilation. For epicondylitis, a 10% hydrocortisone cream and 2% lidocaine jelly over 3 weeks, two to three times per week for 2-3 minutes has been recommended.[8]

About topical treatment, naproxen gel is effective for lateral epicondylitis and other soft tissue injuries which could prove useful in those patients where the side effects of oral NSAID are to be avoided.[12]

Transcutaneous electrical nerve stimulation (TENS) is used to reduce pain. The electrodes are usually placed over the radial turnel and at the dorsal thenar-index web space. Rebox is another apparatus for electrical therapy that operate on much lower currents than SNS (0 to 300 MicroAmpere).[16] Ice massage can be used to decrease pain and oedema. Point friction massage is performed at the lateral epicondyle to decrease pain and reduce venous congestion of the extensor carpi radialis muscle. Massage is performed for 5 to 10 minutes.[14]

In terms of preventing long term consequences of tennis elbow, it is important for the patient to try to increase the power, flexibility and endurance of the forearm. This is best accomplished through physical therapy combined with a variety of activities designed to improve stroke mechanics.[13,15]

Roughly 10-20 % of all patients do not respond to any of the above conservative therapies. In such cases, steroid injections should be tried. They are successful in about 9% but have relapse rates of around 30%. If injections fail to cure the pain after the second dose, further attempts are useless or even harmful. Patients who are unresponsive to steroid should be considered for operation.[3]

Surgery, in those patients who are resistant to conservative therapy or have recurrent problems is only rarely indicated. Several operations have been described. The most common is a tenotomy and stripping of the common extensor origion and is usually combined with debridgement of an chronic granulation tissue that is present. Active rehabilitation and an early return to sport postoperatively are essential.[17]

Aftercare

1. The extensor muscles in the forearm should be passively stretched and exercises given to strengthen then before allowing the patients to return to full activity.
2. Any provoking cause, such as poor sporting technique should be corrected if possible.
3. A support of the forearm is useful to alter the leverage on forearm muscles and relieve strain on the elbow.[18]

Initially manual resistance should be tried with the patient suing the contralateral hand to oppose the movements. The first exercise is flexion of the wrist against the other hand. The resistance is minimal initially and then gradually increased over the following few days as long as the symptoms do not increase in severity.[19-21]

References

1. Murtagh JE. Tennis elbow. Aust Pad Physcician 1988; 17 (2): 90.1-94.5.
2. Wadsworth TG. Tennis elbow: conservative, surgical and manipulative treatment. Br Med J Clin Res Ed 1987; 294 (6572): 621-4.
3. Ernst E. Conservative therapy for Tennis elbow. Br J Clin Pract 1992; 46 (1) : 55- 7.
4. Murtagh J. Tennis elbow. Aust Pam Physician 1984; 14 (1): 51.
5. Allum RLPT, Macphereon IS, Bentley B. Simple lateral release in treatment of tennis elbow. J R Soc Med 1985; 78 (11): 912-5.
6. Kamiea M. A rational managemeat of tennis elbow. Sports Med 1990; 9 (3): 173 -91.
7. Corrigan B. Mailland GD. Practical orthopedic medicine. UK: Butterworth and Co 1983: pp. 71-4.
8. Galleway M, Demaio M, Mangine R. Rehabilitable techniques in the treatment of medial and lateral epicondylitis. Orthopedics 1992; 15 (9): 1089-96.
9. Saartoh T, Eriksson E. Randomized trial of oral naproxen or local injection of betamethasone in lateralepicondylitis of the humerus. Orthopedics 1986; 9(2): 191-4.
10. Schorn D. Tenoxicam in soft-tissue rheumatism. S Afr Med J 1986; 169 (5): 301-3.
11. Binder A, Hedge G, Greenwood AM, Hazleman BL, Thomas DP. Is therapeutic ultrasound effective in treating soft tissue lesions? Br Med J Clin Res Ed 1985; 16: 290 (6467): 512-4.
12. Thorling J, Linden B, Berg R, Sandahl A. A double-blind comparison of raproxen gel and placebo in the treatment of soft tissue injuries. Curr Med Res Opin 1990; 12 (4):

OCCASIONAL NOTE

The Osmani Medical Teachers Association Journal Volume 4 Number 2

Prevention of Noise Induced Hearing Loss

MOHAMMAD ABDULLAH[a], HUSNE QUMER OSMANY[b], MASHUQUR RAHMAN CHOWDHURY[c]

Introduction:

Normal hearing is essential for the development of speech and personality in children. It is also important for an individual to perform his/her activities at an optimum level. Any alteration in this norm leads to disability i.e. varying degree of hearing impairment. Hearing loss is an invisible ill recognized physical disability leading to health and social problem. They live in a silent very uncertain world and unfortunately fail to draw attention or sympathy from their surrounding[1].

