EDITORIAL

Snake Bite

Snake is a subject of entertainment and snake bite is frequently highlighted by the lay press in Bangladesh. It is an important but neglected health problem in the tropics including Bangladesh. The importance of snake bite has been emphasised by the World Health Organization[1]. Epidemiological aspects of snake bite and clinical presentation following bite have been described from different countries. Several measures were taken to characterize the venoms and standardize the antivenoms[2,3,4].

Medically important snake species are the ones that fall into one of the three categories: commonly cause death or serious disability; uncommonly cause bites but are recorded to cause serious effects; commonly cause bites but serious effects are very uncommon[1]. Medically important land snakes in Bangladesh includes Cobra, *Naja, Ghokhro*; Krait, *Bungarus, Shakhinior Kewtey*; Ressell's viper, *Daboia russelli, Chandra bora*; Green pit viper, *Trimeresurus, Gal tawa*; King Cobra, *Ophiophagus, Shankachur* or *khalandhar*.

Effects of snake bite involves different systems depending upon species of snake causing bite. Cobra and krait snake bites are associated with prominent neurotoxicity. Soft tissue necrosis has also been described from some countries including Bangladesh following cobra bite[6,7]. Ressell's viper bite is associated with coagulation abnormalities and renal failure with occasional reports of neurotoxicity, pituitary necrosis and increased vascular permeability.[8] Green snake bite is associated with swelling of the bitten part and coagulation abnormalities. The characteristic features of neurotoxicity following snake bite are ptosis, ophthalmoplegia, difficulty in swallowing, slurred speech, weakness of facial muscles, acial paralysis with loss of tendon jerks and respiratory paralysis[9]. Considerable geographical variation in clinical presentation has been described following bite by same species of snakes in different geographical location of their range including cobra and Russell's viper[10]. Conventional teaching of elapidae snakes as neurotoxic and viperidae snakes as vasculotoxic does not hold good any more. Careful documentation of clinical features following bite by different snakes is needed in Bangladesh.

Diagnosis of species of snake responsible for bite is essential for management of patients. Estimation of venom antigen from swab from wound site, serum or urine by Enzyme Linked Immunosorbent Assay (ELISA) technique has been found to be useful in some countries[11]. Even rapid detection kits are being used in Australia utilizing the same technique. In absence of such facilities, good epidemiological study by identification of brought dead snakes may be helpful in correlating clinical features with type of snakes.

Much ignorance and confusion remains regarding the first aid measures following snake bite amongst medical personnel and general public. Reassurance of the patient, immobilization of the limb, good aseptic washing of the bitten site, non-temparing the wound and rapid transfer of the patient to the nearby health facility should be the aim of the first aid treatment. In health facilities, frequent observation of the patient for appearance of any toxic feature, tetanus prophylaxis, and antibiotic coverage should be immediately started and torniquet, if there is any, should be removed. Basic investgations like bedside clotting test, coagulation profile, full blood count, urine for routine tests, ECG, estimation of urea is needed. In suspected myotoxicity

following bite, serum creatine phosphokinase and muscle biopsy may be helpful.

Appearance of neurotoxicity, clotting abnormality, muscle damage, renal involvement, raised enzymes, leucocytosis, rapidly increasing local swelling, lymphnode involvement are few indications for antivenoms[12]. Antivenoms should be used in the same dose in children as used in adults. Whenever possible, monovalent antivenom is preferable. In Bangladesh, polyvalent antivenom imported from Haffkine's Laboratories (India) are used. Antivenom raised against venoms from locally caught poisonous snakes should be used when local antivenom production starts in the Institute of Public Health, Dhaka.

With the advent of modern techniques and widespread publicity about diseases of modern life (e. g. ischaemic heart disease, stroke, diabetes mellitus) subjects of rural public health importance like snake bite should not be ignored. The identification of key information about the subject by carefully conducted research is needed. What are the medically important poisonous snakes in different parts the country?, what are the clinical features in human victims following their bites?, what are the present traditional treatment methods within the country?, is the antivenom available in Bangladesh is effective in preventing morbidity and mortality?, these are the few questions that need to be answered urgently.

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ORIGINAL ARTICLES

A Hospital Based Study of Snake Bite in Chittagong Medical College

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Summary:

Snake bite is an important but neglected health problem in Bangladesh. Forty-four consecutive cases of snake bite (27 non-poisonous, 17 poisonous) between October 1993 to October1994 were analysed. Age of the patients (mean+s.e.m. years) were 29.6+2.3 with 30 male and 14 female. Six victims brought the dead/living snake to hospital. Identification of poisonous snake was possible in 15 cases (Cobra, *Naja*, eight and Krait, *Bungarus sp*, four and green tree sanke, three) by combination of description and identification by photographs of the snake by the patients or identification of brought snake. The clinical presentations in Cobra bite were cellulitis, necrosis of soft tissue and marked neurological features like ptosis, cranial nerve palsy, muscle paralysis. The features in Krait bite were dominant neurological signs in absence of local swelling/ cellulitis. The mean time lapse before hospitaliaztion was 4.3 hours. Sites of the bites were lower limbs in 27 cases and upper limbs in 17 cases. Traditional treatment and torniquet were applied in almost all cases of poisonous and non-poisonous snake bite before hospitalization. Some of the treatments were found to be harmful and others useless. Polyvalent antivenom (Haffkine) was used in 11 venomous snake bite; in six victims of poisonous snake bite antivenom was not used because of rapid death following admission in two cases, bite by Trimeresurus sp. in two cases and absence of features of toxicity in two cases. The two fatal cases were admitted 2.5 hours and three hours after snake bite.

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Introduction:

Snak bite is an important medical problem in Bangladesh. It is difficult to know the actual incidence as most of the cases of snake bite seek traditional treatment and the snake bite is not reported through the present reporting system of disease profile. Little medical important has, so for, bee given to snake bite, so published information from our country is scanty. An epidemiological study sponsored by the WHO estimated about 8,000 cases of snake bite with over 20% mortality (Hoq, Islam and Sarker, personal communication). The clinical features of envenoming by different species of snakes are available from a number of Asian countries [1,2,3]. There are virtually no detailed reports from Bangladesh where a large number of poisonous and nonpoisonous snakes are present. The only documented information came from Rajshahi Medical College Hospital through a recently published letter where the author mentioned 74 cases of snake bite admitted in medical units over a period of two years [4]. Four of the patients had features of poisoning but detailed information is not available.

The objective of this prospective work was to document various aspects of snake bite including proportion of poisonous bite and clinical features among the hospitalized patients in chittagong Medical College Hospital.

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Materials and method:

Forty four patients bitten by snakes and admitted in Chittagong Medical College Hospital, Chittagong, Bangladesh between October 1993 to October 1994 were prospectively studied The cases with definite evidence of bite (history and fang marks) were only included. Both poisonous and non-poisonous snake bite was included in the study. Indentification of the snake was made by a combination of description and identification by photograph of the snake by the patient or identification of brought snake. Clinical features of the patients were recorded on a standard form. Enquiries about prehospitalization management was noted. Management in hopsital included tetanus prophylaxis, antibiotic and antivenoem as and when indicated.

Lyophilized polyvalent anti-snake venom (Haffkine Biopharmaceuticals Corporation Ltd., Parel, Bombay, India, Batch no. 1527-8, Expiry date Oct. 1995) was used, which contains antivenom against four snakes: Cobra *(Naja naja)*, Common krait *(Bungarus caeruleus)*, Russell's viper *(Vipera russelli)*, Saw scaled *(Echis carinatus)*. No attempt was made to detect venom antigen from wound swab, serum or urine.

Results:

A total of 44 patients (30 male, 14 female) bitten by snakes were studied (Table-I). The snake bite occurred in younger, active group of people with mean age of 29.6 years. The time interval between bite and admission was 4.3 hours. Twenty seven bites (61.4%) occurred on the lower extremity and 17 (38.6%) on the upper extremity. As shown in the table-I, 79% bites occurred in outdoor and mostly by day time (65.9%).

Clinical symptoms and signs are presented in Table II and III. Local pain and swelling were the important indications of systemic envenoming particularly in the patients bitten by cobra and green pit viper. Varying grades of soft tissue swelling and necrosis were found in this study (Fig. 1). Sometimes it was difficult to differentiate the cause of swelling due to occlusion of blood supply by torniquet from the toxic effects of injected venom (Fig. 2). Extensive swelling of the bitten limb due to haemorrhage in the tissue space was found following bite by green tree snake (Fig. 3).

Fig-1: *Photograph of leg showing necrosis of soft tissue on the dorsum of foot exposing the blood vessels following bite by cobra, Naja.*

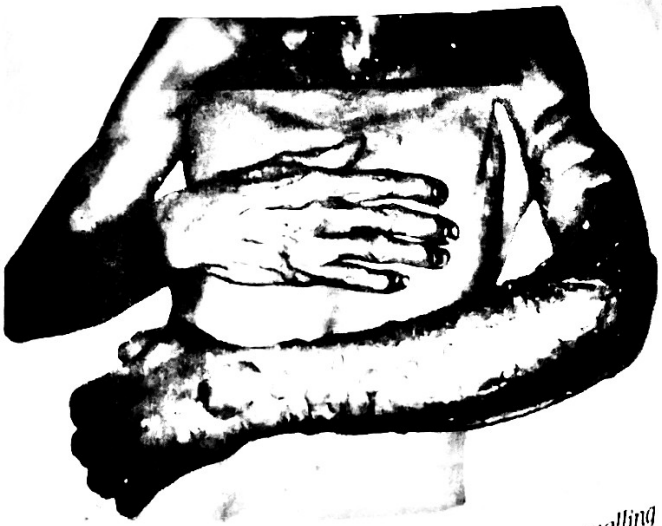

Fig-2: *Photograph shows extensive swelling and multiple blisters involving left forearm. Note: Multiple ligature marks over left arm.*

Neurotoxic features were the predominant features amongst the victims of poisonous bite that included a variety of symptoms and signs. Ptosis (53%), muscle weakness (47%), external ophthalmoplegia (35.3%), dysphagia

Table–I

Characteristics of study patients

Number of patients (M/F)	44 (30/14)
Age (mean ± s.e.m)	29.6±2.3
Bite-admission interval in hours (mean, range)	4.3 (0.5-96)
Site of bite:	
Foot and ankle	27 (61.4%)
Upper extremity	17 (38.6%)
Terrain where bite occurred:	
Outdoor	35 (79.5%)
Indoor	9 (20.5%)
Time of bite:	
Day	29 (65.9%)
Night	15 (34.1%)

Fig–3: *Extensive swelling of left leg following bite by a green snake, Trimeresurus sp. Aspirate showed haemorrhagic fluid. The patient had foot drop as well.*

(29.4%), dysarthria (29.4%), weak grip strength (35.3%) are few important features noted in this study. Cyanosis was present in four patients, in three patients nasal regurgitation was the prominent symptom, two patients had dyspnoea and two patients had broken neck sign (Fig 4).

