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Juvenile Rheumatic Mitral Stenosis in Bangladesh

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Summary :

245 patients younger than 20 years with isolated (pure) mitral stenosis (Juvenile Mitral Stenosis) were studied from July 1984 to June 1987. Age range of these patients were 6 to 20 years with a mean of 14.8 ± 3.05 years and a male to female ratio of 1.07:1. Seventeen patients (7%) were asymptamatic at the time of evaluation but majority (93%) of the patients had functional disability of NYHA class III or IV. Atrial fibrillation occurs only in 2% of the patients and 3% had systemic embolism. Forty four percent of the patients had associated congestive heart failure when first seen. One patient (a 16 years old boy) had Jaccoud's arthritis.

Echocardiographic examination of these patients revealed severe rheumatic changes with thinckening of the rough or appositional zone of the leaflets, but the clear zone (body of the leaflets) were relatively spared and remained pliable with frequent diastolic doming and systolic bulging (buckling). Mitral valve opening were significantly narrowed in most of the patients and 68% of them had their mitral valve orifice area of less than 1 cm^2. Thickening of chordae with mild to moderate subvalvular lesion was frequently (88%) encountered but deformity of subvalvular apparatus with shortening and fusion was severe in only 12% of patients. Calcification of the valve apparatus was observed in 2% of patients. Significant pulmonary hypertension was detected in 78% of the patients. Peak systolic pulmonary arterial pressure ranges from 30 to 135 mmHg with a mean of 68.4 ± 22.3-mmHg in 37 patients who underwent cardiac catheterisation. In 7 cases pulmonary arterial systolic pressure exceeded the systemic pressure. Histological examination of the excised left atrial appendage revealed characteristic Aschoff bodies in 20 patients (80%) out 25, and were seen mostly in the subendocardial region. Ninety percent of them were in the proliferative stage, 10% in healed stage and none were in the exudative stage.

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Introduction :

Rheumatic fever (RF) and reheumatic heart disease (RHD) are still widely prevalent and responsible for significant morbidity and mortality in developing countries of the world including Bangladesh. In these countries RF affects younger groups of patients in a much more severe form and the rapid progression of the disease process produces severe valvular lesions and causes marked cardio-pulmonary impairement. Mitral valve stenosisis the commonest valvular lesion in patients with chronic rheumatic heart disease and about one third of all cases of pure mitral stenosis (MS) were below the age of 20 years as reported in different developing countries of the world. Roy et al[1] introduced the term 'Juvenile Mitral Stenosis'(JMS) for such patients of age below 20 years with isolated MS. A very high incidence of JMS has been reported more or less uniformly by several authors from India. Valvular stenosis is often severe in these patients and surgical intervention become mandatory for majority of them. The reasons for the accelerated progession of stenosis of the mitral valve (and possibly of other rheumatic cardiac lesion) in developing countries are not clear. Available evidence incrimiates poor socio-economic status, unhygienic living conditions, undernouishment and virulent streptococcal infections. Certain peculiarities of JMS have been pointed out in the reports from India and other developing countries[2].

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The aim of the present communication is to present the clinical profile of JMS in Bangladesh.

Patients and Methods :

1427 patients with reheumatic heart disease were selected for the study from the outpatient clinic and inpatient wards of the National Institute of Cardiovacular Diseases, Dhaka, from July 1984 to June 1987. This institute is the central referral centre for cardiovascular diseases. The patients studies came from all parts of the country and have variable social and economic standards. Detail medical history of these patients were recorded and physical examination performed. All the patients had routine haematological and serological tests, urinalysis, chest X-ray and ECG. Two-dimensional echocardiographic examination was performed utilising standard methods. Mobility of the leaflets were studied in parasternal long axis view. In normal persons the anterior mitral leaflet (AML) moved freely and its tip appoximated the interventricular septum during diastole. In the study group with MS the tip of the leaflet demonstrated restricted motion due to fusion of the commissures, producing a convex appearance (doming) towards the interventricular septum during diastole. This doming was considered severe if the convexity of the leaflet was striking and collided with the interventricular septum in diastole, and was considered mild to moderate if the convexity of the AML did not approximate the septum[3] (Fig.1). End-systolic frames were analysed for the presence of systolic bulging of AML (when the body of AML buckles beyond the mitral ring towards the left artium at end systole) (Fig.2). The anterior mitral leaflet was considered pliable when it forms diastolic doming and systolic bulging[3]. Subvalvular changes were examined in the parasternal long axis view

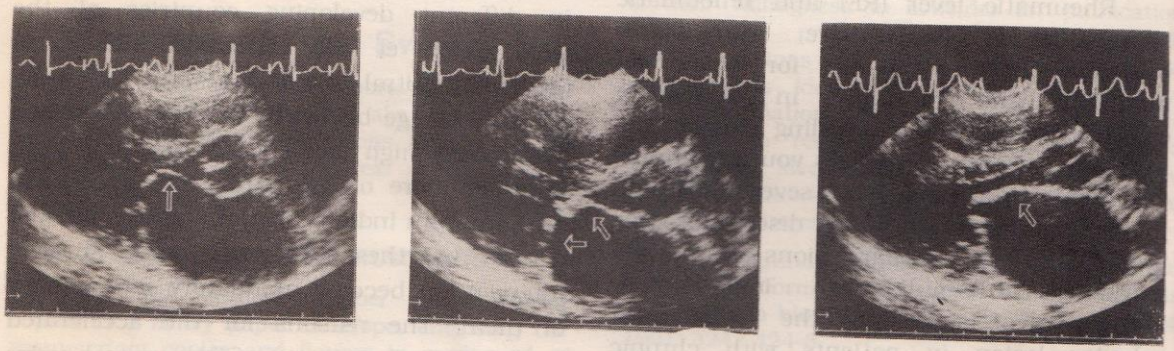

Fig 1 : *Long axis two-dimensional echocardiograms of the left ventricle in diastole showing the mitral valve and subvalvular apparatus from three patients with varying degree of dome formation of AML and severity of subvalvular lesions.* ***Left panel :*** *Little thickening of the leaflets with severe dome formation of AML demonstrating its pliability. The subvalvular apparatus is not significantly diseased.* ***Middle panel :*** *The mitral leaflets are thickened to a grater extent with moderate dome formation of AML indicating some rigidity. The chordae tendineae are thickened as well.* ***Right panel :*** *The leaflets are markedly thickened and rigid with minimal dome formation of AML. The submitral apparatus is also severely involved, with marked thickening and fusion of chordae.*

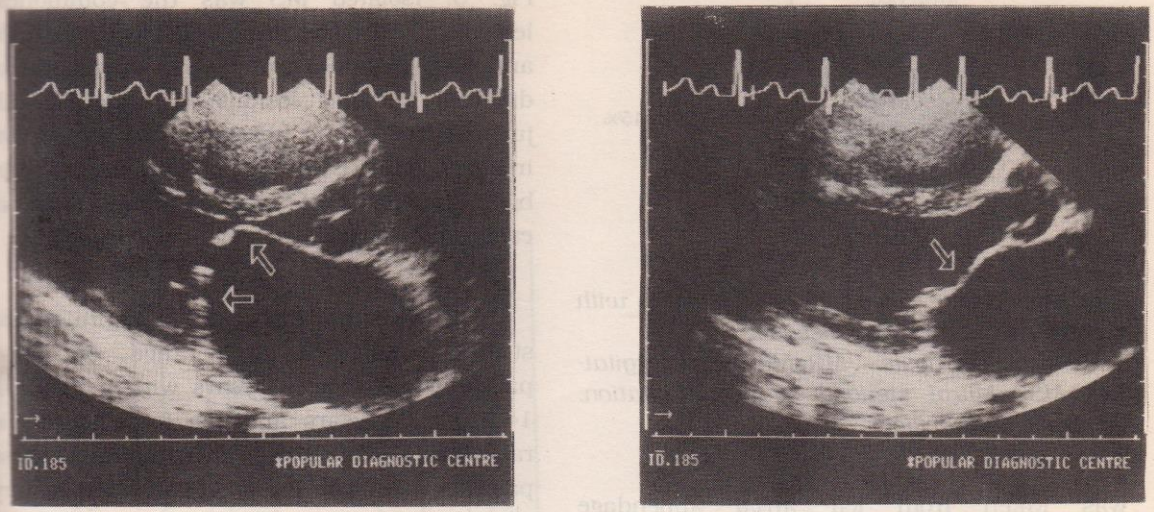

Fig. 2 : *Long-axis two-dimensional echocardiograms in diastole (Left) and in systole (Right) from a patient with mitral stenosis. The diastolic frame shows severe doming of the AML while the systolic frame shows bulging (buckling) of the leaflet demonstrating its pliability.*

and the severity of reheumatic deformity were graded as mild, moderate and severe. In mild cases the chordae of the anterior and posterior leaflets remains seperate and the echo of the chordae was thin. In severe cases, chordae thickened markedly with fusion of the leaflets, chordae and papillary muscles. Sometimes calcfication of the leaflets and /or the chordae was observed. In moderate cases, the subvalvular changes are intermediate between the two (Fig. 1). This morphological gradation may be censidered identical with the three anatomical subsets of mitral stenosis described by Sellors et. al[4]. The mitral valve area was obtained by planimetry outlining the black and white interface of the mitral orfice at the onset of diastole[5] (Fig.3). Thirty seven patients of the series underwent cardiac catheterisation. Biopsy

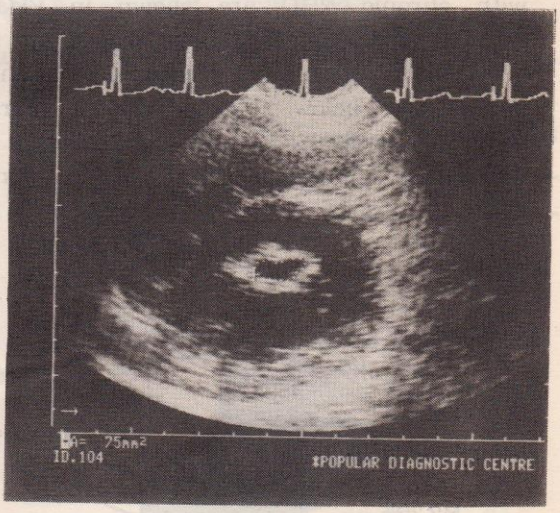

Fig. 3: *Parasternal short-axis view through the tips of the mitral leaflets demonstrating the valve orifice. Planimetry of the mitral valve orifice area was performed at the onset of diastole.*

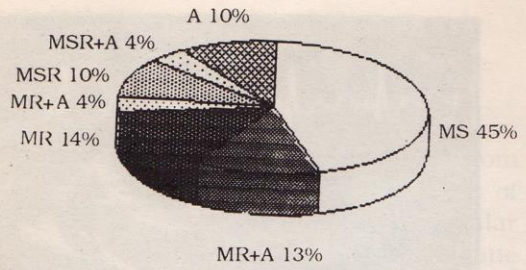

Fig. 4: *Valvular lesions in 1427 patients with chronic rheumatic heart disease. MS=Mitral stenosis, MR=Mitral regurgitation, MSR=Mitral stenosis and regurgitation. A= Aortic valve lesion.*

was taken from left atrial appendage during surgery for microscopic examination from 25 patients.