Noise is defined as any unwanted sound and it inflicts trauma to the hearing apparatus. Sudden exposure to high intensity noise may cause a temporary hearing loss i.e. temporary threshold shift (TTS), where as repeated exposure to noise trauma may change the temporary hearing loss to a permanent hearing loss i.e. permanent threshold shift (PTS) and is called **Noise Induced Hearing Loss (NIHL)**[2]. Besides this noise has been found to be a cause of high blood pressure, insomnia, GI problems, restlessness, mental irritability, harmful effects on fetus,faulty physical and mental development of child & even can cause damage to eyesight [3,4,5].

In Bangladesh about 13 million people are suffering from variable degree of hearing loss of which 3 million are having severe to profound hearing loss. According to WHO noise induced hearing loss is the third common cause of hearing loss in Bangladesh[1].

a Principal, SSMC and Professor, Department of Otolaryngology and Head-Neck Surgery, SSMC, Dhaka.

b Assistant Registrar, Department of Otolaryngology and Head-Neck Surgery, SSMC & Mitford Hospital, Dhaka.

c Associate Professor,ENTD, Sylhet MAG Osmani Medical College

NIHL is a universal phenomenon currently receiving long overdue attention at the global level. It is considered one of the main cause of hearing loss in both the developed and developing countries of the world. Initially recognized as a " boiler maker's deafness" at the end of the 19th century, as it was mainly afflicted the industrial workers. The condition has now grown to cover a much wider canvass in today's world[6,7] .

Definition:

According to report of WHO-PDH Informal Consultation, Geneva, on 28-30 October' 97 definition of Noise Induced Hearing Loss (NIHL) is 100 dB or 83 dB for 50 years exposure. Audiometric criteria for NIHL is bilateral sensori-neural hearing loss in o.5kHz threshold less then 50 dB hearing loss, and at least a 15 dB difference between high and low frequencies averages in under 50 year-olds. NIHL is typically greatest in the 4000 to 6000 –Hz region of an audiogram [8,9].

When sound becomes hazardous?

During conversation sound production level is 60 dB. It is apparent that sound level of 78 to 86 dB cause health hazards. Feeling of discomfort at 120 dB and sensation of pain arises at 140dB sound level [10,11] .

Parameters of hazardous noise depends on-

1. Frequency: Perceived as pitch.
2. Intensity: Perceived as loudness.
3. Nature of noise: steady state vs. impulse/ impact.
4. Duration of noise: length of time a person is exposed.
5. Individual susceptibility: age, sex, and physical condition.

The **frequency** of exposure determines the apex-to-base location of the damage in the organ of Corti.

The **intensity** in decibels (dB) sound pressure level (SPL) of the noise determines the rapidity with which the ear is damaged and the extent of the initial

anatomical lesion. It also determines whether the associated hearing loss will be temporary i.e. temporary threshold shift (TTS) or permanent i.e. permanent threshold shift (PTS) [12].

The **nature of noise** i.e., short duration or repeated moderate exposure cause different type of hearing loss. Intense short-duration exposures such as explosions result in an immediate noticeable hearing loss, this injury is termed ***acoustic trauma***. Shortly after the exposure, the individual has compound threshold shift (CTS), which suggests that the hearing loss has both temporary and permanent components. Thresholds partially recover over 1-2 weeks post- exposure. This recovery represents the disappearance of the TTS. The individual so exposed is often left with a 60 dB PTS at one or more high frequencies.

Exposure to moderate-intensity noise for several minutes or hours initially results in a TTS only. If the thresholds are measured after the individual has been away from the noise for 18-24 hours, his/her thresholds will have return to pre-exposure level. However repeated exposure to moderate-intensity noise gradually results in a permanent deterioration of auditory thresholds. This type of injury is termed **N*oise Induced Hearing Loss***. This has been reported by the classical study by Taylor et al (1965) of progressive hearing loss in a jute weaving industry. On the contrary interrupted exposure to noise (e.g. 6 hours a day for 36 days) initially produces the same magnitude of TTS as a continuous exposure. However as the paradigm continues the thresholds begin to improve and may eventually return within 10-15 dB of the pre-exposure baseline[12].

The **duration of the exposure** has a reciprocal relationship to intensity. The higher the intensity, the shorter the exposure and still cause permanent damage. Conversely, lower intensity noise may be safe even when the ear is exposed for long durations[12].