Fig–4: *"Broken neck" sign–weakness of the flexor muscles of neck following bite by a cobra snake.*

Table-II

Symptoms in poisonous snake bite (n-17)

Symptoms	Number	(Percentage)
Local Pain	14	(82.3)
Local swelling	12	(71)
Muscle weakness	8	(47)
Drooping of eye lids	7	(41)
Bleeding from wound	7	(41)
Vomiting	3	(17.6)
Blurring of vision	4	(23.5)
Double vision	4	(23.5)
Dysarthria	5	(29.4)
Dysphagia	2	(29.4)
Dyspnoea	5	(11.8)
Nasal regurgitation	3	(17.6)
Fainting	2	(11.8)
Unconciousness	1	(5.9)

Indenfification of snakes causing poisonous bite was: cobra (8), krait (4) and green tree snake (3). In two fatal cases, species identification could not be done definitely but the obvious swelling and rapidly developing

neurotoxicity at the time of admission were suggestive of cobra bite. Dead snakes were brought by six patients. Indentification of these snakes were Cobra, *Naja kouthia* (2), Green tree snake, *Trimeresurus* (1), Green cobra, *Macropisthodon plumbicolor* (1), Common cat snake, *Boiga trigonata* (2).

Forty-three patients received torniquet with mean number of 2. 6 (Fig. 5). Victims received traditional treatment by ohzas', some of which were found to be harmful, for example incision causing profuse bleeding requiring blood transfusion, foot drop following nerve injury was not uncommon even in non-poisonous bite. Useless treatment like torniquet by mud, application of seedlings over the fang marks are of interest. Recitation (mantrya, or verses) and suction of the bitten site may have psychological effect.

Table-III

Physical findings in poisonous snake bite (n-17)

Extensive swelling	4 (23.5%)
Blisters	3 (17.4%)
Ptosis	9 (1.4%)
	(Complete 3, Partial 6)
Ophthalmoplegia	6 (35.3%)
Impaired opening of mouth	5 (29.4%)
Difficulty in speech, swallowing, talk	5 (29.4%)
Weak grip strength	6 (35.3%)
Tenderness in muscle	2 (11.8%)
Cyanosis	4 (23.5%)

Fig-5: *Torniquets in situ. Note: Number, different materials, and tightness.*

Antivenom was used in eleven patients (dose, 30-100 ml). In all these cases Haffkine polyvalent antienvom was given intravenoulsy. Skin test was not done prior to antivenom administration. One patient developed wheezing and respiratory distress and that was managed by antihistamine and steroid. No patient developed serum sickness during the period of hospital stay. In six patients of venomous bite antivenom was not used. The reasons were rapid death (2) (45 min and 30 min fllowing admission), no features of toxicity (2), in two patient the bite was due to Trimeresurus sp. All the survived venomous snake bite victims received antibiotic and tetanus prophylaxis. Antibiotic was used inititially by parenteral route and later orally. Fasciotomy was done for limb ischaemia in two patients, skin grafting was required in one patient.

Two fatal cases were admitted 2.5 hours and three hours following snake bite. The clinical features in both these cases were swelling at the bitten site and rapidly developing neurotoxicity with respiratory failure due to involvement of respiratory muscles. The bite occurred in hands in one patient while picking up firewood and in foot in the other while on natural call early in the morning. Respiratory support by artificial respiration was not available.

Discussion:

To our knowledge, this study is the first detailed clinical study on snake bite in Bangladesh. Snake bite is an occupational hazard of young and active male people in Bangladesh like other tropical countries. The morbibity and mortality in such young people

will affect productivity of the country. The delay in admission following bite occurred due to remote rural area of bite and time spent for traditional treatment before hospitalization. Nonvenomous bite outnumbeing the venomous bite in this study is consistent with other observation on the nature of snake bite[5]. Most of the bites in outdoor, at day time, and in the lower limb indicate that wearing shoes may prevent at least some of the venomous bites.

Local swelling and tissue necrosis and marked neurotoxicity are the features following cobra bite noted in this study. The clinical effects of Asian cobra bite has been found to be widespread with considerable geographical variation[6,7,8]. For example, severe neurotoxicity with mild local envenoming has been found in the Philippines, severe tissue necrosis without neurotoxicity in Malaysia, and mixed neurotoxicity and severe local effects in Thailand and Srilanka. Using a multivariate analysis, Wuster and Thorpe opined that Asiatic cobra which was previously thought to be monospecific should really be divided into at least eight full species[9]. These findings are consistent with clinical observations from different localities.

In presumed krait bite, neurotoxicity in absence of local swelling were the predominent features among the victims who were bitten at night. Whether two different types of cobra were causing the symptoms needs to be investigated by confirmation of the venom antigen by Enzyme Linked Immunosorbant Assay (ELISA). Early morning neuroparalytic symptoms have been described amongst jhuggi dwellers and was diagnosed and treated as krait bite even in absence of history of snake bite[10]. Diagnosis was made in a clinical setting where people used to sleep in the floor and ofter krait bite produce a fine puncture without local swelling. Early treatment in such a suspected case may prevent the mortality.

The victims of poisonous green snake suffered for prolonged coagulation defect for few days. There was no other systemic effects. Antivenom that is available in Bangladesh (Haffkine) would not be effective against the green snake bite.

The preference for the traditional treatment by the public is partly due to superstition and ignorance, and may also be due to belief that scientific treatment is not available in hospitals. Various treatment are also practised in other Asian countries. De Silva described different traditional treatment in Sri Lanka [11]. The application of torniquet was not proper in terms of site, number, and tightness in most of the victims in this study. Application of single torniquet and immobilization of limbs like the management of fracture should be emphasised to public and to snake bite 'physicians'. The effect of tight torniquet, ischaemic damage to the limb and peripheral nerve injury must have been overlooked in our population of snake bite. The utility of torniquet is to prevent proximal spread of venom and it has been found to be ineffective by studies in Thailand and Myanmar [12,13].

Haffkine polyspecific antivenom was used in 11 patients and in three patients anticholinesterase, neostigmine, was used as well. As venom antigen was not possible to measure, sigle dose of antivenom was used in this study. The response to antivenom was satisfactory in reversing the neurotoxic features, however, tissue necrosis in some of the patients of cobra bite in this study could not be prevented. Theakston et al also noticed ineffectiveness of Haffkine antivenom in preventing the tissue necrosis following cobra bite in Sri Lanka[6]. Further study is needed to see the efficacy of the antivenom in reversing the neurotoxicity with or without anticholinesterase.

Th utility of anticholinesterase in neurotoxic snake bite was stressed as early as in 1972 by Banerjee et al[14] in Asian cobra bite and later the view was confirmed by controlled trials in the Philippines[15]. Anticholinesterases competitively inhibits the effect of the venom at neuromuscular junction without neutralizing the venom.There is now consensus that

edrophonium (Tensilon) test should be performed in all paralytic envenoming and anticholinesterase may be administered in those cases with positive response.

The two fatal cases died of respiratory failure which indicated the need of ventilatory support by artificial respiration in hospitals dealing with snake bite victims.

This hospital base study indicates the urgent need of further research in the field of snake bite in Bangladesh in both community level and hospitals. Few examples of such priority research includes–patttern of snake bite, methods of traditional treatment, measurement of specific venom antigen, efficacy and side effects of antivenom, and long term effect of snake bite.

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Excision of Pterygium and Its Recurrence

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Summary:

Over two separate periods of time, surgical exicision was performed in one hundred consecutive eyes for primary pterygium of one hundred patients at the National Institute of Ophthalmology and Sher-e-Bangla Medical College Hospital. These patients were evaluated for recurrence of their pterygia in a follow up re-examination study. Follow up period was of six months to two years. The overall recurrence rate was 12% after a common therapy. The recurrence rate was higher in younger age group people and no recurrence occurred above the age of forty. It is concluded that the recurrence is commoner in younger age male people and virtually nil in female patients in all age groups.

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Introduction:

Pterygium, a disease of unknown origin and pathogeneis, is a chronic condition characterised by fleshy triangular encroachment of a pingecula onto the cornea, usually on the nasal side bilaterally[1]. It is thought to be an irritative phenomenon due to utraviolet light, drying, and windy environments, since it is common in persons who spend much of their time outdoors in sunny, dusty, or sandy wind blowing surroundings[1]. It occurs in the interpalpebral fissure and consists of a epithelium of conjunctival type which covers a highly vascularised structure of blood vessels and loose fibrous connective tissue[2]. The pathological findings in the conjunctiva are the same as those of pingecula and in the cornea, there is replacement of Bowman's layer by the hyaline and elastic tissue. The statistics concerning the post-operative recurrences of paterygia vary considerably[3]. This is a fairly common condition and there is no study regarding the post-operative recurrences of pterygium in Bangladesh.The purpose of this study is to see the post-operative recurrence rate in primary pterygia following surgical exicision.

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Materials and method:

The study included one hundred eyes of one hundred patients where corneal involvement was more than 1 mm. Seventy six patients attended the out–patient department of the National Institute of Ophthalmology and twenty four patients were recruited from the eye out–patient department at Sher-e-Bangla Medical College Hospital over two separate time periods. Table I summarises the total number of cases and type of pterygium and Table II shows age and sex distribution of the patients.

For initital treatment topical oxybuprocaine hydrochloride was applied over the involved eye and 2% lignocaine with epinephrine was injected subconjunctivally at the pterygium site.A Keeler's Panaromic loupe having six times magnification was used to facilitate the corneal resection of pterygium. The exicision was started O. 5 mm central to the Cap of pterygium with the help of St. Martin's forceps and razor fragment holder. A superficial keratectomy was performed beneath the pterygium until the limbus was reached. Conjunctiva was then dissected from the pterygial mass at the limbus to reach the insertion of medial or lateral rectus muscle. Pterygial mass was then separated from the sclera and spring scissors were used to excise the pterygium completely. Bleeding was controlled with thremocautery. The sclera between the limbus and the insertion of rectus muscle was scrapped free of all tissues or

debris with the razor fragment holder. Scleral surface was cauterised superficially and covered with conjunctiva in such a manner that 3 to 4 mm bare sclera was left at the limbus. Eye was dressed with chloramphenicol drop and post -operatively treated with topical dexamethasone and neomycin drop four times daily for three weeks. The post-operative follow up was done for six months to two years.

Results:

Table III shows the post-operative results of excised pterygia according to age and sex. In 10-20 years age group two male patients had post-operative recurrence of pterygia. In 20-30 years age group, five male patients out of 16 had post-operative recurrence and one of the females had recurence. Post-operative recurrence occured in three out of 28 male patients in 31-40 age group. No recurrence occurred in any patient of 41-50 and 50 + age group.