Results :

Types of valvular lesion in 1427 patients with chronic RHD are shown in Fig.4. Isolated (pure) mitral stenosis was found in 597 (45%) patients; of them 245 patients (38%) were below the age of 20 years- Juvennile Mitral Stenosis. Incidence of different valvular lesions in juvenile and adult groups of patients are campared in Fig. 5. Isolated MS was the commonest lesion in both the groups (41% in juvenile and 48% in adult group). Sex prevalence of different valvular lesions in patients with juvenile rheumatic heart disease are shown in Fig. 6. Isolate MS was equally prevalent in both the sexes of these patients; 41% of each sex had MS.

245 patients with JMS constitutes the study population. Age range of these patients was 6 to 20 years with a mean of 14.8 ± 3.05 years and the male to female ratio was 1.07 to 1. Age distribution of these patients is shown Table-I. A previous history of RF was obtained from 160 (65%) patients and 86 (35%) patients had recurrences. Interval of between the first attack of RF and the onset of symptomatic MS was I to 8 years, mean 2.6 ±1.8 years.

Clinical feature of the patients with JMS are shown in Table-II. Ninety percent of the patients had dyspnoea of different severity. Palpitation was the next common symptom (63%). Congestive heart failure was observed in 44% of patiens and only 2% had atrial fibrillation. Seven patients (3%) had

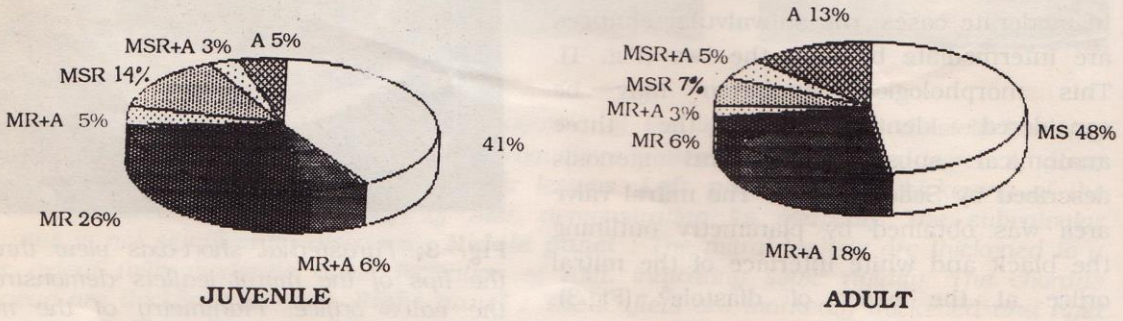

Fig. 5 : *Incidence of rheumatic valvular lesions in Juvenile and Adult patients.*

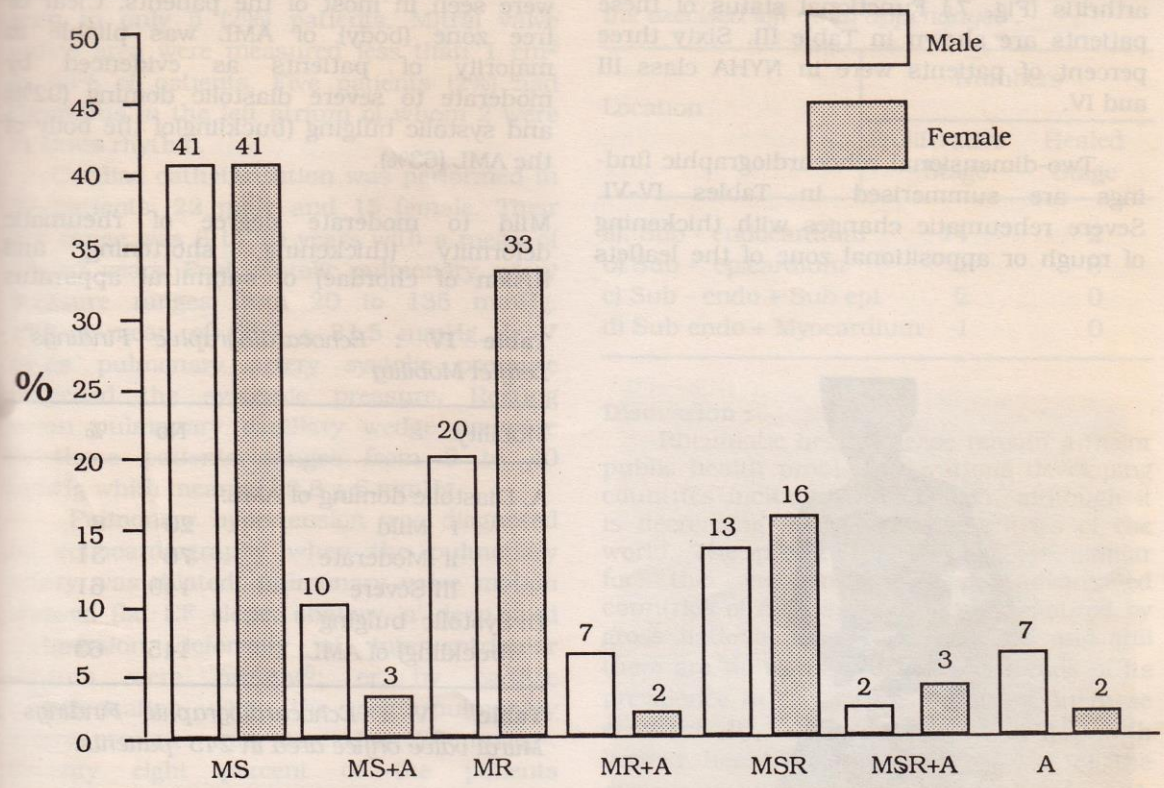

Fig. 6 : *Sex prevalence in patients with Juvenile rheumatic heart disease.*

Table I : *Age distribution of patients with JMS*

Age (Yrs)	Male	Female	Total No (%)
6-10	6	4	10 (4)
11-15	48	47	95 (39)
16-20	73	67	140(57)
Total	127	118	245(100)

systemic embolism: 5 had cerebral and 2 had peripheral manifestation. Seventeen (7%) patients were asymptomatic. One patient, a 16 years old boy, had Jaccoud's

Table II : *Clinical Features of 245 Patients with JMS.*

Features	No	%
H/O Rh. Fever	160	65
Dyspnoea	228	93
PND	39	16
Pulm. Oedema	49	20
Cough	103	42
Haemoptysis	54	22
Palpitation	154	63
Embolism	7	3
Chest Pain	43	18
Fever	76	31
CHF	107	44
AF	5	2
Jaccoud's Arthritis	1	
Asymptomatic	17	7

arthritis (Fig. 7.) Functional status of these patients are shown in Table III. Sixty three percent of patients were in NYHA class III and IV.

Two-dimensional echocardiographic findings are summerised in Tables IV-VI. Severe reheumatic changes with thickening of rough or appositional zone of the leaflets were seen in most of the patients. Clear or free zone (body) of AML was pliable in majority of patients as evidenced by moderate to severe diastolic doming (92%) and systolic bulging (buckling)of the body of the AML (63%).

Mild to moderate degree of rheumatic deformity (thickening, shortening and fusion of chordae) of submitral apparatus

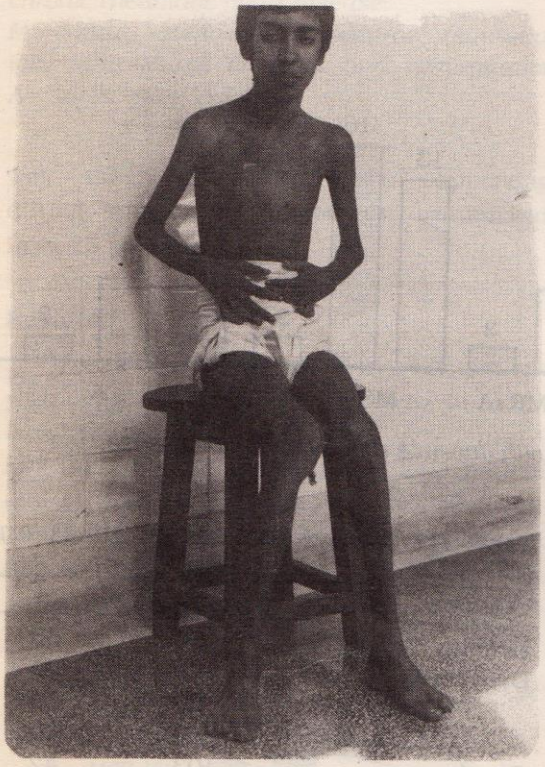

Fig. 7: *The patient with Jaccoud's arthritis.*

Table III : *Functional Status of the Patients*

NYHA Class	No	%
I	17	7
II	74	30
III	115	47
IV	39	60

Table IV : *Echocardiographic Findings : Leaflet Mobility*

Mobility	No	%
A. Diastolic doming of AML:		
i Mild	20	8
ii Moderate	76	31
III Severe	149	61
B. Systolic bulging (buckling) of AML.	145	63

Table V : *Echocardiographic Findings : Mitral valve orifice area in 245 patients.*

MVA (cm^2)	No	%
Less than 0.5	20	8
0.5 ↛1.0	148	60
1.0 ↛1.5	41	17
≥ 1.5	36	15

↛ Denotes upto but not including.