Source of hazardous noise:

a) Main causes are occupational noise and environmental noise from road traffic and the horn of vehicles.
b) Others are-
   I. Concert of rock and band music.
   II. Electronic musical devices e.g. headphone of Walkman.
   III. Noise from constructing works.
   IV. Bomb blast.
   V. Aviation noise at nears the airport.
   VI. Indiscriminate use of loud-speaker.

Individual susceptibility: Onset of noise-induced hearing loss may be at any age, and inter-subject variability is high even for the same exposure[9].

Pathogenesis of NIHL[12]:

Several hypotheses exist. The current hypothesis are-
Reduced blood flow during the exposure (Hawkings. 1971) leading to hypoxia and release of reactive oxygen species in the cochlea.
Metabolic exhaustion of the stimulated sensory cells.
Excessive release of neurotransmitter during the exposure leading excitotoxic damage of the afferent nerve fibers and terminals.
Intermixing of the cochlear fluids through the damage reticular lamina.

Damaging effects of noise on inner-ear structure:

Shortly after a damaging exposure, the cells and tissue of the inner ear are in a dynamic state of injury. degeneration and/or repair. This has been termed as ***acute phase of niose damage.*** With intense exposure (i.e.>/= 140 dB), a portion of the organ of the Corti is displaced from its position on the basilar membrane and is often found floating within scala media[13]. Swollen hair cells are found at the edges of the lesion and signs of damage are apparent in the non-myelinated nerve fibers of the organ of Corti[14].With moderate-level exposures for long durations such as those found in noisy industries (i.e.</= 90dB), a few scattered hair cells probably degenerate within the organ of Corti during each day of work. In general, the noise-induced loos of hair cells is very gradual. The longer the exposure of noise, the greater the number of missing sensory cells[15,16]. Permanent noise damage initially consists of degeneration of hair cells. Although both types of hair cells may degenerate, outer hair cells (OHCs) are more sensitive to noise than inner hair cells (IHCs). With longer exposure or a more intense noise, there is further loss of OHCs. IHCs, and supporting cells. If the cell loss is confined to a narrow region of the organ of Corti, a `focal' hair cell lesion develops. It is also known as **`cookie-bite' defects**[17]. Gradually there is a loss of myelinated nerve fibers (which are the peripheral process of the spiral ganglion cells) within the osseous spiral lamina[18]. Hair cell and supporting cell losses can gradually progress to involve 100% of the cells over a variable length of the organ of Corti. Eventually, the spiral ganglion cells, which originally innervated the degenerated portion of the organ of Corti, are

progressively lost, including their central process, which form the auditory portion of the eighth nerve. Once degeneration of the spiral ganglion cells has begun, there is a corresponding degeneration within the central nervous system including the cochlear nuclei, superior olive and inferior colliculus[19,20].

With interrupted exposures, the pattern of sensory-cell loss is the same as that with continuous noise while the magnitude of damage is generally less[18,21]. The amount of protection afforded by intermittency depends on the amount of rest between successive exposures. Too short a rest (quite) period results in no protection. With longer quite periods, some injured sensory cells may recover and the amount of damage will be proportionally less. The recovery period of a few days to one month allows irreversibly damaged cells to complete the process of degeneration. Remaining supporting cells participate in the formation of scars on the endolymphatic surface of the organ of Corti (i.e. reticular lamina) and on the basilar membrane. This period is termed the ***chronic phase of noise damage .***

Hearing status of NIHL:

Permanent effects of noise-induced hearing loss is difficulty in understanding conversation, particularly in background noise and high frequency hearing loss affects ability to hear consonants. Quality of environmental sound is diminished. Tinitus develops in 21% of noise-exposed workers. Initially, exposure to noise causes loss of sensitivity to high frequency(high pitch)sound, continued exposure results in damage to mid frequency region as well. One can experience progressive high frequency hearing loss and not be aware of it until it becomes severe (doesn't affect loudness perception).

Prevention of NIHL:

"Hearing health" is part of a healthy lifestyle. Noise-induced hearing loss is permanent and only partially treatable, yet virtually one hundred percent preventable. There are several ways of prevention of noise-induced hearing loss. e.g.

1. Development of public awareness
2. Avoidance of noise
3. Ear protection from noise and counseling
4. Job rearrangement/rehabilitation
5. Improved legislation and its implementation

Public awareness developed by taking steps to involve radio. Television, newspaper to raise public awareness about NIHL. Government publication Bureau should be encouraged to publish messages prepared by National Committee on Ear and Hearing Care. Community involvement by involving community leaders (political/trade union, religious), teachers of different educational institutes, Social workers and club members (Lions/Rotary Club etc.)