Table-I

Age and sex distribution of patients of pterygium

Age (yrs)	10–	20	21–	30	31–	40	41–	50	50+	
Sex	M	F	M	F	M	F	M	F	M	F
	2	1	16	10	28	9	8	8	15	3

Table-II

Number of cases of pterygium and their location on the cornea

Unilateral	Bilateral	Binasal and Bitemporal	Total
Nasal 62	Nasal 34	2	100
Temporal 2	Temporal–		

Table-III

Postoperative result of excised pterygia according to age and sex

Age (yrs)	10-	20	21-	30	31	40	41	50	50+	
Sex	M	F	M	F	M	F	M	F	M	F
Recurrence	2	-	5		3	-	-	-	-	
No recureence	1	11	10	25	9	8	8	15	3	

Discussion:

In this series, the overall post-operative recurrence rate of pterygium was 12%. There was also found a relationship between recurrence and age of the patients. In the age group upto 40 years, there were 12 recurrences in a series of 66 cases (18.18%) whereas above the age of 40 years, of 34 cases, no one had recurrence. Zauberman in his study also found a relationship between recurrence and the age. He got 92% recurrence in the age groups up to 40 years and 26.7% above the age of 40 years.[3]

Progressive type of pterygium tends to occur in the younger age group and this may explain the greater tendency of recurrence in that age group and the regressive type tends to occur in the older age group and lack of recurrence in this type may be due to age factor[4].

Lewallan also found that the age of the patient was strongly associated with recurrence regardless of which surgical procedure was followed[4].

In this study, significant difference could be demonstrated in the incidence of recurrance between the two sexes. Out of 31 female cases, none had recurrence of pterygium. This is a new finding. Most of the authtors did not find any significant difference between sexes in pterygium recurrence[3,4,5]. This may possibly be due to less exposure of our female patients to ultraviolet rays following operation.

The statistics concerning the post-operative recurrences of primary pterygia vary considerably. Some authors such as Kemal and Tower did not report of any recurrence[5,6] and Escapini reported a very low recurrence rate, 1% only[7]. On the other hand a high incidence of recurrence was reported by Gibson (30%)[8] and Cameroon (69%)[9]. Lewallan used conjunctival autografting technique for pterygia removal and recurrence rate was 21%[4].

According to Spires , pterygia have recurrence rates of 30% to 50% with currently available surgical procedures[10]. Sebban and

Hirst got 46% recurrence rate using a number of therapies[11]. Ashaye got 40% recurrence rate in his study using 'Bare sclera' method[12]. Recurrences vary from climate to climate. In hot sunny dusty climate, recurrences are more common. The frequency of recurrence partly depends on the operative technique although its role in prevention of recurrence may not be that important[3].

Over the last decades there have been a tremendous work done on pterygium and various treatment modalities employed to reduce recurrence rate in pterygium following excision but even then recurrence rate could not be reduced to a satisfactory level. Various modifications of ß-radiation have been tried to reduce pterygium recurrence but result was not that satisfactory[13-17]. A study of ß-radiation with strontium–90 found the post-operative recurrence rate to be 6.03%[18]. Recently, mitomycin C ophthalmic drop has been used by some authors post-operatively with some success but corneal melting has been reported as complication[18-20]. As mitomycin is an anti-neoplastic agent, it should be used cautiously. Anduze used merest sclera technique (surgery), a procedure in which the injured limbocon-junctival area is covered completely with superior and inferior conjunctival flaps so that the tear film can be reestablished[21]. After a one year follow up recurrence rate was 2.1%. This is a surprisingly low recurrence rate but this was a small series and replication involving a larger population can only be conclusive.

It could thus be concluded that pterygia recurrences are common in youger age male population and virtually nil in females. Newer search should be continued to reduce the pterygia recurrence to a further lower level.

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Psychosocial Stressors in Depression and Schizophrenia

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Summary:

A controlled comparison between 65 depressed patients and equal number of schizophrenic patients was conducted on the measures of psychosocial stressors in the 12 months before the onset of illness. Overall, depressives reported significantly more stressors than schizophrenics and this excess was limited to family arguments, marital discord and sex difficulties. The schizophrenics reported significantly higher frequency of lack of family support. No significant difference of severity of stressors was found between the two groups. The depressives reported excess of stressors extended over a longer period before the onset of illness than schizophrenics. The findings support the quantitative and qualitative influence of stressors in the genesis of these two psychiatric disorders.

(J Bangladesh Coll Phys Surg 1995; 13: 12-18)

Introduction:

It is well established from a large body of research that there is definite causal relationship between psychosocial stressors and psychiatric disorders. This is particularly evident for depressive disorder where depressed patients experienced significantly excess of stressors prior to the onset of disorder[1-5]. Schizophrenic patients were also reported to experience more stressors before the onset of the disease[6-10].

Comparative investigations between depressives and schizophrenics reported that though depressives experienced more stressors than schizophrenics, this excess involved only certain types of stressors. Depressives reported more exit, severe and varieties of undesirable stressors particularly those involving interpersonal relationships than schizophrenics[9,11,12]. Again it is found that differences between depressives and normal controls involved different types of stressors and extended over a long period of time before onset than did differences between schizophrenics and controls[13].

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The present study was designed to compare the measures of psychosocial stressors in the one year before the onset of illness between depressed patients and schizoprenic patients. Different observations will give some idea about the extent of relationship between stressors and these two major psychiatric problems in Bangladesh.

Materials and method:

The study was conducted at Institute of Mental Health and Research and psychiatry department of Sir Salimullah Medical College and Mitford Hospital in Dhaka. Both the institutions have combined psychiatric outpatient and inpatient department. The duration of study was January, 1993 to June, 1993. A consecutive series of 135 depressed patients (100 outpatients and 35 inpatients) and 100 schizophrenic outpatients satisfying DSM III-R diagnostic criteria for major depressive disoder and schizophrenia were collected[14]. The exclusion criteria for depressive disorder was depression secondary to other disorders and that for schizophrenia was mental retardation, substance abuse, epilepsy or otherwise complicated or doubtful conditions. For the present analysis, 65 cases from both groups were matched completely on sociodemographic variables. Of these, 40 were male and 25 were female. The male-female ratio was 1:0.62. Their age ranged between 16 and 37 years with a mean of 24.77 years (SD±4.92). Majority of the subjects were either

educated upto primary level or illiterate with 22 and 19 cases respectively. Graduates were only two. Among the subjects, 17 were housewives, 11 service holders, 10 unemployed and the rest were of other occupations. Urban-rural distributions were 34 and 31 cases respectively. They were predominantly of lower or middle income group with 36 and 28 cases respectively and only one case in each group were of higher income status. Thirty four cases were unmarried and 27 married. Only three and one cases respectively were found separated and divorced

All the patients of the both groups were interviewed by one of the authors with informed consent. In addition to psychosocial stressors, the interview covered sociodemographic variables and physical and mental health status.Psychosocial stressors were assessed on the basis of axis-IV of the multiaxial evaluation system of DSM III-R which provides the Severity of Psychosocial Stressors Scale (SPSS)[14]. Stressors during the 12 months prior to the onset of depression or schizophrenia were recorded. Individual stressors and their types were considered according to this scale with slight modification which was necessary in our socio-economic and cultural context. The severity of stressors were evaluated and rated according to code 0-6 given in the SPSS. The indiviaual stressors were grouped into month by month durations and further specified as either predominantly acute events (duration less than six months) or predominantly enduring circumstances (duration greater than six months).

The data was processed and comparison was made between depressive and schizophrenic group. Statistical analysis involved two-tailed t-tests and chi-square tests with Yates' correction.

Results:

The depressed patients and schizophrenic patients were compared on measures of psychosocial stressors. The depressives reported a total of 164 stressors, with a mean of 2.52 stressors (SD±0.95) while schizophrenics reported a total of 95 stressors, with a mean of 1.46 stressors (SD±.93). The difference was significant. Overall, depressives experienced approximately 75% more stressors than schizophrenics (Table-I).

Frequency of individual psychosocial stressors were next examined and for each stressor the significance of difference between the two groups were tested. This analysis indicated that though overall increased frequency of stressors in the depressives was paralleled by increased frequency of the most of the individual stressors but for only three stressors, the differences were significant at 2% level or better: (1) family arguments, (2) sex difficulties; (3) marital discord. Most of the other stressors were also reproted more in the depressives, but they occurred too infrequently in either population for differences to achieve statistical significance. Eight stressors were reported more frequently in the schizophrenics: lack of family support, marriage, birth of first child, neglect of parent, problem with neighbours, excessive work load, death of child and serious physical injury. Among them, only one stressor namely, lack of family support reached the level of significance at 1% level. Otherwise, general frequency of these stressors was also very low and difference in their presence between two groups were not significant (Table-II).

The types of psychosocial stressors are set out in Table -III to explore further possible

Table-I

*Comparison of depressed patients and schizophranic patients on measures of psychosocial stressors**

Measure	Depressed group	Schizophrenic group	t-test
Total stressors	2.52±0.95	1.46±0.93	2.95;p<0.01
Severity rating on SPSS	3.98±1.08	3.57±1.31	1.95;p>0.05
Duration of stressors in months	7.7 ±3.47	6.2 ±3.8	1.85;p>0.05