Table VI : *Echocardiographic Findings : Severity of rheumatic deformity of subvalvular apparatus.*

Subvalvular Lesion	No	%
Mild	91	37
Moderate	125	51
severe	29	12

was obeserved in 88% of the patients. Calcification of mitral valve apparatus were seen in only 5 (2%) patients. Mitral valve orifice area were measured less than 1 cm^2 in 68% of patients. Five patients (2%) had thrombus in the left atrium of whom 2 were in sinus rhythm.

Cardiac catheterisation was performed in 37 patients, 22 male and 15 female. Their age range was 8 to 20 years with a mean of 13±3.7 years. Peak systolic pulmonary artery pressure ranges from 20 to 135 mmHg. with a mean of 68.4 ± 21.5 mmHg. In 7 cases pulmonary artery systolic pressure exceeded the systemic pressure. Resting mean pulmonary capillary wedge pressure in these patients ranges from 8 to 40 mmHg whith mean of 23.8 ± 6 mmHg.

Pulmonary hypertension was diagnosed by echocardiography when the pulmonary artery was dilated; pulmonary valve motion showed flat EF slope, shallow 'a' deep. and compersion deformity of interventricular septum were present[6]; or by cardiac catheterisation, when the mean pulmonary artery pressure was more than 20 mmHg[1]. Seventy eight percent of the patients showed such evidence of pulmonary hypertension.

Twenty (80%) of the 25 patients showed Aschoff bodies in the excised left atrial appendage (Fig.8), mostly found in the subendocardial region (Table-VII).

Fig. 8: *Subendocardial Aschoff body in the atrial appendage excised during mitral commissurotomy(X 160).*

Table VII: *Locations of the Aschoff bodies in the execised left atrial appendages .*

Location	Numbers	
	Proliferative Stage	Healed Stage
a). Sub - endocardium	14	2
b) Sub - epicardium	1	0
c) Sub - endo + Sub epi	2	0
d) Sub endo + Myocardium	1	0

Discussion :

Rheumatic heart disease remain a major public health problem in various developing countries including Bangladesh, although it is decreasing in developed countries of the world. The prevalence of RHD are similar for the economically less advantaged countries of Asia and Africa as measured by gross national product per caput[7], and still there are no signs of downward trends of its prevalence in these poor countries. In these economically underprivileged areas with limited health facilities the course of the disease is often malignant and accelerated, so that well established mitral valve disease occurs in childhood[8]. High incidence of RHD below 20 years of age has been reported from developing countries[9-12]. Forty two percent of patients in our series were below the age of 20 years. The pattern of valvular lesions in the juvenile group is somewhat different than that of the adult patients. Incidence of MR and MSR were more in the juvenile group while incidence of isolated aortic valve lesion was relatively more in the adult group.

In the western world mitral stenosis (MS) takes many years to develop and is mostly a disease of adult life. This is in striking contrast to the developing countries where MS can develop rapidly and therefore often affects adolescents and

even children as young as five years of age[1]. It appears puzzling that even when rheumatic fever was common in the west, only a tiny minority of the patients undergoing commissurotomy were in their teens or younger, whereas in developing countries as many as one-third of all mitral commissurotomies are performed in patients under 20 years of age[11-16]. Established MS was being observed in the young from the time RHD had been recongnised as an important cause of cardiovascular disorders in India[17]; but the term 'Juvenile Mitral Stenosis' was first introduced by Roy et al[1] in 1963 for patients with MS under 20 years of age. Juvenile mitral stenosis is of special interest because of its high prevalence reported from developing countries. Symptomatic JMS constitutes 25 to42%of all cases of isolated mitral stenosis in India in both clinical[18-20] and surgical series[14-16].

Thirty eight percent of our patients with isolated mitral stenosis were under 20 years of age (JMS) and they constitutes 17% of the total patients with RHD.

Sex distribution of the patients with JMS was almost equal with a male to female ratio of 1.07 : 1. Roy et al[1] reported male to female ratio of 1.6 : 1 among patients with JMS. Similar male predominance was reported by Mathotra and Gupta[21]. However, others from India and other developing countries found sex distribution to be near equal as in our series [9,11,22].

History of rheumatic fever among patients with JMS was reported to be 49% by Manabe[23], 66% with recurrence in 28% by Roy et. al[1], and 53% with recurrence in 12% by Cherian et, al[14]. Though the incidence of RF (65%) was similar, frequency of recurrences (35%) was higher in our patients with JMS.

Except for a minorty of patients, all cases of JMS came under observation after they have developed symptoms. Seventeen patients (7%) in our series were asymptomatic.

The process of fusion of cusps and valvular stenosis develops early and progress rapidly in developing countries to become clinically manifest in childhood and adolescence. The latent period between the initial attack of RF and onset of symptoms is much shorter in these countries in comparison to that in the western countries[17]. In our patients the interval between the first attack of RF and development of clinical MS was 1 to 8 years with a mean of 2.6 $\pm$ 1.8 years. This time interval was two years or less in 53% of our patients with JMS. In developing countries this latent period can be as short as one year[24], and in some cases even six months[25]. On the other hand in the west it usually takes 20 to 30 years for the mitral valve to become stenotic enough to produce a significant haemodynamic alteration[26].

Clinical features of JMS in our country do not differ to those met in the adults except that atrial fibrillation and systemic embolism are less frequently observed (2% and 3% respectively).

Jaccoud's arthritis, so called chronic post-rheumatic fever arthropathy, is a rare, indolent, slowly progressive process that deforms the fingers and sometimes the toes[27]. The deformity consists of ulnar deviation of the fingers, flexon of the metacarpophalangeal joints and hyper extension of the proximal interphalangeal joints; just as it occurs in rheumatoid arthritis. In classic cases Jaccoud's athropathy appears after multiple, prolonged, and severe attacks of RF. It is thought to be the end result of the repeated inflammation of the fibrous articular capsules in the small joints of the hands, perhaps depending on

individual predisposition[28]. We have encountered one such patients with Jaccoud's arthropathy in our series.

The initial insult of reheumatic carditis involves the valve leaflets. In the early phases of rheumatic valvular involvements the most conspicuous lesion consists of minute translucent nodules (verrucae) located along the lines of closure or contact of the leaflets[29]. In recurrent reheumatic valvulitis the gross changes observed during the acute phase are accentuated. The valve surfaces become thinkened and irregular. The thickening is most severe in the distal third of the valve leaflets. This thickening of the mitral valve may extend to involve insersions of the chordae tendenea, which become thickened and shortened, thus bringing the popillary muscles closure to the leaflet margins. Adjacent thickened chordae tendineae may also fuse. When mitral stenosis is sypmtomatic, the anatomic features consist of thickened mitral leaflets with or without calcific deposits, fusion of the valve commissures, and shortening and fusion of the chordae tendinea[30]. The various anatomic findings described above can be imaged adequately by two-dimensional echocardiography. The thickening of the mitral leaflet in rheumatic heart disease appears to spread from leaflet tips (rough zone) to base and appears to involve the annulus only when the entire leaflet is thickened[3]. In majority of our patients (81%) rheumatic changes of the leaflets were severe with thickening which predominantly confined in the rough zone while the clear zone (body of the leaflets) was relatively spared. Moderate to severe diastolic doming and systolic bulging (buckling) of the body of AML are demonstrated with two-dimensional echocardiography when the leaflets are pliable[3]. As the AML becomes more fibrotic or calcific, diastolic doming is reduced and the systolic bulging or buckling motion is less apperent or absent. Most of our juvenile patients had their valve leaflets pliable as evidenced by higher incidence of moderate to severe diastolic doming (92%) and systolic bulging (63%) of the AML. Severe degree of subvalvular lesions were less commonly seen in our patients with JMS. Echocardiographic evidence of valve calcification and left atrial thrombus were rarely noted.

Natural history of rheumatic stenosis progresses faster in the developing countries than in the west[14]. The ravages of the disease are reflected in our patients by severe deterioration of their functional capacity (NYHA class III or IV) in 63% of patients and by the severity of mitral valvular stenosis (68%) had mitral valve orifice area of less than 1 cm^2. Thrombus in the left atrium was infrequently encountered (2%)in these patients.

The haemodynamic data of our patients with JMS was markedly abnormal. Significant pulmonary hypertension was found in 78% of them. Similar high incidence of pulmonary hypertension has also been reported by authors from India[1,16,18] and they ascribed the cause of pulmonary hypertension to a hypersensitive reaction to the pulmonary vasculature, a fulminating rheumatic process or to a tissue response to multiple overt attack of RF. Incidence of congestive heart failure is reported to vary in direct proportion to the severety of pulmonary hypertension[2]. Our Data also speak in support of this view where 44% of patients had congestive heart failure.

Aschoff lesions are manifestations of tissue reaction to injury, and convincing evidence has been presented over the years that they are specific for RF. With the

advent of surgery, atrial appendages have been examined and in chronic RHD, Aschoff nodules, indicating an active rheumatic process, have been found with varying frequency in the endocardium (37 to 75%) depending on the criteria used for diagnosis[31]. Virmani and Roberts[32] reviewed the cases of 1,740 patients of 18 reports in the literature. In these reports the percentage of patients with Aschoff bodies ranged from 19% to 74% with a mean of 38%. The presence of Aschoff bodies has been considered to indicate activity. Aschoff bodies in the excessed left atrial appendages were found in 80% of our patients with JMS most of which were in the endocardium. Ninety percent of them were in the proliferative stage and 10% in healed stage.