Usually the best way to prevent future injury from noise is to **avoid exposure** to noise. It is also generally prudent to avoid things that might contribute to ear damage-avoid ototoxic drugs like aspirin, and avoid situations, which damage the ear like scuba diving[10].

Hearing protection devices decreases the intensity of sound that reaches the eardrum. Protection from noise is required when steady noise measured at 85 dB or greater and impulse noise measured at 140 dB or greater. They come in three forms: earplugs (pre-formed and hand-formed), earmuffs and helmet. Properly fitted earplugs or muffs reduce noise 15 to 30 dB. The better earplugs and muffs are approximately equal in sound reduction, although ear plugs are better for low frequency noise and earmuffs for high frequency noise. Simultaneous use of earplugs and muffs usually adds 10 to 15 dB more protection than either used alone. Combined use should be considered when noise exceeds 105 dB. Susceptible persons should be made aware of the availability of hearing protection devices and ways to use these correctly[10].

Persons placed in occupations, which is of high risk for the development of NIHL should undergo **job rearrangement or rehabilitation**. The following procedures should be adopted for early detection and prevention or NIHL.

1. Baseline screening of all the persons by pure tone audiometry at the entry of the job should be mandatory
2. Annual screening of hearing by pure tone audiometry and those who are found to be vulnerable to NIHL should not be placed in high noise producing working environment.
3. Limit exposure time to noise by implementing shift work schedules. The exposure time should be reduced to half for each 5dB increase in overall noise exposure above 85dB/8working hours.
4. Susceptible person should change the job.

Improved legislation and implementation [6]:

The concern authority for the reduction of NIHL in Bangladesh should immediately take the following measures.

1. Identfication of noise producing machineries and taking measures to control those equipments and machineries are very important steps to be taken immediately.
2. Increase of forestation and ban on indiscriminate destruction of forest.
3. Measurement of noise level at the places where it is occurring mostly and taking necessary measures to keep the noise at the acceptable limits.
4. Education regarding noise pollution at the working place
5. Control on loud sound producing vehicles
6. Ban on hydraulic horn of vehicles
7. There should be regulations for controlling concert of rock & band music

Moreover person-having NIHL from occupational noise should get compensation from the concern authority. Punitive measures should be taken against the responsible farm if it fails to ensure preventive measures to its employees.

Present government has taken a revolutionary step in withdrawing noise-producing two-stroke vehicles from the roads of Dhaka and also proposing modification and improvement of existing legislations regarding various environmental pollutions. The new regulations lie with the Ministry of Law for final approval.

Conclusion:

NIHL is a universal phenomenon currently receiving long overdue attention at the global level. In Bangladesh it is now high time to take measures against noise induced hearing loss, which is 100% preventable.

Reference:

1. M Abdullah. Primary Ear and Hearing Care: The Foreseeable Solution for Prevention and Control of Deafness in Bangladesh-(Editorial)- Journal of College of Physicians and Surgeons of Bangladesh- Page 1-3, Vol-21, No. 1, Jan' 03
2. Peter M, Rabinowitz. Noise-Induced Hearing Loss. American Academy of Family Physicians. 2000
3. Mahmood R, Khan GJ, Alam S, Safi AJ, Salahuddin, Amin-ul-Huq. Effect of 90 decibel noise of 4000 Hertz on blood pressure in young adults. J Ayub Med Coll Abottabad 2004;16.30.3
4. Muzi G, Locati EH, Murgia N, Ambrosio G, Abbritti G. Evaluation of occupational noise on the cardiovascular system. Med Lav 2004;95:145-57. [Abstract from Medline, article in Italian]
5. Turkkahraman S, Gok U, Kelel E, Oturk A. Findings of standard and high frequency audiometry in workers exposed to occupational noise for long durations. Kulak Burun Bogaz Ithis Derg 2003;10:137-42. [Article in Turkish, abstract in Medline]
6. WWW. association-of noise-consultants. co.uk
7. Peter A. Traumatic Sensorineural Hearing Loss. In: Mowson's Diseases of the ear. 6th edn. ed. Ludman H, Edward Arnold, UK< 1998:483-493.
8. McBride DI< Williams S. Audiometric notch as a sign of noise-induced hearing loss. Occup Environ Med 2001;58:46-51.
9. Ginger MD, Kathleen CMC. Audiology, Pure tone testing 2004.
10. Roger FG, Maurice H. Synopsis of Otolaryngology. 5th edn, Butterworth-Heinemann Ltd, England. 1992, 39-72.
11. Noise Induced Hearing Loss, American Hearing Research Foundation (AHRF) [WWW. american- hearing.org]
12. Barbar AB. Gary WH. Noise-induced Hearing Loss.http//oto.wust1.edu/bbears/noise.htm
13. Lurie MH. The degeneration and absorption of the organ of Corti. Ann Otol Rhinhol Laryngol 1942;51:712-717.
14. Bohne BA. Mechanism of noise damage in the inner ear. IN: The effect of noise on hearing. Ed. Handerson D, Hamernik RP, Dosanjh DS, Mills JH. Raven Press, NY. 1976:41-68
15. Bredberg G. Cellular pattern and nerve supply of the human organ of Corti. Acta Otolaryngol 1968, S236:1-135.
16. Bohne BA, Clarke WW, Studies of noise induced hearing loss using an animal model. Hear insturmts 1990:13-16.
17. Johnsson LG, Hawkins JE Jr, Degenerative pattern in human ears exposed to noide. Ann Otol rhinol Laryngol 1976:85:725-739.
18. Bohne BA, Yohman L, Gruner MM. Cochlear damage following interrupted exposure to high- frequency noise. Hear Res 1987; 29:251-264.
19. Kim J, Morest DK, Bohne BA. Degeneration of axons in the brain stem of chinchilla after auditory over-stimulation. Hear Res 1997; 103:169-191.
20. Morest DK, Kim J, Potashner SJ, Bone BA. Long term degeneration in cochlear nerve and cochlear nucleus of the adult chinchilla following acoustic over-stimulation. Micro Res Tech 1998; 41:205 216.
21. Workers Compensation Board of British Columbia, Hearing Conservation section. Industrial audiometry training and reference manual. 8th Edn, Vancouver, BC 1992.

Bird Flu-An Emerging Threat to Mankind

Bird flu is a contagious disease of animals caused by type A influenza viruses that commonly infects birds and less commonly pigs and other animals. The disease in birds has two forms, the second form of which is known as highly pathogenic avian influenza. The disease is first recognized in Italy in 1878, and was extremely contagious in birds, rapidly fatal and mortality was 100%. Influenza A viruses can be divided into subtypes on the basis of their surface proteins- haemagglutinin (HA) and neuraminidase (NA). A total of 15 HA and 9 NA subtypes have been known. All subtypes were found in birds. Only 3 subtypes of HA (H1, H2, H3) and 2 subtypes of NA (N1, N2) are circulated in humans.[1] The virus is killed by heat (at 60 degrees Celsius for 30 minutes) and by common disinfectants, such as formalin and iodine compounds. The virus can survive, at cool temperature in contaminated manure for three months. The virus can survive for 4 days at 22 degrees Celsius and more than 30 days at 0 degree Celsius. One gram of contaminated manure can contain enough viruses to infect 1 million birds.

How it spreads?

Certain water birds act as hosts carrying the virus in their intestine. Infected birds shed virus in saliva, nasal secretions and faeces, contaminate dust and soil. Susceptible birds are infected when they have contact with contaminated material.

Routes of transmission:

Transmitted through respiratory and faecal-to-ora routes.[1] Mode of spread is from one country to another, through international trade in live poultry. Migratory birds have in the past implicated in the international spread of highly pathogenic avian influenza. Migratory waterfowl (wild ducks) serve as natural reservoir but these birds are also most resistant to infection. They carry the virus over greater distances and excrete it in their droppings, yet develop only mild and short-lived illness.

What is the present situation?

Outbreaks of highly pathogenic avian influenza have been reported in chickens and ducks in a number of Asian countries from mid- December 2003. Infection in several species of wild birds and in pigs have been reported. Rapid spread in several countries at the same time is historically unprecedented and of great concern for human health and agriculture.

Confirmed human cases of Avian Influenza A (H5N1)[2] reported to WHO on 10th August 05 are as follows. (Table I)

Table I: Confirmed Human Cases of Avian Influenza

Country	Total number (%) of cases	
	Total	Death
Indonesia	05	03
Thailand	17	12
Vietnam	91	41
Cambodia	04	04 (100)
Total:	117	60

Why so much concern about the current outbreak? Firstly most of the major outbreaks reported in Asia was caused by highly pathogenic "H5N1" strain. This strain has a unique capacity to jump over the species barrier and causes severe disease and high mortality in humans. Greater concern is the possibility that the present situation could give rise to another influenza pandemic and WHO warned that this could kill millions of people globally.