* Data are expressed as mean ± SD

Table-II

Frequency of individual psychosocial stressors

	Depressed group		Schizophrenic group		Significance*
	Number (N=65)	%	Number (N=65)	%	
1. Family arguments	30	46.15	13	20.00	<0.01
2. Sex difficulties	11	16.92	0	0.00	<0.01
3. Marital discord	25	38.46	0	16.92	<0.02
4. Serious financial problem or loss	20	30.77	10	15.38	NS
5. Unemployment	6	9.23	4	6.15	NS
6. Broke up with boy/girl friend	5	7.69	2	3.08	NS
7. Recurrent physical abuse by husband	5	7.69	0	0.00	NS
8. Extreme poverty	5	7.69	3	4.61	NS
9. Marital separation	4	6.15	2	3.08	NS
10. Divorce	4	6.15	1	1.54	NS
11. Husband abroad	4	6.15	1	1.54	NS
12. Trouble with boss	4	6.15	3	4.46	NS
13. Serious chronic illness in self	4	6.15	0	0.00	NS
14. Extramarital relationship of husband	3	4.61	0	0.00	NS
15. Loss of job	3	4.61	2	3.08	NS
16. Rejection/Neglect by husband	2	3.08	1	1.54	NS
17. Death of parent	2	3.08	0	0.00	NS
18. Extreme job dissatisfaction	2	3.08	2	3.08	NS
19. Failure to go abroad for employment	2	3.08	0	0.00	NS
20. Serious illness of other family member	2	3.08	1	1.54	NS
21. Physical abuse by others	2	3.08	1	1.54	NS
22. Death of spouse	1	1.54	0	0.00	NS
23. Second marriage of husband	1	1.54	0	0.00	NS
24. Death of family member	1	1.54	0	0.00	NS
25. Problem with friends	1	1.54	1	1.54	NS
26. Problem with associates	1	1.54	1	1.54	NS
27. Failure in examination	1	1.54	1	1.54	NS
28. Threat to personal safety	1	1.54	0	0.00	NS
29. Husband left home/absconded	1	1.54	0	0.00	NS
30. Arrest	1	1.54	0	0.00	NS
31. Law suit or trial	1	1.54	0	0.00	NS
32. Serious chronic illness of child	1	1.54	0	0.00	NS
33. Unwanted pregnancy	1	1.54	0	0.00	NS
34. Lack of family support	2	3.08	15	23.08	<0.01
35. Marriage	3	4.61	7	10.77	NS
36. Birth of first child	1	1.54	4	6.25	NS
37. Neglect of parent	0	0.00	2	3.08	NS
38. Death of child	1	1.54	2	3.08	NS
39. Problem with neighbours	2	3.08	3	4.61	NS
40. Extreme work load	2	3.08	3	4.61	NS
41. Serious physical injury	0	0.00	1	1.54	NS

*x^2 with Yates' correction

NS = not significant

Table-III

Stressors grouped by types

Type	Depressed group	Schizophrenic group	Significance*	Stressors included in type
Conjugal	56	22	<0.001	Marriage, Discord, Divorce, Separation, Death of spouse, Sex difficulties, Second marriage of husband, Extramarital relationship of husband Recurrent physical abuse by husband, Rejection/Neglect by husband
Family	37	35	NS	Arguments, Neglect of parent, Lack of support, Death of parent, Death of closed family member, Death of child, Birth of first child
Financial	25	13	NS	Serious financial problem/loss Extreme poverty
Occupational	18	17	NS	Unemployment, Loss of job Excessive work load Trouble with boss, Extreme job dissatiafaction, Failure to go abroad for employment, Failure in examination
Other interpersonal	10	4	NS	Broke up with boy/girl friend Problem with friends Problem with associates Problem with neighbours
Living circumstances	6	1	NS	Threat to personal safety Husband abroad, Husband left home/ absconded
Physical illness or injury	7	2	NS	Serious chronic illness in self Serious illness of family member Serious illness of child Serious physical injury
Legal	2	0	NS	Arrest, Law suit or trial
Other stressors	3	1	NS	Physical abuse by others Unwanted pregenancy Physical abuse by others

* x^2 with Yates' correction

differences. The individual stressors were grouped into types according to the social area of activities. For each type, frequencies were again calculated and significance of differences were tested. Of the nine types, only conjugal type of stressors was significantly higher in depressives than schizoprenics. The stressors related to financial, other interpersonal, physical illness or injury and living circumstances were also found more in depressives than schizophrenics although differences did not achieve statistical significance. In the family and occupational affairs frequencies were similar in both groups. Other types were though found higher in depressives, they were too infrequent to reach the statistical significance.

As far as the severity of the psychosocial stressors is concerned, mean score for depressives on SPSS severity rating was 3.98 (SD±1.08) and that for schizophrenics was 3.57 (SD±1.31). This indicated that overall severity of the stressors was found slightly higher in depressives than schizophrenics but the difference here was not significant (Table-I). Further, the severity of the stressors was recorded according to the frequency of each code of severity on SPSS and differences of significance were tested as before between two groups. It also revealed that no significant difference was found between two groups in all levels of severity. Only, the extreme form of severity was found with greater difference of frequency in depressives but again this difference did not reach significant level (Table-IV).

The mean duration of psychosocial stressors was found 7.7 months in depressive group and 6.2 months in schizophrenic group. The difference was not significant (Table-I). Predominantly enduring circumstances were found more in depressives with 97 (59.15%) events and predominantly acute events were found excess in schizophrenics with 55(57.89%) events. Only significantly higher frequency of predominantly enduring circumstances were found in depressives than schizophrenics ($P<0.001$). When monthly distributions were tested between the two groups, it revealed that significantly higher differences were found in eight months, nine months, 10 months and 12 months in depressives than schizophrenics at 5% level or higher.

Table—IV

Severity of stressors

Severity		Depressed group		Schizophrenic group		Significance*
Code	Term	Number	%	Number	%	
1	None	3	4.61	9	13.85	NS
2	Mild	2	3.08	5	7.69	NS
3	Moderate	11	16.92	12	18.46	NS
4	Severe	28	43.08	30	46.15	NS
5	Extreme	19	29.23	9	13.85	NS
6	Catastrophic	2	3.08	2	3.08	NS
	Total	65	100	65	100	

*x^2 with Yates' correction NS= not significant

Discussion:

Before the evaluation of the findings of the study, the possibility must be considered that there might be reporting or methodological artifacts. Retrospective reporting of the experience of psychosocial stressors before the onset of illness may be distorted by the presence of psychiatric disturbance. Onset is harder to date in schizophrenics than depressives and some patients tend to drift into illness without clear-cut onset. Moreover, patients may search for events to explain the onset of illness. Stressors which occur after the onset and are consequences of the disorder may be confused with its causes. Multiapproach techniques were applied to overcome these problems. Investigation was delayed until after improvement and information also obtained from the informants. A high agreement between patients and relatives was found in our study which is similar to a previous report [13].

SPSS was used to measure the psychosocial stressors which was not standardized in our socio-cultural setting, hence some difficulties were experienced during their administration on subjects. It contains some events which are not considered as stressors and lacks many events, are perceived as stressful in this setting. Again some severe stressors are actually not so severe in our society. Reverse is also true in case of some other stressors. Though slight modification was done to overcome some gross anomaly, yet we admit the existence of limitation of scale to quantify stressors on the subjects.

In this study, overall, depressives experienced 75% more stressors before the onset of the disease than did schizophrenics. The finding has the general similarities with the findings of other studies.[6-10] In a previous study, depressives and matched schizophrenics were compared with respects to life events experienced during the six months before the onset of illness and overall the depressives experienced approximately 50% more events than the schizophrenics.[9] The excess of stressors in depressives involved only certain types of strtessors in present study. The depressives reported significantly excess of family arguments, sex difficulties and marital discord. Financial problem or loss was also found notably higher in depressives. In a broad category, only conjugal type of stressors were found significantly higher in depressives than schiophrencs. Almost all stressors in this type were undesirable or exit in nature. Two other types, financial and physical illness or injury were also found more in depressives with suggestive differences. This result is more or less consistent with previous findings[9,12,13]. In one of the studies, depressives reported more exit and undesirable events, particularly those involving interpersonal arguments and events related to finance and health[9]. In contrast, schizophrenics experienced significantly excess of lack of family support than did depressives in our study which indicates its strong relationship in developing schizophrenia. It was suggested in a previous report that schizophrenics may be particularly sensitive to disruption of family life.[15] Though entrance events like marriage, birth of first child were experienced more frequently by schizophrenics with notable differences, no conclusion can be drawn about the relationship of entrance or desirable events in the genesis of schizophrenia as the differences were not significant between the two groups. Stressors related to family and occupational events were reported at about the same levels by the both groups, suggesting that they were involved in the same extent in developing both the disroders.

Lack of significant difference in mean severiety between the two groups suggests that severity of the stressors has equal effect in the genesis of schizophrenia and depression. Analysis of the individual severity coding also revealed no significant difference in all levels of severity between two groups which is in favour of the above statement. However, extreme form of severity was found more in

depessives with a suggestible difference between the two groups which indicates that higher grade of severity may have more influence on the onset of depression than schizophrenia.

Though not significant, mean duration of stressors in depressive group before the onset of illness has been found longer than that of schizophrenic group. The broad categories of duration further confirmed this statement where predominantly enduring circumstances were found significantly higher in depressives than schizophrenics. In month by month recording of stressors between two groups revealed that the longer duration of stressors in depressives was particularly evident in five months, nine months, 10 months and 12 months at significant levels. These findings simulate with the previous observation where the excess of stressors were reported to extend over a longer period before onset for depressives than for schizophrenics.[13]

The result of the study points out that the psychosocial stressors are involved both quantitatively and qualitatively in the genesis of depression and schizophrenia, but the relationship is stronger for depressives. Moreover, it specifies certain types of stressors which are particularly important in this respect. The role of severity of the stressors in genesis of these disorders needs further investigations.

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Management of Peripheral Vascular injury: Experience of 32 Cases at Medical College Hospital, Rajshahi

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Summary:

Thirty two consecutive cases of peripheral vascular injury, admitted in the surgical and orthopaedics units of Rajshahi Medical College Hospital during one year period from July '89 to June '90 were studied.

patients were between the age of 10 and 55 years. Out of 32 cases, 17 were admitted with vascular injury following road traffic accidents (RTA) and fall from height, five following gun shot and 10 inflicted by sharp cutting weapons or instruments. Among the vessels involved, popliteal vessel injury was the most frequent followed by radio-ulnar, brachial, femoral and tibial vessels.

Primary vascular repairs in the form of either lateral repair or end to end anastomosis or vein patch angioplasty or autovenous grafting in all the case within six to ten hours of trauma. Restoration of blood flow was achieved in all cases. Post-operative follow-up of 18 patients upto one year showed normal patency of repaired vessels.

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Introduction:

Historically, death from haemorrhagic shock or limb amputation was a common outcome of major vascular injuries. Even in the present time an increase in violent crime coupled with increased number of vehicular trauma accounts for large number of vascular injuries often with fatal outcome.

The majority of reported vascular injuries occur in the extremities. This may be due to the long course of vessels in the extremities as well as the higher prehospital mortality of truncal vascular injuries[1].

Isolated vascular injuries in many cases may ultimately lead to disability, amputation of this part or even death. These complications of vascular trauma are very common in those places where ligation of a bleeding vessel is still the treatment of choice.

This report, first of its kind from Rajshahi Medical College Hospital, includes an analysis and result of 32 successfully managed cases of peripheral vascular injury.

Materials and method:

This report includes 32 cases who have undergone different types of vascular reconstructions for peripheral vascular injuries. All patients were from the surgical and orthopaedics departments of Rajshahi Medical College Hospital and treated during one year period from July 1989 to June 1990.

Cases included in this report had history of either RTA or injury inflicted by sharp cutting weapons or gun-shot. All patients were managed on emergency basis within six to ten hours of trauma. Patients with associated bony fractures received simultaneous orthopaedic and vascular care.