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Total Intravenous Anaesthesia; Experience with Ketamine-Diazepam Combination

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Summary

Total Intrasvenous Anaesthesia (TIVA) with ketamine has been used in different centers around the world. Many of the drugs used in conjunction with ketamine in those studies, including some of the equipment are not available in Bangladesh. Our present study was conducted on 40 patients with ketamine and diazepam in infusion, supplemented with a single pre induction intravenous bolus dose of morphine. Muscular relaxation was provided with pancuronium bromide and supplemented later as and when deemed necessary. Artificial ventilation was maintained with oxygen enriched room air. Cardiovascular variables and metabolic effects as reflected by change in blood glucose level and recovery characteristics were monitored.

Results were compared with a similar group of patients under a balanced general anaesthetic. No significant differences in cardiovascular parameters were observed while it was found that TIVA with ketamine produced a significant degree of hyperglyscaemia (P < 0.001). Time reuired for the return of cough reflex and opening the eyes were shorter after ketamine infusion (p<0.001). Wake-up time was similar in both the groups (p>.5). In terms of cost involvement TIVA was reasonable less expensive than balanced general anaesthesia.

(J Bangladesh Coll Phys Surg 1991; 9: 46-52)

Introduction :

General Anaesthesia when administered in a 'balanced 'technique involves expensive apparatus, and volatile anaesthetic agents. Commonly used nitrous oxide is now claimed to impair folate metabolism[1] while halothane has been incriminated definitively for causing liver damage[2]. Procurement of both continuous flow anaesthetic machines and nitrous oxide requires huge some of money. On the other hand transport strike is almost a regular phenomenon these days, hindering smooth supply of gases. Although infrequent, natural calamity, remains a constant threat for the future, to affect regular suply of anaesthetic gases. These are only few of our limiting factors in delivering anaesthetic and thus surgical services at the peripheral hospitals of our country. Total intravenous anaesthesia on the other hand has certain advantages over balanced general anaesthesia, such as, avoidance of inhalational anaesthetics and expensive apparatus.

With the existing resources, the need to offer safe as well as round the year anaesthetic service at Mymensingh Medical College Hospital, has prompted us to try 'Ketamine' in the form of total intravenous anaesthesia. Since the agents best suited with Katamine, for intravenous anaesthesia, are not available in our country, we have tried this in combination with Diazepam in the same infusate.

With this technique, our aim was to assess the Cardiovascular function, changes in blood glucose level, post anaesthetic recovery pattern and finally cost involvement. We compared all these parameters with those of a similar group of patients provided with a balanced general anaesthetic (BGA).

Materials :

A total of 76 patients of ASA physical status I, between 20 to 50 years of age and destined to undergo elective cholecystectomy, were randomly allocated into two

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groups, to receive either Ketamine induced anaesthesia (Group 1) or a balanced general anaesthetic (Group 2).

Methods:

After being randomly allocated, patients in Group 1 received Total intravenous Anaesthesea (TIVA) with Ketamine, while in Group 2, balanced general anaesthetic was administered. No premedication was used in any of the patients in either of the groups. The two anaesthetic regimens we used is shown in Table I.

Table -I : *Methods of Anaesthesia in the Two Group*

	GROUP 1	GROUP 2
1. Prior to induction	Morphine sulfate 0.15 mg.kg^{-1},i.v	Morphine sulfate,0.15 mg.kg.$^{-1}$ i.v.
2. Intravenous fluid	Two intravenous channels One for Ketamine-Diazepam infusion & other for fluid balance.	Single channel only For normal fluid balance normal the
3. Drug in infusion	500 mg of Ketamine and 10 mg of Diazepam mixed in 500 ml of isotonic saline.	Nil
4. METHOD OF DRUG INFUSION	Initially at a rate of 70-80 drops. min^{-1} for 3 minutes then reduced to 40-45 drops. min^{-1} till 2-3 minutes before the reversal. This of infusion delivered Ketamine 40-50 mic.gm. kg^{-1}. min^{-1}. and Diazepam 0.8-1.0 mic.gm.kg^{-1}. min^{-1}.	Not applicable
5. Sleep induction	Thiopentone sodium, 5 mg.kg^{-1} with Atropine 0.02 mg kg^{-1}	Thiopentone sodium, 5 mg.kg^{-1} with Atropine 0.02 mg.kg^{-1}
6. Muscular relaxation	Pancuronium bromide, 0.1 mg kg^{-1} and later supplemented if clinically necessary (1/3rd of the inital dose).	Pancuronium bromide, 0.1 mg.kg^{-1} and later supplemented if clinically necessary (1/3rd of the initial dose).
7. Ventilation	With a self inflating bag fitted with a non-return valve, using Oxygen (2L. min^{-1}) enriched room air.	Through a Mapleson D circuit, with a fresh gas flow of 125 ml. kg^{-1} min^{-1} (70% N 20 in 0.2L) supplemented with Halothane 0.25-0.5 vol % Halothane was put off 2-3 minutes before reversal.
8 Reversal of neuromuscular block	Neostigmine 2.5 mg with Atropine 1.2 mg.	Neostigmine 2.5 mg with Atropine 1.2 mg.

Most of the parameters were same in both the groups. Only important differences were drug infusion and the nature of the ventilating gas.

Monitoring :

Pulse rate was counted manually and blood preessure were measured with an aneroid sphygmomanometer and stethoscope over the brachial artery. Recordings were made preoperatively (PO), immediately before the induction of anaesthesia (T_1) and then at 10 minutes interval (T_{2-11}). Finally two more recordings were made, 15 and 30 minutes after the reversal of anaesthetic (R_{15} & R_{30}).

Blood glucose was measured thrice during the whole procedure by utilising Glucose oxidase method using a Reflolux II(R). The first being made immediately before starting any infusion (PO), the second at 30 minutes after incision (I_{30}) and finally 30 minutes after the end of anaesthetic (R_{30}).

Later during the immediate recovery period 'time taken for the establishment of cough reflex' (time since reversal for the patient to cough on command, in seconds), 'Eye opening time' (time taken since reversal in minutes, to open eyes on command) and 'wake up time' (time taken to tell own's name on command since reversal, in minutes) were noted. Twenty four hours after the anaesthetic, patients were interviewed for any 'incidence of dreams or awareness' under anaesthesia.

Statistical Analysis: Student's paired 't' test was applied where applicable and a value of $P < 0.05$ was taken as significant.

Results :

Demographic data of all the patients in the two groups are presented in table II.

Table II. *Demographic data of patients in the two groups; mean (SEM)*

Parameters	Group1	Group2
Number of patients	40	36
Age (in years)	36.5 (1.12)	34. 05 (1.41)
Weight (in kg)	47.87 (0.93)	46.28 (0.91)
sex: Male	20 (50.00%)	19 (52.78%)
Female	20 (50.00%)	17 47.22%)
Preoperative BP		
a) Systolic	116.75 (2.38)	122.22 (2.56)
b) Diastolic	72.87 (1.39)	74.03 (1.52)
Pulse rate (bpm)	75.67(1.85)	79.03 (1.41)
Operation time (min)	75.68 (1.84	78.47 (2.00)

Patients in both the groups were similar and fairly comparable. Change in pulse rate as observed in our study is shown in Figure 1 while the trend in mean blood pressure is shown in figure 2.

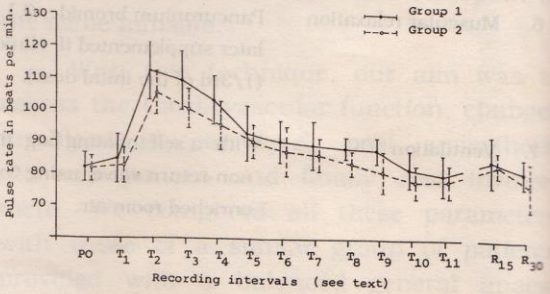

Fig 1 : *Trend in pulse rate*

Reflolux II, a bedside blood glucose estimator of Boeringer Mannheim GmRH, Germany.

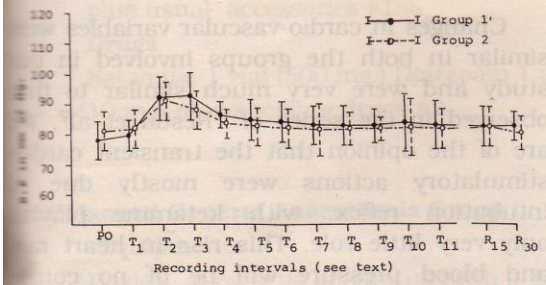

Fig. 2 : *Trend in mean blood pressure (SEM)*

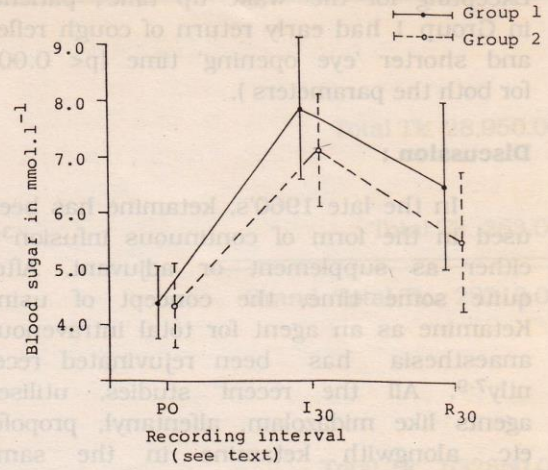

Fig. 3 : *Change in blood Sugar*

It is evident, that both heart rate and blood pressure went up from their basal values ($p<0.001$). This occurred in both the groups. At around 40 minutes after induction, both the parameters in both group of patients came down to non-significant levels ($p > 0.05$).

We have plotted the change in blood glucose levels in figure 3. Although it was raised in both the groups, 30 minutes after the induction patients who received ketamine had higher values ($p<0.005$). Around thirty minutes after the reversal, these values, in both the groups, fell to a much lower level ($p>0.05$), but still was higher than their preoperative figure. At this stage also, patients who received ketamine were more hyperglycemic than their control counterpart ($p<0.05$).