Because the avian and human influenza viruses can exchange genes when infected with viruses of both species. The process of gene swapping can give rise to completely new subtype of virus to which few, if any, human have natural immunity. If the new virus contains sufficient human gene, transmission directly from one person to another can occur and in successive generation, the disease will be more fatal with high mortality.

Background pandemic:

An influenza pandemic is a global outbreak when a new influenza virus emerges, spreads and causes disease worldwide[3] pandemic in 20th century. During 1918-19, "Spanish flu" [A (HlNl)] caused the highest number of flu death. The completely new virus emerged and spread globally in around 4-6 months. Severe wave of infection occurred over 2 years killing an estimated 40-50 million peoples. During 1957-58, "Asian flu" [A(H2N2)] caused about 70000 deaths in the United States. During 1968-69, "Hong Kong flu"[A (H3N2)], caused approximately 34000 deaths in the US. Once a new pandemic influenza emerges and spreads, it typically becomes established among people and circulates for many years. Beginning in late July, 2005, official reports to the OIE from

government authorities indicate that the H5N1 virus has expanded its geographical range. Both Russia and Kazakhstan reported outbreaks of avian influenza in poultry in late July, and confirmed H5N1 as the causative agent in early August.[3]

Deaths in migratory birds, infected with the virus, have also been reported. Outbreaks in both countries have been attributed to contact between domestic birds and wild waterfowl via shared water sources. Tests conducted by the World Organization for Animal Health (OIE) have today confirmed the presence of highly pathogenic H5N1 avian influenza in samples taken from domestic birds in Turkey.[4] The outbreaks in Russia, Kazakhstan and Turkey provide evidences that H5N1 viruses have spread beyond their initial focus in the south-east Asian countries, where outbreaks are now known to have begun in mid-2003.

Despite aggressive control efforts, FAO has warned that the H5N1 virus continues to be detected in many parts of Viet Nam and Indonesia and in some parts of Cambodia, China, Thailand, and possibly in Laos. The south-east Asian outbreaks, which have resulted in the death or destruction of more than 150 million birds, have had severe consequences for agriculture, and most especially for many rural farmers who depend on small backyard flocks for their income and food. Human cases, most of which have been linked to direct contact with diseased or dead poultry in rural areas, have been confirmed in four countries, namely Viet Nam, Thailand, Cambodia, and Indonesia. Only a few instances of limited human-to-human transmission have been recorded. Virologists have failed to find any human genes in the virus samples taken from the patients. However, urgent control of all outbreaks even by low pathogenic strain, is important because certain viral strains, initially of low pathogenicity, can rapidly mutate (6-9 months) into highly virulent strain if allowed to circulate in poultry.

Can a Pandemic be averted?

WHO is optimistic if right action is taken quickly, an influenza pandemic can be averted. Major line of defense is to reduce human exposure to virus infected poultry. This can be achieved by rapid detection of outbreak, introduction of control measures, destruction of infected poultry and proper disposal of carcasses. As the number of human infection increases, the risk that a new virus subtype emerges, triggering an influenza pandemic, will also increase.

Is there a vaccine effective against H5N1 in humans?

Currently available vaccines will not protect against disease caused by H5N1 strain in human. Vaccine composition changes each year to match changes in circulating virus due to antigenic drift.

Are presently available vaccines useful in averting an influenza pandemic?

Yes, but in a precisely targeted way. Current vaccines, when administered to high risk groups protect against circulating human strain and thus reduce the risk of those people who might be infected with human and avian virus at the same time. Such dual infection gives an opportunity to exchange genes, possibly resulting in a new influenza virus subtype with pandemic potential.

Are there drugs available for prevention and treatment?

Yes, two classes of drugs are available, like M2 inhibitors (amantadine and rimantadine), and neuraminidase inhibitors (oseltamivir and zanimivir). These drugs are used for the prevention and treatment of human influenza. But initial analysis of virus isolated from the fatal cases in Vietnam indicates that the viruses are resistant to M2 inhibitors.

Bangladesh perspective:

No bird-flu case yet has been detected in our country. Recently many people died from unknown disease (24 out of 66) in Joypurhat, Rajbari, Goalonda, Tangail and other districts in the country was due to Nipah encephalitis.

Prevention is better than cure. Prudence, not panic is required to face the challenge of birds-flu and unnecessary panic will serve no purpose but disrupt social and public life.