Assessment of patient as a whole (type of injury, amount of blood loss etc.) in the pre- and post-operative periods were based on clinical observations. Due to lack of facility, vascular laboratory investigations (Doppler flowmetry, angiography etc.) could not be done.

All operations were performed under general anaesthesia. Duration of operations varied from 1.5 hour to three hours.

Besides whole blood (one to three units) transfusion, high molecular weight dextran,

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antibiotics and heparin (including regional heparinization) were used during operation and in the postoperative period routinely. Oral anticoagulants were not used due to the lack of laboratory monitoring facility for coagulation of blood.

For injured vessel repair, 4/0, 5/0 or 6/0 atraumatic vascular silk or prolene were used.

Out of 32 subjects, 18 could be followed up at three, six and 12 months interval. Results of vascular repair were assessed clinically. Absence of chronic vascular insufficiency or oedema and presence of satisfactory peripheral pulsation were found in all 18 cases.

Results:

Among the study population there were only three females and rest were males. Age distribution of the patient in this study are shown in Table-I. Most of the patients were in the age group of 21 to 30 years followed by those between 31 and 40 years.

Popliteal and superior extremity vessels were affected most frequently (Table-III). Out of 12 popliteal vessel injuries three were associated with bony fracture around knee joint or lower one third of femur. There were six cases of supracondylar fracture humerous associated with brachial artery injury and one Colle's fracture with radial artery injury. In one patient, the brachial artery was contused following blunt trauma over mid arm.

Table-I

Age distribution of patients undergoing emergency vascular surgery

Age group in years	Number of patients
11-20	6
21-30	14
31-40	9
41-50	2
above 50	1
Total	32

Table-II

Types of vascular injury

Sl.No.	Types of Injury	Number
1.	Non penetrating	
	a) Following road traffic accidents (RTA)	
	i) With bony fractures	5
	ii) Without bony fracture	7
	b) After fall from height with bony fractures	5
2.	Penetrating	
	a) Stab injury	6
	b) Accidental	4
	c) Gun-shot	5
	Total	32

Most severe form of soft tissue and vascular injuries occured after RTAs and gun-shot. In four cases of RTA, there were extensive lacerations of soft tissues of thigh and popliteal vessels without any major bony discontinuity. Proper and timely care of lacerated tissues and popliteal vessels (1-lateral repair and 3-end to end anastomosis) saved those limbs. Four patients with gun-shot injury and another two of RTA (not included in this study) had extensive femur fragmentation and lacerations of femoral and popliteal vessels and nerves.

Table-III

Incidence of injury of different blood vessels

Sl. No.	Name of Artery	Number	Name of vein	Number
1.	Popliteal	12	Popliteal	10
2.	Radial/Ulnar	6		
3.	Brachial	7		
4.	Femoral (common & superficial)	4	Femoral	3
5.	Tibial anterior	3		
	posterior	1	Internal jugular	1
Total Artery involved		33	Vein involved	14

Efforts to establish vascular patency and external fixation or immobilisation of bony fractures were made but failed. All of them had to undergo amputation of limbs. Two other patients admited in the hospital not less than 14 hours after vascular trauma had irreversible ischaemic changes of leg and amputations were done.

Repair of popliteal vessels in all 12 cases were accomplished by medial incision. Small tangential lacerations in four cases (Table -IV) were repaired directly. In five cases vessels were mobilised and repaired by end to end anastomosis. In three patients the lacerated popliteal artery segments were more than 2. 5 cm, hence they required grafting. Great saphenous veins from the opposite extremity were used for the purpose All 10 popliteal vrins were repaired–five lateral repairs, one segment replacement and four were mobilised and repaired by end to end anastomosis.

Among the superior extremity vessels, affected brachial artery segment in three cases were replaced by cephalic vein graft and two other were repaired by end to end anastomosis. One case of brachial bifurcation injury received vein-patch angioplasty. Both radial and ulnar arteries in four cases and isolated radial artery in two cases were repaired by end to end anastomosis.

Femoral vein in three patients needed direct lateral repair whereas two superficial femoral arteries were repaired by end to end anastomosis. Another case of lacerated injury of common femoral artery near bifurcation needed vein-patch angioplasty to avoid narrowing of the segment. Replacement of lacerated artery with great saphenous veins of opposite limbs were performed in five arteries of the femoro-popliteal segment.

The patient with stab injury to internal jugular vein was one of the difficult cases in respect to repair approach. The injured venous wall could be approached only after cleidiotomy and lateral suture repair under digital control was done.

Table-IV

Types of reconstructive vascular surgery performed

SL.No.	Types of reconstruction or repair of vessels	No. of patients
1.	Lateral repair/direct suture to	
	a) arterial wall	9
	b) venous wall	6
2.	End-to-end anastomosis	20
3.	Vascular segment replacement with autovenous grafts:	
	a) femoro-popliteal segment	5
	b) popliteal artery	3
	c) popliteal vein	1
	d) brachial artery	3
4.	Vein-patch repairing of vascular defect	2

Three patients with anterior tibial artery injury and one with posterior tibial artery injury (just distal to origin) needed lateral repairs.

No postoperative complication except moderate oedema of inferior extremity in three patients was observed, which subsided without any treatment within three to five days of operation.

Out of total 32 patients, 18 could be followed up at three, six and 12 months interval. Among those, six had superior extremity vacular repair, one internal jugular vein repair and rest 11 inferior extremity vascular reconstruction. Absence of chronic vascular insufficiency or oedema and presence of satisfactory distal pulsation were found in all 18 cases.

Discussion:

The prevalence of trauma, both blunt and penetrating, has established the need for increasing knowledge in the management of vascular trauma. The problems associated with vascular trauma are often related to delays in diagnosis and treatment or to lack of efficient manpower and facility.

Vascular injuries can result from both nonpenetrating or blunt and penetrating trauma. Those resulting from blunt trauma comprise approximately 10% of vascular injuries and appear to be increasing in frequency[2]. Nonpenetrating arterial injuries may result from direct compression or torsion and shearing forces, often as a result of rapid deceleration. Such injuries are most often observed following vehicular acceidents (RTA) and fall from height. The usual pathological result of this type of trauma is injury to the tunica intima with a variable degree of disruption of the intima and vascular wall and subsequent thrombosis. All these happens without significant haemorrhage or blood loss. Loss of arterial pulsation, neurological deficit or distal ischemia may be the first indication of occult arterial injury secondary to blunt trauma. However, the presence of a pulse does not exclude the possibility of blunt arterial injury[3].In our series, there was only one such case with successful outcome as a result of timely intervention. In this regard we like to emphasise on the need for exploration even without doing angiography if question of breach of vascular integrity arises.

Penetrating vascular injuries ordinarily result from penetrating bodily injuries such as stab, gunshot or fragment wounds, lacerations and may also result from nonpenetrating injury when fractured bones lacerate the vessels. As a result of injury there may be laceration, penetration or perforation. The common complications of such injury are hypovolumia, ischaemia, haematoma, false aneurysm and arteriovenous fistula. Haematoma is the most common feature of penetrating arterial injury. Nearly one-half of the patients with penetrating arterial injuries also have injuries to adjacent veins [8].

Drapanas et al[4] reported that 90.2% of arterial injuries were due to penetrating arterial trauma and transaction or laceration encountered in over 80%. Out of 32 patients in our series, 17 were of non-penetrating and 15 penetrating vascular injury (Table-II). Out of 17 non-penetrating injuries 10 had vas laceration by fractured bones. Thus number of penetrating injury to blood ves was 25.

All inferior extremity vascular injuries w managed within six hours of trauma, wher in case of superior extremity vesse satisfactory results could be obtained ev when the time lapse was upto 10 hours. In o opinion, it was possible due to the effecti collateral circulation in superior extremi One thing should be pointed out here th injuries to the radial and ulnar arteries a most important when they occu simultaneously. Because in thes circumstances the hand may be significantl ischemic and both arteries should be repaired

Among all major vascular injuries, brachia artery injury account for nine to 28%. Many o these injuries are associated with significant nerve or bony injuries[5]. Fortunately, in the case of brachial artery, successful treatment is aided by the rich collateral blood flow to the arm. We had seven such cases and one of them was successfully repaired even after 14 hours of trauma.

Injuries to the superficial femoral artery are quite common, whereas injuries to common femoral and profunda femoris arteries are quite rare (5% and 1% respectively)[6]. In our series, out of 32 patients only one was with lacerated injury to common femoral artery near bifurcation.

Injury to the popliteal vessels poses a more difficult problem as emphasised by early reports of amputation rates as high as 72.5% from world War II. However, recent reports have noted a decrease in the amputation rate to as low as 10%[7]. Yet, amputation rates for popliteal injuries continue to exceed those for upper extremity vascular injuries and iliac and femoral artery injuries. This increased rate of amputation may be related to delay in recognition of these injuries, inappropriate management of associated soft tissue or popliteal vein injury[5]. We observed that most of the lower limb amputations were after

popliteal vessel injury either after fracture of femur or tibia around knee joints.

Postoperative oedema is one of the distressing complications frequently ecountered after successful vascular reconstructon. This occurs mostly in the lower extremity and may progress to produce necrosis of entire muscle group. In order to avoid this type of compication, specially in cases where a major vessel injury has been present for more than six hours, and a successful vascular reconstruction has been performed, a concomitant fasciotomy should always be done[8]. We had three cases of postoperative oedema which subsided within three to five days of operation.

Analysing the results reported here, we found that the severity of an vascular injury often determines the type of vascular repair to be performed, and to an extent, may also influence the ultimate result. Most injuries can be repaired by simple suture or resection and anastomosis. When an anastomosis can not be done without tension, an autogenous vein grafting is the treatment of choice. We avoided using synthetic grafts in traumatic vascular injuries of the extremities because of the high thrombosis rate and the increased incidence of infection.

The present report comes from a centre where there is no casualty unit, vascular laboratory investigation facility or sufficient trained manpower. Even then 32 cases were successfully managed. In fact, when managing any vascular injury emphasis must be placed on prompt and meticulous repair. We believe that proper assessment and awareness of potential complications will increase our success rate in the management of vascular trauma.

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REVIEW ARTICLE (J Bangladesh Coll Phys Surg 1995; 13: 24-27)

Chronic Suppurative Otitis Media–A Major Cause of Hearing Impairment in Developing Countries

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Chronic suppurative otitis media (CSOM), defined as chronic perforation of tympanic membrane with painless purulent otorrhoea lasting for two weeks or longer, is the commonest cause of persistent mild to moderate hearing impairment in children and young adults in developing countries. Studies in Bangladesh, India, various countries in Africa and amongst certain disadvantaged ethnic groups have shown that CSOM may have a prevalence of between two and 17% among children. The condition usually develops following untreated or inadequately treated acute otitis media usually in the first five years of life and is related to poor socio-economic conditions.