Table III shows the recovery characteristics of the two groups.

Table III. *Mean time for return of recovery of Characteristics (SEM)*

	Group 1	Group 2	Result
Return of Cough Reflex (in seconds)	69.87 (1.53)	88.61 (9.46)	P<0.001
Eye opening time (in minutes)	4.95 (0.11)	7.32 (0.11)	P<0.001
Wake-up time (in minutes) (to tell own's name)	11.39 (0.15)	11.46 (0.18)	P<0.5
Emergence reaction	Nil	Nil	

Excepting for the 'wake up time', patients in Group 1 had early return of cough reflex and shorter 'eye opening' time ($p < 0.001$ for both the parameters).

Discussion :

In the late 1960's, ketamine has been used in the form of continuous infusion[3-6] either as supplement or adjuvant. After quite some time, the concept of using Ketamine as an agent for total intravenous anaesthesia has been rejuvinated recently[7-9]. All the recent studies, utilised agents like midazolam, alfentanyl, propofol etc. alongwith ketamine in the same infusion. White in 1982, has recommended preferentially for Midazolam for use with Ketamine[10]. Unfortunately, this midazolam is not yet available in this country. Exorbitant price of all these drugs, is another disavantage. In contrast, we found in our present series that Diazepam can also be mixed with ketamine without any untoward effect, rather present findings suggest that they act as complementary to each other.

Huge cost involvement required for BGA mostly for procuring Boly's apparatus and nitrous oxide, has restricted its wide use, not only in the district hospitals of our country but also in most of the Medical College Hospitals outside the capital. To delivery cheap as well as safe anaesthetic in these centers, we focused our attention of TIVA with Ketamine. To overcome the disadantages of ketamine mostly dreams and hallucination, we tried the cheapest agent-diazepam, not as a premedicant, but in the same infusion and this we did deliberately, with the intention to cutdown the total dose of diazepam.

Because of the inherent major disadvantages of ketamine, predominantly the emergence reaction, its continuous infusion alone has rather been discouraged by Morgan in 1983[11]. But with our present drug combination, no single case reacted that way, which is in conformity to findings presented in some recent literatures [7-10].

Changes in cardio-vascular variables were similar in both the groups involved in our study and were very much similar to that observed in the series of Restal et al[7]. We are of the opinion that the transient cardio-stimulatory actions were mostly due to intubation reflex, with ketamine playing only very little role. This rise in heart rate and blood pressure will be of no consequence if used in otherwise young healthy adults free from hypertension or cardiac diseases, rather we believe that these may favourably be exploited in patients with hypovolaemic states.

Heperglycaemia after ketamine, as observed in our study, has also been reported by others[12]. However for all practical purpose, hyperglycemia after ketamine as seen in this study, which also occurs to a variable extent after balanced general anaesthesia, may also be ignored unless the patient is a known diabetic.

As regards to recovery characteristics, excepting for the wake up time, other parameters were better in the 'Ketamine-Diazepam' group and were similar to that observed with Ketamine-Midazolam combination[8]. Quick return of cough reflex in ketamine group is one of the positive advantage observed in this study. And this we feel is one of the safety feature after any form of general anaesthesia.

Ketamine is fairly expensive, but when considered along with other drugs as used in this study, we found that, this combination to be reasonably cheaper than that required for 'Balanaced General Anaesthesia'.

Table-IV : *Comparison of cost involvement*

Total Intravenous Anaesthesia group :

Equipment

Self inflating bag, Connecting hose, plus usual accessories #1 — Total Tk. 28,950.00

Drugs

Ketamine 1 vial (500 mg.) Diazepam 1 amp (10 mg) plus ancillary drugs #2 — Total Tk. 263.00

Grand Total Tk. 29213.00

Balanced General Anaesthesia Group :

Equipments

Boyle's anesthetic apparatus (with a 'D' circuit, Nitrous oxide cylinders (2 nos), plus usual accessories #1. — Total Tk. 532850.00

Drugs

Nitrous oxide plus ancillary drugs #2 — Total Tk. 188.00

Grand Total Tk. 533038.00

	TIVA	BGA
Fixed cost	Tk. 28950.00	Tk. 532850.00
Recurring cost	Tk. 263.00	Tk. 188.00
Total	Tk. 29213.00	Tk. 533038.00

#1 Endotracheal tubes (7 nos.: sizes 7.0 through (10.00 mm), Laryngoscope set, Oxygen cylinder with flowmeter, Connectors

#2 Thiopentone sodium (250mg), Atropine 3 amp (o.6mg), Neostigmine 5 amp. (0.5mg), 0.9% Sodium chloride solution (500ml), Morphine (7.5mg), Oxygen

Our total cost involvement with both types of anaesthetic was calculated and presented in Table IV. It is obvious that TIVA with ketamine is fairly cheap. Moreover it has the additional advantage of avoiding the regular maintenance and servicing of anesthetic apparatus whcih is difficult in remote peripheral situations.

Conclusion:

Considering the advantages of "Total Intravenous anaesthesia" using Ketamine and Diazepam in combination, we would like to emphasize that this method can be a safe alternative to other existing methods of anesthesia for use in our peripheral hospitals where fund and maintenance facilities for equipments are very much lacking.

Acknowledgment:

We are thankful to Medimpex promotion Office in Bangladesh for suplying CALYPSOL (R) (Ketamine hydrochloride) for this clinical trial. This study is also partially supported by Bangladesh Oxygen Limited, who suplied the Glucose-oxidase,' strips for measuring blood glucose. We are indebted to them.

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Blood Pressure Responses to Intrahypothalamic injection of Carbachol in Anesthetized Cat

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Summary :

The blood pressure response to micro-injection of carbachol into the posterior hypothalamic nucleus was investigated in alpha-chloralose and urethane anesthetized cats.

Microinjection of carbachol 1-10 μg into the posterior hypothalamic nucleus produced a dose-dependent decrease in blood pressure and variable changes in heart rate. The depressor response to carbachol was antagonized by intravenous injection of atropine, however, the specific M_1 subtype of muscarinic receptor antagonist, pirenzepine, and the antinicotinic agent mecamylamine, given intravenously, could not block the depressor response. The depressor response to carbachol was a centrally mediated action, as the peripheral antimuscarinic drug methscopolamine, given intravenously, failed to block the fall in blood pressure. Microinjection of the specific M_1 subtype of muscarinic receptor agonist, McN-A-343, into the posterior hypothalamic nucleus, could not produce any effect on blood pressure or heart rate.

These results suggest that cholinergic mechanisms concerning with cardiovascular responses exist in the posterior hypothalamic nucleus, where, microinjection of cholinergic agonist produces depressor response, and that the M_1 subtype of muscarinic receptors are not involved in the mechanism.

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Introduction :

Stimulation of central muscarinic receptors with direct acting cholinomimetics or cholinesterase inhibitors evokes an increase of blood pressure in several species by increasing sympathetic nervous activity[4]. In contrast, experiments with intravenous injection of the cholinesterase inhibitor, physostigmine, intracerebroventricular injection of cholinergic agonists and topical ventral medullary applications of cholinomimetics produce centrally mediated depressor responses in anesthetized cats[1,2,3,10].

Cholinergic agonists evoke qualitatively varying responses from different areas in the brain. At least two brain regions respond to cholinomimetics by evoking definite cardiovascular effects; the ventrolateral medulla[17] and the posterior hypothalamus[5]. In the latter reports, microinjection of the cholinoceptor agonist, carbachol and several cholinestrase inhibitors into the posterior hypothalamic nucleus of anesthetized or conscious rats increase blood pressure [5,16].

The hypothalamus is known to contain significant concentrations of acetylcholine[8], and especially, the posterior hypothalamic nucleus contains high levels of the enzymes responsible for the synthesis of acetylcholinesterase[11]. Electrical stimulation of this brain region in the rat always evokes a hypertensive response[5], indicating that there is a mechanism in the posterior hypothalamic nucleus which serves to mediate a rise in arterial blood pressure.

Cardiovascular responses to cholinomimetics appear contradictory with both pressor and /or depressor responses which can be attributed to the state of the animal, i.e., conscious or anesthetized, the choice of the anesthetic agent and the locus

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of injection within the hypothalamus or other areas in the central nervous system. It can be presumed that in the cat, a cholinergic mechanism concerning with cardiovascular responses exists within the posterior hypothalamic nucleus. The present study was undertaken to investigate whether the blood pressure response to microinjection of carbachol into the posterior hypothalamic nucleus is a pressor or a depressor one in alpha-chloralose and urethane anesthetized cats, and whether the M_1 subtype of muscarinic receptors is involved or not.

Materials and Methods :

Experiments were performed in adult mongrel cats of either sex weighing 2.2—4.2 kg and anesthetized with alpha-chloralose 40 mg/kg and urethane 600 mg/kg intraperitoneally. Tracheostomy was done and cannulae filled with physiological saline were introduced into the femoral artery and vein for measurement of systemic blood pressure and injection of drugs respectively. The arterial cannula was connected with a pressure transducer (Model MM-2A, Kyowa, Japan) coupled to a pen-recorder (Type 3066, Yokogawa, Japan). Subsequently, the arterial cannula was filled with heparinized saline solution. The heart rate was measured by a pulse tachometer (type RM-5, Nihon-Kohden, Japan). Temperature of the cat was maintained at 37°C by a thermostatically regulated heat pad.

Cats were placed in a stereotaxic apparatus and a burr hole was made over the posterior hypothalamic nucleus. An injection guide cannula was directed towards the area at surface co-ordinates: 8.5 mm anterior to the interaural line, 0.9 mm lateral to the midline and 0.5 mm below the zero point, according to the atlas of cat brain by Snider and Niemer[18]. The cannula was anchored to the skull and to a small stainless screw inserted near the opening with acrylic cement. Drugs were injected by a microsyringe connected to a 25 gauge injection cannula which was lowered through the guide cannula to a depth of 2.0 mm below the tip of the guidecannula, so as to reach a depth of 2.5 mm below the zero point. Drugs were injected over a 30 sec period in a volume of 10µl. After the experiment, cats were decapitated and the brain was removed and fixed in a solution of 10% buffered formalin. The injection sites were verified histologically.