DR. MD. ISMAIL PATWARY
Associate Professor of Medicine
Sylhet MAG Osmani Medical College
Sylhet

Reference

1. www.who.int/CSR/Avian influenza A (H5N1)-update.htm
2. www.who.int/CSR/Avian influenza A (H5N1)/ cumulative number of confirmed human cases of human influenza A (H5N1) reported to WHO.htm
3. www.who.int/CSR/WHO geographical spread of H5N1 avian influenza virus in birds - update 28.htm
4. www.who.int/CSR/WHO/Avian Influenza- new area with infection in birds -update 34

INFORMATION FOR THE CONTRIBUTORS

THE OSMANI MEDICAL TEACHERS ASSOCIATION JOURNAL **(OMTAJ)** IS THE OFFICIAL ORGAN OF THE TEACHERS ASSOCIATION OF SYLHET M A G OSMANI MEDICAL COLLEGE AND IS PUBLISHED BI-ANNUALLY *(JANUARY AND JULY EACH YEAR)*

The guidelines are in accordance with the "Uniform Requirements for Manuscripts Submitted to Biomedical Journals".[1]

Subscription

The annual subscription rate for the non-members: medical students Taka 100/- and doctors Taka 200/- only.

Submission of manuscripts

The OMTAJ considers manuscripts for publication reporting original clinical or laboratory studies, reviews, case reports, medical progress and brief communications. Manuscript must not be longer than 2700 words. Please provide a word count excluding abstract and references.

Each manuscript must be accompanied by a covering letter from the corresponding author with a statement that the manuscript has been seen and approved by all authors and the material has not been previously submitted to or published elsewhere wholly or partially. A manuscript in duplicate together with tables and illustrations along with a copy in word 97/2000/word XP format in a 3.5" diskette/ CD should be sent to the Editor.

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement) provided they do not contain any material that has been submitted or published elsewhere.

Please note the following: *Your letter must be typewritten and triple spaced; *Its text, not including references, must not exceed 250 words, if it is in reference of a recent journal article, or 400 words in all other cases (please provide a word count). *It must have no more than five references and one figure or table. *It must not be signed by any more than three authors. *Please include your full address, office-time telephone (and/or mobile) number, and e-mail address.

Preparation of the manuscript[2]

All papers must be written in English. All sections of the manuscript should be typed double-spaced, with left alignment in MS Word documents and on one side of good quality bond papers of A4 size (21x 29.7 cm) with margins of at least 2.5 cm., beginning each of the following sections on separate pages: title page, abstract, text, acknowledgments, references, individual tables, and legends for illustrations. Number pages consecutively, beginning with the title page.

Title page

The title page should contain: (1) the title of the article; (2) a short running head of fewer than 40 letter spaces; (3) name of the author (s); (4) institutional affiliation of each author; (5) name and address of the corresponding author.

Abstract

The second page should carry an unstructured abstract of not more than 150 words.

Text

The text of observational and experimental articles should be divided into sections with headings: Introduction, Methods, Results, and Discussion.

Acknowledgments

All acknowledgments including financial supports should be mentioned under the heading 'Acknowledgments' and not as a footnote on the first page or in the text.

References

Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legends by Arabic numerals (1, 2, 3....). Follow the form of references used in the *Index Medicus,* including the style of abbreviations. Try to avoid using abstracts as references: 'unpublised observations' and 'personal communication' may be inserted in the text.

Information supplied in the references section of any manuscript is not usually checked by the editorial

staff, and hence, the concerned author(s) bear total responsibilities of the references.

Following are few examples of references:

1. Standard Journal Article: (List all authors when six or less; when seven or more, list only first three and add *et al).* Akhter A, Haque R, Kholil M, Sultana Z, Fakir MAH. Effects of Oral Garlic on Testicular Micro-architecture of Adult Rat. Osmani Med Teachers Assoc J 2002; 1(1): 1-3.

2. Corporate Author in Journal: Committee for Computer Application in Clinical Microbiology. Bacterial Antimicrobial Susceptibility Pattern, 1988. J Infect Dis Antimicrob Agents 1991; 8: 25-39.

3. Letter to Editor: Yagupsky P, MA Menegu. Intraluminal colonization as a source of catheter-related infection. Antimicrob Agents Chemother 1989; 33: 2025. (Letter)

4. Corporate Author in Book: World Health Organization. On being in charge: a guide to management in primary health care, 2nd ed. England: World Health Organization 1992.

5. Chapter in Book: Wenzel RP. Organization for infection control. In: Mandell GL, Douglas RG, Bennett JE, eds. Principles and practice of Infectious Disease, 3rd ed. USA: Churchill Livingstone Inc 1990: pp. 2176-80.