The natural history of the disease is to continue for months or even years with persistent otorrhoea, ossicular destruction and worsening hearing impairment. The prevalence of CSOM is much lower and showing a decreasing trend in developed countries where the prevalence of otitis media with effusion is relatively higher. When the auditory deprivation produced by CSOM occurs during the first two years of life, it is likely to have serious effects on the critical period of young victims' language development, and later can cause significant delays in school progress. CSOM may also lead to serious infective complication such as acute mastoiditis, meningitis and cerebral abscess.

CSOM without cholesteatoma occurs as a complication of untreated or inadequately treated acute otitis media. The disease may be unilateral or bilateral. The length of time of continuous otorrhoea through a perforation necessary to make the diagnosis has been variously set at from three[1] to six weeks[2] to two to three months[3]. Three weeks appears to be an acceptable minimum time since irreversible tissue damage can occur in an animal model within two to three weeks following the onset of acute otitis media[4].

CSOM is of two types viz, Attico-antral and Tubotympanic. Attico-antral type is associated with cholesteatoma and serious complications[5]. The attico-antral type is rare in most developing countries especially Africa [6] and amongst other disadvantaged ethnic groups where the tubotympanic variety is common[7]. However, here we will try to review the available literature in relation to CSOM with different types of hearing impairment with special attention to developing countries.

Due to inadequacy of international statistics, relationship of hearing impairment and the incidence of CSOM is yet to be known. However, the studies so far done in different parts of the world showed almost identical results.

In India, Pal et al had their study on 4, 528 individuals of 835 families living in the area of Alambagh Urban Health Centre of the Department of Social and Preventive Medicine

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(known as Lucknow study)[8]. The hearing loss had been classified by them into four grades depending upon the clinical classification of Mawson[9]. None of the individual had total deafness (over 90 dB loss). Four hundred and ninety-nine (62.8%) had mild, 213 (26.8%) had moderate and 83 (10.4) had severe degree of deafness. Their observation showed that 85.5% of the individuals with severe hearing loss complained of it as a disability. About half (53.8%) having moderate deafness complained of it as symptom, while only about one fifth (21.8%) having mild deafness were just aware of it. They also observed that 451 (56.7%) individuals had conductive, 326 (41.0%) perceptive and 18 (2.3%) mixed type of deafness. According to the study of Pal et al, CSOM is the cause of conductive deafness amongst 50% of the cases below five years, 16.4% between five and 44 years, 16.9% between 15 and 24 years, 21.9% between 25 and 44 years, 19% between 45 and 64 years and 5% above 75 years[8]. Similar studies carried out in groups of school children of India by Misra et al[10], Kamath[11], Gupta[12] and Jain[13] showed disagreement with one another.

In Bangladesh no extensive survey was made at national level. So far three surveys were published.[14,15,16] Amin et al in their survey amongst 2,005 school going children of three to 15 years of age found 340 (16.95%) to have bilateral CSOM with moderate degree of hearing impairment and 101 (5.03%) unilateral CSOM with unilateral mild to moderate hearing impairment[16].

Holborow et al in their study titled 'A study of deafness in West Africa' surveyed four states comprising of 20% of total population of Nigeria to find out the commonest cause of deafness[17]. A total of 803 subjects were found profoundly deaf of which 36% were of congenital unknown origin followed by those due to measles and meningitis numbering 19.35% and 18% respectively. CSOM contributed for three (0.6%) of the cases. But if the survey would have included all catagories, that is, mild, moderate and severe deafness, CSOM would emerge as a major cause.

McPherson and Holborow carried out a study on various aspects of deafness and causes of severe to profound hearing loss in childhood throughout the nation in the Republic of Gambia[18]. A total of 25,961 children of two to 10 years of age were examined and 71 severe to profoundly deaf children were identified with an incidence of 2.7/1000 children. No difference was noted between the incidence of childhood deafness in urban and rural areas. The generally accepted incidence of profound deafness in children living in developed contries is one to two per 1,000. In Gambia, meningitis topped the list as the cause of deafness with an incidence of 31% of the total. 3.1% of the children seen had the CSOM. Otitis media following measles is a real threat to a child's hearing in may parts of Africa. 3.7% of ears examined gave evidence of present or previous middle ear disorders. Same authors did the detailed analysis of clinic attendance of over 6,200 patients (4,500 rural and 1,700 urban), of which two-thirds were children below 16 years of age, where 1500 cases of childhood otitis media were found. Presenting complaints of those patients of urban and rural population justifies two facts: first, otitis media is a disease of young children affecting those up to eight years of age; second, otitis media is slightly more in rural than in urban people. Finally, the authors concluded that otitis media is common in young Gambian children and by 14 years, almost 20% usually suffer from it, leaving them with hearing loss and chronic ear disease (CSOM).

Bastos et al in their survey in Luanda showed that a major portion of sensorineural hearing loss (SNHL) was associated with CSOM and proposed to undertake a prevalence study of the population to find out the relationship of CSOM to SNHL. They recorded 1,110 patients with CSOM, which amounts to 2.7% of the age group between three months and 15 years with a mean age of five years[19].

Sensorineural hearing loss has been described in patients with CSOM by several authors[20,21,22,23,24]. In those studies, deterioration in bone conduction has been used as an indication of SNHL. Paparella compared bone conduction loss in diseased and normal ear of same person of different age groups and found an increased indidence of SNHL in patients with CSOM in all age goups, suggesting that those SNHL were not due to presbyacusis[20].

Extended high frequency (EHF) hearing was studied in children with or without history of chronic or recurrent otitis media. Children with histories of CSOM had poorer EHF hearing than children without such history. The EHF hearing in otitis media children appeared to be related to the severity of otitis media[25].

Similarly, Kennedy made a statistical study on the sensorineural component hearing loss of 595 patients suffering from CSOM, of those, 195 with unilateral CSOM were taken into consideration. They used the criteria of valuation which excluded other possible causes of SNHL, such as exposure to acoustic trauma, ototoxic drugs, cardiovascular disease, past injury and hereditary causes. The contralateral ear was considered as normal. They determined the average sensorineural component in the hearing loss in relation to the age of onset and duration of the disease. On the basis of the data obtained, they concluded that the sensorieural component in hearing loss does not change with respect to the age of onset of CSOM but the duration of CSOM has a significant role[26].

Wilson in his article, "Deafness in developing countries", narrated a real picture of the problem with the hearing disabled, which had also been adopted by the United Nations Assembly. There are about 450 million hearing disabled people in the world, 80% of them live in developing countries, mostly in the poorest communities of Asia, Africa and Latin America[27].

CSOM was the commonest cause of hearing impairment in a study conducted by the Liverpool Hearing Impairment Research Group in Swaziland in 1987 (Unpublished).[28] Prevalence of CSOM was found to be between 14 and 22 per1,000 children aged five to 14 years and a very high prevalence of CSOM amongst certain other disadvantaged ethnic groups was also noticed. A population survey of 3,568 aboriginal children under 14 years of age in Queensland, Australia showed 16.5% to have unilateral or bilateral tympanic membrane perforations of which two-thirds were wet type[29].

Finally, it can be concluded here that acute and chronic otitis media are frequent disorders in children in industrialized as well as in developing countries. Racial, geographical and socioeconomic condition are factors that influence the prevalence. In developing countries, malnutrition, lack of pure water and sanitation are important health problems. These conditions increase the risks of infectious diseases, which in their turn predispose to otitis media and hearing impairment. It is, therefore, important to diagnose and treat otitis media as early as possible to prevent the bad sequelae of CSOM.

Treatment trial with available simple procedure like dry mopping, local instillation of antibiotic drops, removal of predisposing conditions (englarged adenoids, infected tonsils, sinusitis etc) and health education will help at least 50% perforation of the tympanic membrane to heal. Amin et al[16] recommended the following advice for patients with tubo-tympanic CSOM: not to allow water entry into the ear, not to blow nose too hard and not to clean ear with indigenous objects.

Hearing aids actually help mainly in conductive hearing loss. But cost of the aids and the annual cost of batteries to power such aids is a real problem for the people of economically less advantaged countries.

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CASE REPORTS

Three Cases of Disseminated Intravascular Coagulation and Their Treatment Outcome

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Summary:

Three cases of disseminated intravascular coagulation were treated by the author in different military hospitals. One was due to plasmodium falciparum, one due to shigellosis and the third one was due to acute myeloid leukaemia. Two patients survived and one with acute myeloid leukaemia died.

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Introduction:

Disseminated intravascular coagulation (DIC) is a life-threatening thrombohaemorrhagic disorder occurring as a complication of variety of diseases. It is characterized by activation of coagulation cascade that leads to formation of microthrombi throughout the microcirculation. As a consequence, there is consumption of platelets, fibrin and other coagulation factors. Hence it is also called consumption coagulopathy. Secondary activation of fibrinolytic mechanism aggravates the haemorrhagic diathesis[1].

Case reports:

Case No. 1. A twenty-three year old sepoy of an infantry battalion was admitted to Combined Military Hospital, Chittagong Cantonment, on 12 April, 1987 with five days history of intermittent fever, headache and generalised weakness. He was working in Chittagong Hill-Tracts. Patient was toxic and dehydrated; pulse rate was 120/min, and temperature was 103°F. He had no hepatosplenomegaly. Heart and lungs were normal. Blood film showed plasmodium falciparum (count-160/cumm). He was given chloroquine (600 mg stat, 300 mg after six hours and 150 mg b. d. for three days). Symptomatic measures were taken. On the fifth day of admission he suddenly developed profuse haematemesis and melaena associated with epistaxis and haematuria. He became unconscious. Clinical examination showed pulse-150/min, blood pressure-80/50 mm of Hg, temperature-103°F. Optic fundi showed haemorrhage. Haematological examination showed haemoglobin-10.2 gm/dl, PCV-32 L/L, total leucocyte count-5.0×10^9/L with neutrophil-49%, lymphocyte-44%, eosinophil-6% and monocyte-1%, platelet count was 60×10^9/L. Bleeding time was 21 minutes 26 seconds (Duke's method) and clotting time 32 minutes 25 seconds (capillary method). Prothrombin time was 26 seconds (control, 14 seconds). Activated partial thromboplastin time was 58 seconds (control, 33 seconds). Urinalysis showed haematuria. Blood urea was initially normal (45.0 mg/dl), but later raised to 187.0 mg/dl, when he developed acute renal failure. Blood group was AB (Rh-positive). X-ray chest showed pulmonary oedema. Patient was managed with transfusion of 12 units of fresh whole blood, intravenous quinine dihydrochloride (10mg/kg eight hourly in the drip) and co-trimoxazole (two tablets 12 hourly). Intravenous frusemide and vit-K were given as required. Fluid and electrolyte balance was maintained strictly. After about 72 hours, bleeding was controlled gradually, temperature subsided and pulmonary oedema regressed. Blood urea returned to normal after 15 days. Patient was discharged after one month of hospitalization.