Drugs used were carbachol chloride, methscopolamine hydrobromide, mecamylamine, pirenzepine hydrochloride and McN-A-343 (4-(3-chlorophenylcarbamoyloxy) -2-butynyl trimethylammonium chloride). For intravenous injection, drugs were dissolved in saline and injected in a volume of 0.1 ml/100 g of body weight.

Results are expressed as mean ± S. E. M. Statistical evaluations were made using one-way analysis of variance (ANOVA)and paired Student's t-test.

Results :

Pretreatment levels of mean blood pressure and heart rate averaged 119 ± 1.3 mmHg and 244.7 ± 6.1 beats/min, respectively (n=20). Microinjection of 10µl of physiological saline into the posterior hypothalamic nucleus produced no effect on blood pressure (n=5). Microinjection of carbachol 1-10µg into the posterior hypothalamic nucleus produced a dose-dependent fall in blood pressure (Table-I) and variable changes in heart rate. Onset of the depressor response was within 2-3 min and peak fall reached at 15-20 min. Blood pressure remained decreased for about 60-90 min. The changes in heart rate were inconsistent; both tachycardia and bradycardia were observed and no attempt was made to further analyze their effects.

Table-I : *Dose-dependent depressor response to intrahypothalamic injections of Carbachol and McM-A-343 in anesthetized cats*

Treatment	n	BP (mmHg) Before treatment	After treatment	Δ BP
Saline (10µl; PH)	5	119.2±2.6	12.8±3.1^{NS}	0±2.7^{NS}
Carbachol (1µg;PH)	5	114.8±2.0	91.6±2.2**	-22.8±1.4**
Carbachol (10µg; PH)	5	123.2±2.2	86.4±2.9**	-36.8±1.2**
Carbachol (10µg;PH)	5	122.0±2.4	73.2±2.3**	-48.8±1.2**
McN-A-343(10µg;PH)	5	124.2±2.9	118.2±4.3^{NS}	-2.2±2.2^{NS}

Results are expressed as mean ± S. E. M. and n represents the number of cats. Saline, carbachol and McN-A-343 are injected into the posterior hypothalaimc nucleus (PH) and the peak changes or reductions are calculated. Carbachol produced a dose-dependent decrease in blood pressure (BP). Δ BP indicates the change BP. NS indicates not significant. ** P<0.01, compared to values before treatment (ANOVA and Students's t-test).

The depressor response to intrahypothalamic injection of carbachol was antagoinzed by intravenous injection of the antimuscarinic drug, atropine 0.25 mg/kg. Figure-I shows typical response of blood pressure to microinjection of carbachol 3µg into the posterior hypothalamic nucleus. The dose of 3µg of carbachol, which produced a decrease in blood pessure of 36.8 ± 1.2mmHg (n=5; <0.01) was used as a standard in subsequent investigations.

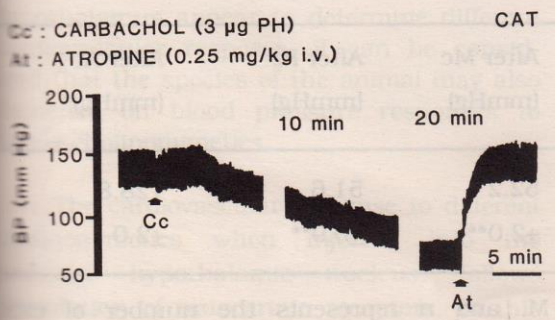

Fig. 1: *Representative record of the decrease in blood pressure (BP) to injection of carbachol 3µg into the posterior hypothalamic nucleus (PH) in an anesthetized cat. The decrease in BP was antagonized by intravenous injection (i.v.) of atropine.*

The depressor response to intrahypothalamic injection of carbachol was not antagonized by subsequent intravenous injection of methscopolamine 1mg/kg, the peripheral antimuscarinic agent. Again, mecamylamin 1 mg/kg, an antinicotinic drug, given intravenously, failed to antagonize the depressor response to carbachol. Furthermore, the specific M_1 subtype of muscarinic receptor antagonist, pirenzepine 2 mg/kg, given intravenously, could not produce any effect on the decrease in blood pressure. However, subsequent atropine injection blocked the depressor response to carbachol. Typical response of blood pressure and decrease in blood pressure to intrahypothalamic injection of carbachol

followed by subse quent intravenous injections of methoscopolamine, mecamylamine, pirenzepine and atropine are shown in Fig. II and Table II respectively .

Cc : CARBACHOL (10 µg PH)
Ms: METHSCOPOLAMINE (1mg/kg i. v.)
Mc: MECAMYLAMINE (1 mg/kg i. v.)
Pz : PIRENZEPINE (2 mg/kg i. v.)
At : ATROPINE (0. 25 mg/kg i. v.)

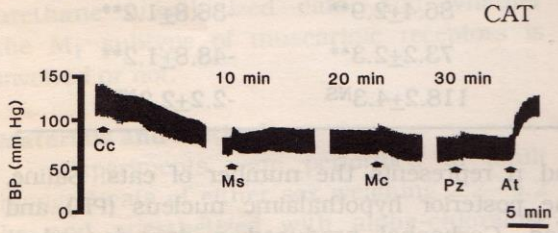

Fig. II: *Representative record or the effects of durgs on the blood pressure (BP) response to carbachol 10µg, injected into the posterior hypothalamic nucleus (PH) in an anesthetized cat. The fall in blood pressure was not antagoinzed by intravenous injection (i.v.) of methscopolamine, mecamylamine or pirenzepine, but blocked by atropine.*

The specific M_1 subtype of muscarinic receptor agonist, McN-A-343, was microinjected into the posterior hypothalamic nucleus in a high dose of 10µg (n=5), and there was no effect on blood pressure observed for 120 min (Table 1; Fig III. there was also no effect on heart rate (Data not shown).

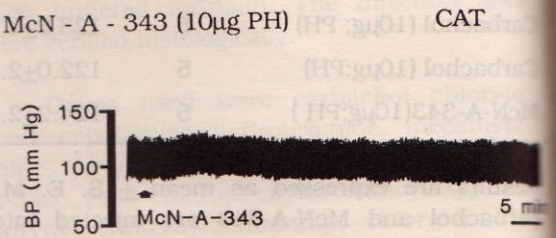

Fig. III : *Representative record of the effect on Blood pressure (BP) to McN-A-343 10µg, injected into the posterior hypoth-alamic nucleus (BP) in an anesthetized cat. BP did not change continuously observed for 120 min.*

Table-II : *Effects of drugs on the depressor response to intrahypothalamic injections of carbachol (Cc) in anesthetized cats.*

Control (mmHg)	After Cc (mmHg)	After Ms (mmHg)	After Mc (mmHg)	After Pz (mmHg)	After At (mmHg)
127.2 ±1.9	63.2 ±1.5**	59.2 ±2.2**	52.2 ±2.0**	51.6 ±0.9**	128.8 ±2.0

Results are exprressed as mean ± S. E. M. and n represents the number of cats. Abbreviations and doses of drugs used are: Cc-Carbachol 10µg; Ms- Methscopolamine 1mg/kg i. v. ; Mc - Mecamylamine 1 mg/kg i. v. ; Pz-Pirenzepine 2 mg/kg i. v. ; At -Atropine 0.25 mg/kg i. v. The depressor response to intrahypothalaimc injection of carbachol was not blocked by methscopolamine, mecamylamine or pirenzepine, but antagonized by atropine. ** P<0.01, compared with control (ANOVA).

Discussion :

The hypothalamus contains high concentrations of noradrenaline, acetylcholine and other neurotransmitters[19] and injection of these substances into this brain region or electrical stimulation of this area evokes rapid and pronounced changes in blood pressure and heart rate. In rats, the cholinergic agonist, carbachol, increases blood pressure when injected into the posterior hypothalamus[5] or into the anterior hypothalamus[16]. However, the mechanism underlying these effects are still poorly undrstood. In an attempt to observe further effects and to characterize the nature of receptors involved, carbachol was injected into the posterior hypothalamic nucleus in a-chloralose and urethane anesthetized cats.

In this sutdy, microinjection of carbachol into the posterior hypothalamic nucleus in the cat produced a decrease in blood pressur, The depressor response to carbachol was opposite to that evoked by microinjection of equal doses of carbachol into the posterior or anterior hypopthalamus in rats, using the same anesthetic preparation. since it has been known that the state of the animal, i. e., anesthtized or conscious, the choice of anesthetic agent and the locus of injection within the hypothalamus appear to determine different cardiovascular resposes, it can be considered that the species of the animal may also influence on blood pressure responses to varius cholinomimetics.

The cardiovascular response to different cholinomimetics when injected into the posterior hypothalamic nucleus, follows stimulation of muscarinic receptors. In this study, carbachol produces the depressor response through stimulation of muscarinic receptors located in the posterior hypothalamic nucleus.

Onset of the pressor response following centrally administered acetylcholine, carbachol or other cholinergic agonists in about 1-2 min in the rat[5,12,14]. In this study, onset of the depressor response to carbachol appeared due to leakage to other brain regions, or to the ventriculus tertius, or to the peripheral circulation. The peripheral leakage could be excluded, peripheral antimuscarinic agent, methsco-polamine, in a high dose, failed to block the depressor response, Again, little or no change in blood pressure occurred when carbachol was microinjected in an area close to the posterior hypothalamic nucleus but outside it (1 mm dorsal, 1 mm rostral and 1mm lateral). However, slow onset of action of carbachol may also suggest a low concentration of muscarinic receptors in the area.

The fall in blood pressure to carbachol was antagonized by systemic adminis-tration of the muscarinic antagonist, atro-pine, but was unaffected following admin-istration of the antinicotinic agent, mecamy-lamine. These findings indicate that the cholinergic receptors involved in the depressor response to carbachol have a sturctural requirement for muscarinic agonists.