6. Thesis/ Dissertation: Kaplan SJ. Post-hospital home health care: the elderly's access and utilization [dissertation]. St. Louis (MO): Washington University 1995.

7. Formally published abstracts: Geesy GG, Costerton JW. Bacterial population adherent to submerged surfaces in a pristine mountain stream. Abstracts of the Annual Meeting of the American Society for Microbiology 1977: 235.

8. Articles from symposium volumes: Hamilton LD. Immunogenic polynucleotides. In: Beers RF Jr (ed). Biological effects of polynucleotides: Proceedings of the symposium on molecular biology. New York, Heidelberg, Berlin: Springer Verlag 1971: 107-28.

9. Insert from commercial product: Zyvox (linezolid). Peapack NJ: Pharmacia & Upjohn 2000 (package insert).

10. Web site: Division of tuberculosis elimination. Surveillance reports: reported tuberculosis the United States, 2000. Atlanta: Centres for Disease Control and Prevention, 2001. (Accessed June 27, 2001, at http:/ www.cdc.gov /hchstp/tb/surv/surv2000.)

11. On-line only Journal: Scientist JQ. 2 October 1998, Posting date. History of virology. Am Virol J 1998; 30-150. *(Page numbers may not be available)* [Online.] http://cbxiou.pgr (last accessed October 10, 1998)

12. Online version of print journal: Scientist JQ. History of clinical microbiology. Clin Microbiol 1999; 100: 123-345. [Online]

13. Online version of print books: Scientist JQ. 4 October 1998, Posting date. Culturing methods, 750-800 In: Gavier (ed). Practical procedures for Laboratory, 5th ed. [Online.] DEF Publishing Co. Boston, Mass Http://cbxiou.pgr. (last accessed October 10, 1998).

Abbreviations

Except for units of measurements, abbreviations are discouraged. The first time an abbreviation appears, it should be preceded by the words for which is stands.

Drug name

Generic names should generally be used. When proprietary brands are used in research, include the brand name in parentheses in the methods section.

Permissions

Materials taken from other sources must be accompanied by a written statement from both author and publisher to the OMTAJ for reproduction.

Review and Action

Manuscripts are examined by editorial staff and usually sent to reviewers.

Rejected manuscripts will only be returned if accompanied by stamped and self-addressed envelope.

Letters about potentially acceptable manuscripts will be sent to the corresponding author after the review process is complete.

[1] Uniform requirements for manuscripts submitted to biomedical journals. International Committee of Medical Journal Editors. *Med Educ 1999; 331(1): 66-78. or* http://www.icmje.org/inedx. html.

[2] Additional information regarding manuscript preparation and relevant editorial policy is available in the editorial office.

Covering letter to the Editor for submission of manuscripts

To
The Editor
The OMTAJ
Sylhet MAG Osmani Medical College,
Sylhet-3100

Subject: Submission of manuscript

Dear Sir,

I/we am/ are submitting along with a manuscript for Original Article/ Case Report/ Review Article/ Medical Progress/ Occasional Note/ Others, having title:----------
---

---------- for publication in the OMTAJ.

I/we mention that the manuscript had not been **submitted to,** or **accepted for publication,** or **published** in any form, in any other journal partially, or completely.

We also agree with the following orders of authorship to the manuscript, and we also vouchsafe that the authorship will not be contested by anyone whose name is not listed here.

Authors chronology	Signature
1.	
2.	
3.	
4.	
5.	

Corresponding Author: ----------Signature:----------

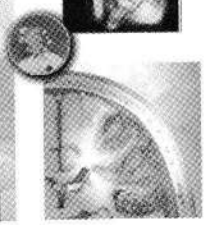
Address:----------

With the best complements

ORION INFUSION LIMITED

NARAYANGANJ

Subarachnoid Hemorrhage (SAH)



- a life-threatening situation

First Time in Bangladesh

Nimocal® tablet

Nimodipine 30 mg

The first line therapy in SAH

Nimocal® provides damage control for SAH

- **R**educes the incidence and severity of neurological deficits in SAH patients.
- **E**ffectively crosses the blood brain barrier due to high lipophilicity.
- **I**mproves the neurological outcome by reducing the incidence and severity of ischemic deficits in SAH patients.
- **I**mproves the outcome of SAH patients with poor neurological status by improving good recovery while decreasing severe disability and vegetative survival.
- **S**ignificantly reduces the risk of cerebral infarction and poor outcome in SAH.

SQUARE
PHARMACEUTICALS LTD.
BANGLADESH

CERTIFIED ISO 9001 COMPANY

Heart for Life SQUARE

www.squarepharma.com.bd