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Case No. 2. A four and half a year old child weighing eleven and half kilogram was brought to Combined Military Hospital, Rangpur, by his parents on 18 August, 1991 with the history of frequent passage of loose stool containing muscus and blood, tenesmus, occasional vomiting and fever for 10 days. One day after hospitalization the child developed melaena, haematuria and epistaxis. Clinical examination revealed that the child was toxic, severely dehydrated and severely anaemic but not icteric. His pulse rate was 130/min and temperature was 101°F. Abdomen was soft with tenderness over the colon. There was no hepatosplenomegaly or lymphadenopathy. Heart and lungs were normal and there was no feature of meningeal irritation. There was no history suggestive of peptic ulcer or intake of NSAID. Stool examination showed numerous pus cells and red blood cells. Stool culture revealed growth of shigella group of organism. Haemoglobin was 5.46 gm/dl, PCV was 0.21L/L, total leucocyte count was 15.8x109/L and differential count showed neutrophil-71%, lymphocyte-27%, eosinophil-1% and monocyte-1%. Platelet count was 75,000/cumm. Bleeding time was 11 minutes and 40 seconds and coagulation time was 12 minutes 50 seconds. Prothrombin time was 30 secons (control, 14 seconds). Activated partial thromboplastin time was 60 seconds (control, 30 seconds). Urinalysis showed haematuria with mild albuminuria. Serum electrolytes were sodium-130.0 mmol/L, potassium-3.0 mmol/L and chloride-90.0 mmol/L. Widal test finding was TO-1/80, AO-1/40, BO-1/20. Blood urea, glucose and chest X-ray were within normal limits. The child was managed with nalidixic acid 60 mg/kg body weight daily initially and later with pivmecillinum 40 mg/kg daily for 10 days. He was given adequate fresh blood transfusion. Fluid and electrolyte balance were maintained. Bleeding was controlled after four days. After 26 days of hospitalisation the patient was discharged fit to resume his normal activities. His haemoglobin was restored to 12.0 gm/dl and electrolyte imbalance was corrected.

Case No. 3. A twenty-three year old soldier of an infantry battalion was admitted to Combined Military Hospital, Rangpur Cantonment, on 28 July, 1992, with history of bleeding from gum for two days which was aggravated after dental surgery. Later he developed bleeding from gastrointestinal tract, genitourinary tract and venipuncture sites. He had no purpura. The patient also developed low grade fever after admission. He did not give any history of bleeding tendency or jaundice in the past nor any family history of bleeding diathesis. Clinically he had moderate anaemia but no icterus. He had no lymphadenopathy and hepatosplenomegaly. Bony tenderness was present. Optic fundi showed extensive bleeding. Gum was swollen, spongy and a blood clot was found. Haemoglobin was 8.6 gm/dl, ESR 40 mm fall at the end of first hour, total leucocyte count was $10.0x10^9$/L, differential count was neutropohil-20%, lymphocyte-58%, immature cell (myeloblast)-22% and platelet count-$25x10^9$/L. Bone marrow examination revealed a hypercellular marrow, reduced erythropoiesis and presence of plenty of abnormal myeloblast. Megakaryocytes were markedly reduced. These findings were suggestive of acute myeloblastic leukaemia. Bleeding time was 15 minutes and coagulation time 10 minutes 20 seconds. Prothrombin time one minute and five seconds, activated partial thromboplastin time one minute and 55 seconds. Urine showed haematuria. Blood urea was 64.0 mg/dl. X-ray chest, blood glucose and serum electrolytes were within normal limits. He was transfused with 20 units of fresh whole blood. Heparin was given to prevent thrombin generation. Antibiotic was given to control secondary infection. He became unconscious following intracranial haemorrhage and died on the fourteenth day of admission.

Discussion:

Most of the patients of DIC present with extensive bleeding from multiple sites. Intracranial bleeding, massive gastrointestinal bleeding, shock, renal failure and acidosis are

terminal events. Laboratory manifestations include prolonged bleeding time, prothrombin time, activated partial thromboplastin time, thrombin time, presence of thrombocytopaenia, reduced fibrinogen level and elevated fibrin degradation product.

Patient should be managed promptly by correction of precipitating factors and control of major symptoms, either bleeding or thrombosis. Appropriate antibiotic in adequate doses is required in case of severe sepsis. Prompt delivery of dead foetus and placenta will reverse DIC in case of retained dead foetus and abruptio placenta. In case of severe bleeding, transfusion of fresh frozen plasma, cryoprecipitate and platelet concentrate are required to replace depleted clotting factors and to correct thrombocytopaenia[2]. Treatment with heparin and antithrombin III concentrate are required to reduce thrombin generation and to prevent further consumption of clotting proteins[3-5]. Antifibrinolytic therapy in addition to heparin therapy may be required in patients with acute promyelocytic leukaemia showing hyperfibrinolysis[6]. Each patient must be judged individually according to clinical situation.

Of the three cases presented here, shigellosis and falciparum malaria are treatable infections. If we are aware about DIC and take adequate measures promptly, we can reverse the grave conditions and save the life.

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Hydatid Cyst of the Orbit—A Case Report

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Summary:

Hydatid disease is an uncommon condition in our country. It mostly involves liver and lung. A farmer, aged 30 years, reported to National Institute of Ophthalmology and Hospital with complaints of a swelling in the inner canthus of right eye for two years followed by watering, dimness of vision and headache. The cyst was excised. It was diagnosed as hydatid cyst after histopathological examination. Involvement of the orbit by hydatid cyst is, so far, not reported in Bangladesh. This rarity has led us to report this case.

(J Bangladesh Coll Phys Surg 1995; 13: 31–33)

Introduction:

Hydatid disease is world-wide in distribution. But, it is more a disease of temperate climate than of tropical zone[1]. It is a common health problem in cattle and sheep-raising areas of the world, such as South America, Australia, the Baltic areas, Northern Canada and the Middle East. The nature of the disease produced in man by larval cestodes (hydatid cyst) of the genus Echinococcus depends upon the species of cestode involved. Two species known to be important pathogens *E. granulosus* and *E. multilocularis*[2,3,4]. The most common species is *E. granulosus*, which causes cystic hydatid disease (CHD). Much less common is *E multilocularis* which produces alveolar hydatid disease (AHD) and also an invasive tumour-like replacement of liver tissue. The primary host for the Echinococcus parasite is the dog and other canines. Human, sheep and cattle are the intermediate hosts in which the encysted form of the parasite occurs. Dogs are infected by eating the infected viscera of herbivores. The sheep appears to be the optimum intermediate host for *E granulosus*. Humans are infected by eating food contaminated by the ova passed in the stool of the definitive host. Inhalation is a possible alternative route for lung hydatidosis[5].

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As hydatid cyst of the orbit has not been reported so far in Bangladesh, it is felt worthwhile to report this case of hydatid cyst affecting orbit.

Case Report:

A 30-year-old farmer was admitted in National Institute of Ophthalmology and Hospital on November 02, 1993 with the history of a swelling in the inner canthus of the right eye for the last two years. The patient noticed a very small swelling initially which progressively increased in size. The patient also developed watering for one and half a years and dimness of vision of the right eye for one year. For the previous one month, he developed right sided headache. He lives in a village, works in the field as a farmer and has the history of close contact with dog, sheep, cattle and goat.

On general examination, he was normotensive, apyrexial but looked slightly pale. Liver, spleen, kidney and regional lymph nodes were not palpable. On local examination of the right eye, a discrete swelling approximately measuring 2 cm in diametre at the inner canthus was noticed. It was cystic, non-tender, mobile, below the medial palpebral ligament and the overlying skin was free. Ocular movements were normal. There was no nystigmus or diplopia. The cornea, anterior chamber, pupil, iris and the intraocular pressure were normal. Funduscopic findings were as follows: Disc: nasal margin was ill defined, veins engorged, c:d were 0.3, maculo-

Fig-1: *Photomicrograph showing pericyst, ectocyst and endocyst with brood capsule and scolices of hypatid cyst.*

foveal reflex was faint. The left eye was without any abnormality. The visual acuity of right eye was 6/24 while that of the left eye 6/6.

Laboratory data were as follows: haemoglobin 10.4gm/100 ml; ESR -30mm in first hour; total count of WBC-9. $8x10^9$/L; differential count, neutrophils -56%, lymphocytes -34%, monocytes -02% and eosinophils-0.8%. Routine stool and urine examination were normal; fasting blood glucose-5.2 mmol/L; blood urea-4.5mmol/L. X-ray Chest was normal. Casoni's test, complement fixation test and precipitation test were not done because hydatid cyst was not suspected due to its rarity. Dermoid cyst was the provisional diagnosis.

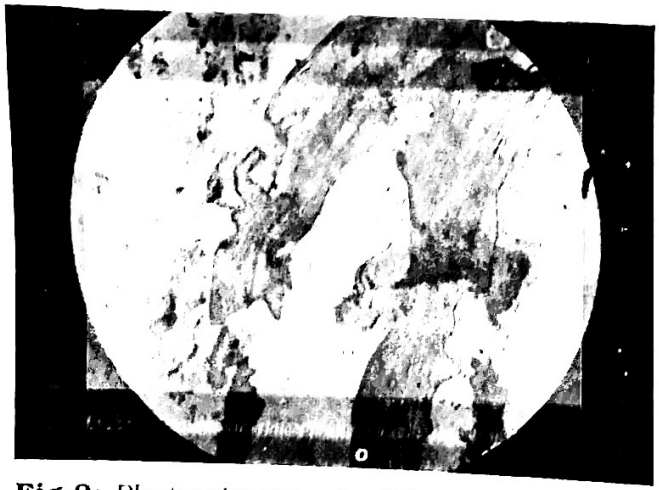

Fig-2: *Photomicrograph of the wall of hydatid cyst showing outer hyaline laminated cuticular layer and inner cellular germinal layer*

The cyst was removed by operation under general anaesthesia. An horizontal incision was given on the lower eye lid, orbicularis muscle was splitted. A solitary cystic mass measuring about 2.2x1.9 cm. was seen. On further dissection, the cyst wall was burste and fluid mixed with tissue debris (hydatid sand) came out. The cyst wall was then removed by careful dissection. Haemostasis was achieved and the wound was closed in layers. The post-operative period was unremarkable. The dissected cyst wall was sent for histopathological examination.