In recent years, different muscarinic cholinergic receptors have been identified of which M_1 and M_2 are the well-known subtypes[6,7]. At present, pirenzepine is the selective antagonist for M_1 receptor and McN-A-343 is the selective M_1 agonist[9]. Though some reports suggest that M_2 receptor subtype is involved in cholinergic cardiovascular regulation in the rat[15,20], studies with specific M_1 agonists and antagonists were not performed in cats. In this study, it was seen that the specific M_1 muscarinic receptor agonist, McN-A-343, microinjected into the posterior hypo-

thalamic nucleus could not produce any change in blood pressure. Furthermore, the depressor response to carbachol, microinjected into the posterior hypothalamic nucleus, was not blocked by high dose of pirenzepine, the specific M_1 muscarinic receptor antagonist. These results suggest that muscarinic receptor concerning with depressor mechanism in the posterior hypothalamic nucleus in cats are of M_2 subtype, which can be stimulated by the relative M_2 receptor agonist, carbachol, and not by the specific M_1 agonist, McN-A-343. However, investigations with specific M_2 receptor agonists and antagonists are necessary to identify the subtype of receptors involved.

Electrical stimulation of the hypothalamus produces an increase in sympathetic nervous outflow[13]. In addition, central administration of cholinomimetics in rats produces similar increase in sympathetic nerve activity[14]. In contrast, recent findings show that intravenous injection of physostigmine or central application of cholinomimetics in cats produces a depressor response through decreassing the sympathetic nervous activity[1,10]. The sympathetic activity was not measured in this study, however, it can be suggested that the depressor response to carbachol injected into the posterior hypothalamic nucleus may be mediated through decreases in sympathetic outflow to the heart and peripheral vasculature. Further studies wih measurement of sympathetic nervous activity are requried to clarify the underlying mechanism of the depressor response.

In conclusion, results of the present study demonstrate that cholinergic mechanism exists wihtin the posterior hypothalamic nucleus in cats, which, upon stimulation with carbachol, mediates a depressor response. the muscarinic receptors of the M_2 subtype are possibly involved in the mechanism, and definitely, the M_1 subtype is not concerned with it. However, the physiological significance of muscarinic receptors in the posterior hypothalamic nucleus is yet to be established.

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Echocardiographic Analysis of 352 Cardiac Cases in a Military Hospital

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Summary:

During past four years, 352 cardiac patients were analysed echocardiograpically. Valuvular heart disesase predominates over all other cardiac abnormalities. 162 cases (46.02%) had valvular lesions. 96 patients (27.27%) were detected to have congenital heart disease. Mitral valve disease predominates over all other valve lesions. Out of 162 patients 103 (63.58%) had mitral valve disease in the form of mitral stenosis, mitral regurgitation or both. Pure mitral stenosis is the commonest group among all other valve diseases. 65 patients (40.12%) were detected to have pure mitral stenosis. Atrial septal defect appears to be the commonest congenital heart disease in this study. Out of 96 patients 48 (50%) had ASD and 31 (32.29%) had VSD. ASD is the commonest congenital heart disease in adult and VSD in children. Near about 75% of patients of ASD were in their second and third decade. 42% patients of VSD were in 1-5 yrs age group. Incidence of ASD was more common in female (F:M=1.35:1) and incidence of VSD is more common among males (M:F=1.38:1).

Mitral stenosis appears to be very common among young adults. 49.23% of MS patients were in 20-30 yrs age group. Female predominates over male (F:M=3.06:1) among patients with MS.

32 patients were detected to have cardiomyopathy in this study of which 22(68.75%) had hypertrophic cardiomyopathy.

(J Bangladesh Coll Phys Surg 1991; 9 : 60-63)

Introduction :

Echocardiography is a unique non-invasive method of imaging the living heart. It is based on the detection of echoes reflected back from tissue interfaces in the heart by a beam of inaudible ultra-sound pulses transmitted through a transducer applied to the anterior chest wall. These returning echoes are received by the same transducer and fed into the main unit for coversion into images which are displayed on a Video-monitor[1]. Since its introduction into clinical practice in the mid 1950s, echocardiography has developed into a highly sophisticated and indispensable, non-invasive cardiac diagnostic tool[2]. The management of patients with cardiovascular disease requires a comprehensive evaluation of both morphological and pathophysiological abnormalities associated with the disease process. To this end, a diagnostic technique that is non-invasive, painless, biologically safe, readily repeatable, economical and highly accurate would be ideal. Echocardiography satisfies these criterias and in so doing has emerged as the diagnostic procedure of choice in many instances. Echocardiography can supply detailed anatomical information of cardio-vascular structures[3].

Valvular heart disease (acquired rheumatic) is a major problem in Bangladesh. Echocardiography plays an important part in the diagnosis of all sorts of valvular heart disease.

The objectives of this study are to find out the incidence and distribution of cardiac diseases and to find out major cardiac problems in practice.

Materials and Methods.

This study was conducted in a cardiology department of a military hospital from July 1986 to September 1989. The patients include armed forces personnel, their families and other civilian patients referred from different private clinics. During this period 843 endividuals under-

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went echocardiographic examination. Out of these, 352 patients were detected to have some sort of cardiac abnormality. Each and every patient were analysed by M-mode and 2D-echocardiography. Along with echocardiography, all patients were assessed clinically and radiologically. Twelve leads electrocardiogram were done in all patients and in selected patients cardiac catheterisation were performed.

Results

Out of 843 cases examined, 352 patients have some form of cardiac lesion. Among them valvular heart disease and congenital heart disease constitute the major group. 46.02% patients had valvular heart disease, 27.27% had congenital heart disease and 7.1% ischaemic heart disease (Table-I). Mitral valve disease appears to be the commonest valve lesions and tricuspid and pulmonary valve involvement is very rare. Only 3.09% patients had either pulmonary or tricuspid valve disease (Table-II).

Table-I : *Types of heart diseases in 352 Patients.*

Type	No.	Percentage
Valvular heart disease	162	46.02%
Congenital heart disease	96	27.27%
Cardiomyopathy	32	9.09%
Ischaemic heart disease	25	7.10%
Others	37	10.51%

Table-II : *Distribution of Valve Lession (n=162)*

Type	No.	Percentage
Mitral valve disease	104	64.28%
Aortic valve disease	20	12.35%
Both aortic and mitral valve disease	28	17.28%
Pulmonary valve involvement	5	3.09%
Mitral and Tricuspid valve lesion	5	3.09%

Though ASD and VSD constitute the major group of congenital heart disease the number of TOF (8.33%) & PDA (4.17%) are also significant (Table-III). One each of single ventricle and Ebstein anomaly were also detected.

Table-III : *Congenital heart disease (n=96)*

Type	No	Petcentage
ASD	48	50%
VSD	31	32.29%
TOF	8	8.33%
PDA	4	4.17%
Ebstein Anomaly	1	1.04%
Bicuspid Aortic Valve	1	1.04%
Single Ventricle	1	1.04%

Mitral stenosis appears to be more common in 20-30 yrs of age group (49.23%) (Table-IV) and its incidence in female is much higher than male (F:M=3.06:1). 32 patients (9.09%) were detected to have cardiomyopathy in this series (Table-V).

Table-IV : *Age Incidence of Mitral Stenosis (n=65)*

Age	No.	Percentage
Below 10 yrs	2	3.08%
11 - 20 yrs	11	16.92%
21 - 30 yrs	32	49.23%
31 - 40 yrs	12	18.46%

Table-V : *Cardiomyopathy (n=32)*

Type	No.	Percentage
Hypertrophic	22	68.75%
Dilated	9	28.13%
Restrictive	1	3.13%

ASD is the commonest congenital heart disease detected in adult population. ASD is more common among females than male (F:M 1.35L1). VSD is very common in 1-5 yrs age group and it's incidence is higher among males, (M:F=1.38:1).

Discussion:

In this study the valvular heart disease appears to be the largest group comprising 46.02% of patients, followed by congenital heart disease 27.27%. Such observation are in agreement with the study done in Institute of Cardiovascular Diseases from 1981 to 1985[4]. This study corroborates the view that valvular heart disease (acquired-rheumatic) is a major problem in Bangladesh. The second group of heart disease in this series came as congenital heart disease with atrial septal defect predominating in adult and ventricular septal defect in children. It may be mentioned that ischaemic heart disease which is showing a small number in this analysis is not real representation of the problem because selected cases of coronary heart disease are only referred for echocardiography.

Echocardiography enables the accurate definition of morphology of the valve apparatus with reference to fibrosis, calcification and mobility of valve leaflets and the determination of the mitral valve orifice area by planimetry5. In this series out of 162 patients with valvular heart disease 103 (62.58%) had mitral valve disease in the form of mitral stenosis, mitral regurgitation or both. 65 patients had pure mitral stenosis. Detail morphology of the mitral valve apparatus is necessary for valvular surgery and this is more important in our situation in Bangladesh where a good number of patients are undergoing closed mitral commissurotomy (CMC). Nazrul Islam et al[6] suggested 2DE criteria for suitability for CMC :- (a) Thickened rough zone with pliable leaflets with (b) no calcification along the commisures and (c) with mild to moderate subvalvular lesion. Rheumatic mitral regurgitation occurs when leaflet coaptation is incomplete[7]. It is otherwise impossible to document mitral regurgitation by means of a two-dimensional echocardiogram. Doppler technique, in combination with an M-mode and 2D-echocardiography, can however detect and document mitral insufficiency[8]. The diastolic fluttering motion of the anterior leaflet of the mitral valve, when present, is considered as the echocardiographic hallmark of aortic insufficiency. Although it is possible to detect diastolic mitral valve fluttering by two dimensional echocardiography, its detection on an M-mode tracing is much easier and far more certain[7].

Thirty two patients were detected to have cardiomyopathy (9.09%) in this series out of which 22 were of hypertrophic variety. Detection of twenty two patients of hypertrophic cardiomyopathy in a small series is significant. Erroneous diagnosis of IHD is possible in hypertrophic cardiomyopathy. The characteristic features of hypertrophic cardiomyopathy revealed by echocardiography are narrowness of the ventricular cavity, asymmetrical hypertrophy of the interventricular septum and the SAM of the mitral echo[9]. Asymmetrical septal hypertrophy remains a sensitive and specific indicator of hypertrophic cardiomyopathy[10].