Discussion:

About two third of human *E. granulosus* are found in the liver, 5-10% in the lungs and the rest in bones, brain or other organs[2]. Echinococcosis of the orbit is a very rare condition. Only two cases have been reported so far by Roy et al in 1967 from India[1]. The symptoms of the disease depends upon the site and the rate of the growth of the cyst. Eosinophilia is present in less than 25% of cases[3]. We have got mild eosinophila. Due to our biasness in diagnosis of dermoid cyst, more specific tests for hydatid disease were not done. The specific diagnosis of the cystic swelling was accomplished by histopathological examination[6].

As there is no effective medical treatment, surgical removal is the choice for larger cysts[4]. In some cases, mebendazole in the dose of 400 to 600 mg three times a day for 21 to 30 days has been found to be useful[7,8]. It limits glucose uptake by the parasite leading to glycogen depletion and death[7]. But the efficacy of high dose of mebendazole is still under trial[8,9,10]. Subjective improvement has been reported in upto 70% patients[11]. In our case, mebendazole was used postoperatively to prevent recurrence[6,12,13].

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Tropical Splenomegaly Syndrome: A Case Report

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Summary:

An elderly farmer from Chittagong presented with recurrent fever and progressively enlarging abdominal mass. He was anaemic and had massive splenohepatomegaly without any other systemic manifestation. His bone marrow was hyperplastic and serum IgM level was very high. A diagnosis of tropical splenomegaly syndrome was made. After one month of antimalarial therapy, his IgM level fell back to normal with regression of splenohepatomegaly and correction of anaemia, thereby confirming the diagnosis.

(J Bangladesh Coll Phys Surg 1995; 13: 34-36)

Introduction:

In tropical Africa, New Guinea, Brazil, and parts of Indian subcontinent and Southeast Asia, many patients with otherwise unexplained large splenomegaly fulfill the criteria of the tropical splenomegaly syndrome (TSS)-a disorder characterized by hepatic sinusoidal infiltration, high malaria-antibody titres and very high levels of serum IgM[1]. TSS is thought to be an abnormal immune response to persistent malarial antigenic stimulation in endemic regions[2,3]. Bangladesh is an endemic zone of malaria, but review of literature shows scanty discussion on this intriguing disorder. Recently we have come across a case of TSS, which we are reporting here.

Case Report:

S. M., a 55 years old farmer from Chittagong was admitted in the medical unit V of the Dhaka Medical College Hospital on 21 August, 1993 with the complaints of a progressively enlarging mass in the left upper abdomen, with associated recurrent fever for the last one and a half year. During this period, he suffered about eight bouts of sudden-onset high fever associated with characteristic chills and rigor. Chittagong being a malaria-prone area, each time the patient took some chloroquine or quinine tablets for fever, though never completing the whole course. He became gradually weak and anorexic. He had been seen by qualified physicians and was once treated with parenteral anti-kala-azar medication for two weeks without recourse to any specific investigation. All these, however, failed to improve his condition, and the left hypochondriac mass continued to enlarge further. The patient did not, however, have any history of change in skin colour or bleeding from any site.

On examination, the patient was found moderately anaemic, non-icteric and without any lymphadenopathy. There was no bleeding manifestation and skin colour and hair distribution were normal. The abdomen was asymmetrically distended. Spleen was enormously enlarged (Fig-1). It was 17 cm from the left costal margin along the axis of 10th, well beyond the umbilicus, with rounded border and a well-defined notch. It was firm, non-tender and smooth-surfaced. Liver was palpable 7.5 cm in right midclavicular line (RMCL) from the costal margin. It was firm, smooth-surfaced, non-tender and had no bruit on auscultation. There was no ascites.

Laboratory investigations revealed a haemoglobin level of 6.9 gm/dl and ESR 150 mm in first hour. Total count of WBC was

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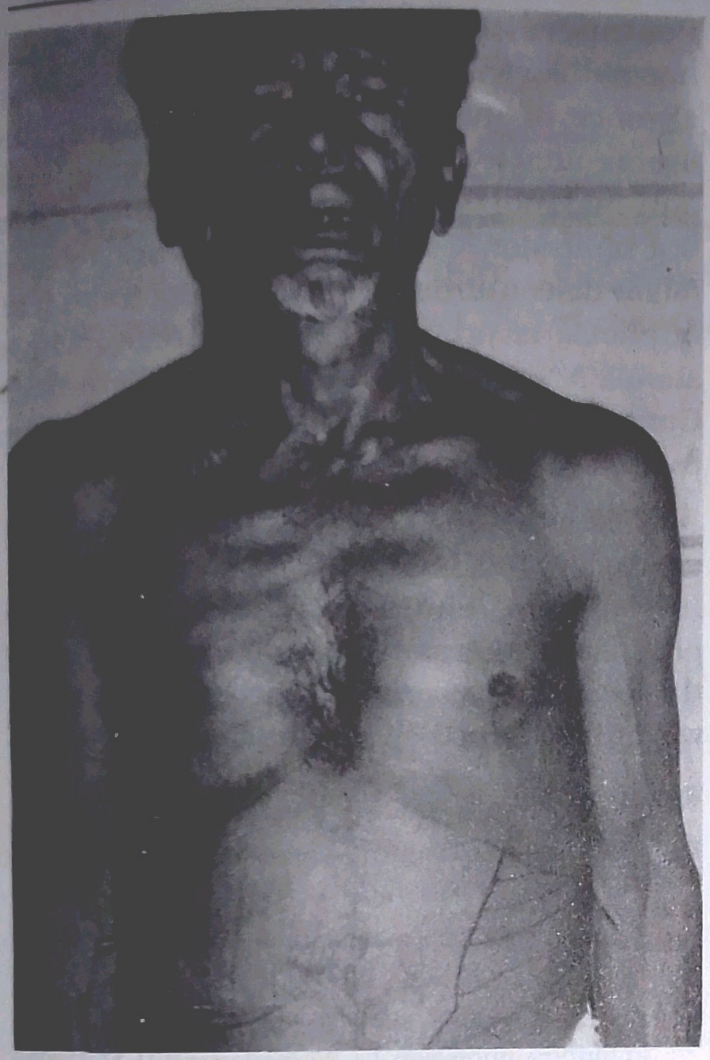

Fig–1: *Showing huge hepatosplenomegaly*

8,000/cumm with 48% neutrophils, 48% lymphocytes and 4% monocytes. Platelet-count was 165,000/cumm. Malaria parasite was absent in both thick and thin smears. Blood film showed anisochromia with anisocytosis of RBC, but normal WBC and platelet distribution. Bone marrow examination showed a hypercellular marrow with reduced M/E ratio. Erythropoiesis was hyperactive and normoblastic. Granulopoiesis was also hyperactive but normal. Megakaryocytes were normal too. There was a mild increase in plasma cells and reactive histiocytes, but no parasites or abnormal cells were seen.

Direct Coombs' test and CFT for kala-azar were negative. Liver function tests were normal but serum IgM study revealed a very high level of IgM, 483 mg% (normal 60-250 mg%) on the eighth day of admission.

The patient was diagnosed as a case of TSS and started on proguanil 100 mg with folic acid 5 mg daily. Follow-up examination a month later showed a marked improvement with only mild anaemia, a greatly reduced spleen (10.5 cm from the left costal margin) and liver (3 cm in RMCL). Serum IgM level after four weeks was nearly half the previous level, 243 mg%. The previous treatment was continued and the patient was advised for regular follow-up.

Discussion:

The diagnostic criteria for tropical splenomegaly syndrome (TSS) is less precise. Fakunle[2] considered serum IgM levels of more than two standard deviation above the local mean as a major diagnostic criteria in TSS.

Reval et al[4] diagnosed TSS on the basis of following criteria:

a. Chronic massive splenomegaly, i. e. splenomegaly of grade 4 or 5 of Hackett's classification, for which no other cause could be found;

b. Elevated serum IgG levels higher than in recurrent or chronic malaria;

c. Elevated serum IgM levels at least 2SD above the local mean (>1000 IU/ml); and

d. A good clinical response to anti-malarial drug therapy with regression of splenomegaly, elevation of haemoglobin level and decreasing serum IgM levels.

Hackett's grade-4 refers to an enlarged spleen with the lowest palpable edge below the umbilical level but not projected beyond a point situated halfway between the umbilicus and the symphysis pubis, while grade-5 refers to a spleen with lowest point palpable beyond the lower limit of iliac crest[4].

TSS is rarely diagnosed before the age of eight years[1]. Patients with TSS present with abdominal mass with history of recurrent fever. It is more common in females and in certain racial and family groups[5]. The condition differs from simple malarial splenomegaly by rarity of parasitaemia and absence of malaria pigment in the macrophages of the liver or spleen[1]. Hepatomegaly is invariable, but liver

function tests are usually normal. Jaundice and ascites are usually absent, though portal hypertension may develop[1-6].

There is an enormous overproduction of IgM with levels reaching three to 10 or 20 times the local mean value[5,7]. Patients have high malaria-antibody titres, most of which are in IgM fraction[1]. IgM aggregates are phagocytosed by reticuloendothelial cells in the spleen and liver and can be demonstrated by immunofluorescence in a liver biopsy section[5]. The hepatic sinusoidal infiltrate seen by light-microscopy consists of macrophages and activated lymphocytes with only scanty plasma cells[1].

In most patients, the haemoglobin is between 5-10 gm/dl despite a normal or raised red cell mass; the bone marrow is typically hyperplastic. Anaemia is principally due to a large splenic red cell pool and haemodilution in an expanded plasma volume[1]. Red cell life span is moderately reduced, but may fall drastically during pregnancy. Conventional antiglobulin tests are negative, but there is an increased frequency of direct Coombs' test in untreated TSS patients[1].

Our patient had moderate anaemia and hepatomegaly with Hackett's grade-4 splenomegaly. Malaria parasites were not found, liver function tests were normal and bone marrow was hyperplastic. We could not compare his IgM level with local mean value, as the latter is not known. However, his IgM level was obviously very high, but with anti-malarial treatment the level rapidly fell back to normal with regression of hepatosplenomegaly and correction of anaemia.

TSS has a substantial morbidity and mortality. In 75 patients in New Guinea observed for 72 months, the morbidity was between 29% and 57%, depending upon the spleen size[8]. Splenectomy may sometimes be necessary because of pain or hypersplenism[1,9]. Postoperatively IgM levels fall and haematological parameters improve, but in absence of malaria chemosuppression, the liver is often further enlarged[1]. With long-term anti-malarial therapy, prognosis is favourable but remission of symptoms is slow and relapse follow discontinuation. The present treatment of choice is proguanil 100 mg daily, which should be continued life long to prevent relapse. Complicating folate deficiency should be treated with folic acid 5 mg daily[5].

TSS is an intriguing condition which, if left untreated, has high morbidity and mortality. In malaria endemic Bangladesh, the physicians, therefore, should always think of this condition when they encounter a patient with unexplained enormous splenomegaly.

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