The Echo-Doppler technique has indeed earned itself a well deserved place among the armamentarium of noninvasive cardiac diagnostic tools. Its present capabilities has opened new windows to the heart and thus

new vites in our understanding of the morphological and pathophysiological changes associated with disease of cardiov-ascular system[11].

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Tuberculosis of the Breast : Report of Three Cases

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Smmary :

Three cases of tuberculous mastitis are presented. Presenting complaints were farm lump with discharging sinus in two and recurrent abscess formation with multiple cischarging sinuses in one. Diagnosis was based on histopathological appearance, raise derythrocyte sedimentation rate, positive Mantoux test and response to chemotherapy. Excision of the lump including sinuses and chemotherapy proved effective in all the cases.

(J Bangladesh Coll. Phys. Surg 1991; 9 : 64-65)

Introduction:

Tubercular mastitis was first described by Sir Astley Cooper in 1829 as scrofulous swelling in the bosom of young woman[1]. It is a relatively uncommon condition of the breast. Since then from time to time there have been reports of the disease. Till 1971 over 500 cases were reported[2] Most recent report are from India[3,4] and South Africa[5,6]. Though tuberculosis spares no race, region, nation or individual but it is prevalent in third world countires like Bangladesh, India and Pakistan and the dreadful disease spares no organ. So is to breast. We like to report three cases of tuberculous mastitis diagnosed at Sher-e-Bangla Medical College Hospital, Barisal during the last two year period, 1989-90 when approximately 120 breast cases were admitted and operated for different types of breast lesion.

Materials and Methods:

In 1989 and 1990, 3 cases presenting to the surgical units (I & IV) of Sher-e-Bangla Mdical College Hospital were found to have tuberculosis of the breast. During the same period 120 cases were admitted and operated for different types of breast lesion. All the 3 cases were female. There age was 25, 29 and 35 years.

Presenting complaints in two were painless farm lump in the breast with discharging sinus. One patient had history of recurrent abscess formation with multiple nonhealing discharging sinuses. The last patient had palpable nontender axillary lymph nodes. All the 3 cases were treated outside for quite sometime with different types of antibiotics. In two cases the lump was incised elsewhere before getting admission to the hospital with the purpose of drainage of abscess resulting to nonhealing sinus formation. In the other case the abscess that was formed bursted spontaneously. One patient had associated pulmonary tuberculosis.

Diagnosis was made by biopsy and histopathological examination in all the cases. Presence of tubercle with central caseation and granulomatous inflammatory cells including foreign body giant cell were the diagnostic criteria. Erythrocyte sedimentation rate was high in all the cases. Mantoux text was in the positive range in all the three (More than 10 mm at 72 hours).

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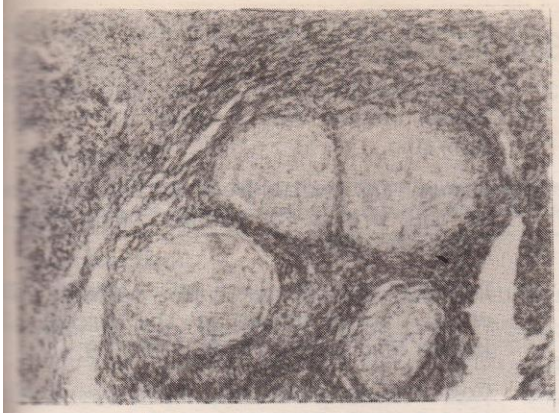

Fig-1 : *Section of breast showing granulomatous inflammation characterised by the formation of well defined tubercles (H&E X 88).*

Two cases were treated by excision of the lump with the sinus and in one case wedge biopsy of the abscess cavity including the skin sinuses. Chemotherapy was started in all the cases and the ulcers and the sinuses were healed by just one month after the starting of the drugs.

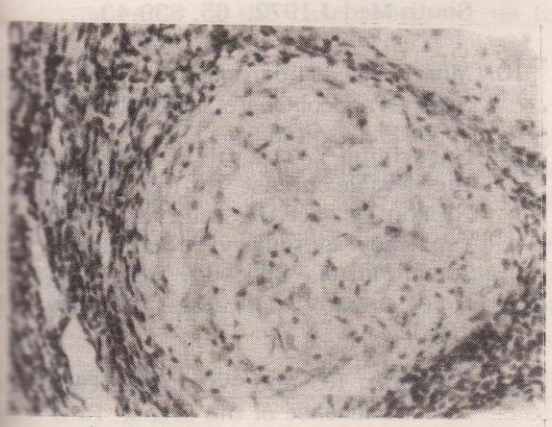

Fig . 1 : *Section of breast showing granulomatous inflammation characterised by the formation of well defined tubercles (H & E X 352).*

Discussion :

Tuberculosis is rapidly disappearing infectious disease in the developed countries[7]. It is a systemic infectious disease and it has a broadspectrum of clinical menifestations that may referable to any organ and doubt should be cost of tuberculous nature of any systemic disease[8]. It is said that breast, skeletal muscle and spleen appears to enjoy an immunity to tuberculosis[7]. Among the vastness of reports of extrapulmonary tuberculosis in general, tuberculous mastitis is rare[2]. Incidence is 0.5 to 1.4 percent of breast lesions in south Africa[2] and less then 4.5 percent reported from India[3]. In our report it is 2.5 percent.

Diagnosis of tuberculous mastitis is difficult specially in the early stage. Sinuses are not the sole characteristic feature of tuberculosis and the sinuses appear late in the disease process.

Non-tuberculous granulomatous mastitis may present with same type of sinuses[7]. Diagnosis is histopathological in most of the series. Non-tuberculous granulomatous mastitis, plasma cell mastitis and fat necrosis can present difficulties even in histopathological diagnosis. Cohen[5] had shown that in her series that none of the 34 cases were suspected of tuberculosis clinically. The patient who presented with a lump in her breast is clinically indistinguishable from carcinoma of the breast and there is no pathognomic feature to distinguish tuberculosis of the breast from breast cancer[2,7]. Though actinomyosis is rare in the breast but it should also be considered in the differential diagnosis[9].

Mycobacterium may reach the breast in any one of the following routes 1) a penetrating wound of the skin of the breast 2) through the lacteferous duct from the nipple 3) the blood stream 4) the lymphatics and 5) direct extension from the lungs

and chest wall[2,10].. Retrograde extension to the breast from the cervical or axillary lymphnodes is the usual mode of spread is conceded by most but not the sole mode of spread[2,11,12]. In 60% of the reported cases of tuberculosis of the breast lesion is primary with no evidence of tuberculosis elsewhere and the remainder 40 percent are secondary to wide spread of tuberculosis[5,13]. Prior to the development of specific therapy for tuberculosis treatment of tuberculous mastitis include curettage, cauterization, aspiration, incision and drainage to simple or radical mastectomy.

Wilson and Macgregor[13] in their opinion said that treatment should be either medical or surgical and due to long term costly therapy and for recurrence after medical treatment they prefered the line of simple mastectomy. Algaratnam and Ong[7] suggested conservative surgery like lumpectomy with chemotherapy as the treatment of choice. On the other extreme Dharkar et al[4] suggested surgery in advanced complicated cases only.

Among the reported cases incidence of tuberculosis of the breast is more in nulliparous and non-nursing mothers[7,10]. Breast feeding may give some form of immunity to the breast from tuberculosis.

Treatment of the disease does not pose any problem if it is once diagnosed. Main problem is to consider its presence and to have a great suspicion about it in case of breast lump or recurrent breast abscess formation with nonhealing discharging sinus. It is more true in the regions where tuberculosis is preponderent.

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Tuberculosis of the Penis : A Case Report

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Summary :

One case of tuberculosis of the penis mimicking carcinoma of penis presented. It stressed need for histopathological confirmation of diagnosis of all penile ulcer before mutilating Surgery.

(J Bangladesh Coll Phys Surg 1991; 9 : 67-69)

Introduction:

Tuberculosis is a chronic granulomatous inflammatory condition caused by Mycobacterium tuberculoisis. Even with the modern therapy of present day, both pulmonnary and extrapulmonary (especially bone, cervical, intestinal) tuberculosis are very common in Bangladesh. But tuberculosis of the penis is a very rare entity. In 1878 Fournier described the first case of primary tuberculosis of the penis[1]. Ch'i An-Sheng's report showed, 4% of Chinese patient with genitourinary tuberculosis have associated penile lesion[2]. There were several case reports of tuberculosis of the penis from our neighbouring country, India. But from Bangladesh there is no known reported case of tuberculosis of the penis. Extreme rarity of the case prompted us to report the case.

Case Report:

K. M., a 53 year old male Muslim factory clerk from Comilla was admitted in Dhaka Medical College Hospital on August 1988 with a large ulcer in the glans penis for 10 months. Five years back the patient noticed a small painless swelling on the right side of the distal shaft of the penis. Ten months back he developed several swellings in the glans penis. Inspite of treatment these nodules enlarged in size and become ulcerated. He was treated with antisyphilitic drugs but without effect. He was impotent for the last 5 years. There was no history of chronic cough or contact tuberculous patient. The patient married thrice. His first two wives and their issues were dead. His second wife had 2 still born.

On examination, the patient was moderately anaemic. There was a large fungating ulcer (5 X 4 cms.) on the under surface and the sides of the glans penis. Ulcer was oozing, non tender. Margin was everted in most of the places and undermined in some places, and base was indurated.

There were few nodules measuring less than 1 cm on the right side of the distal penile shaft. Penis was moderately stiff. Epidediymis and testes were normal. Inguinal lymph node not enlarged. Provisional diagnosis was carcinoma of the penis. (Photo-1).

Wedge biopsy from ulcer margin was done. Histopathological examination showed few granuloma composed of epitheloid cells and Langhans giant cells with early caseation necrosis. Overlying stratified squamous epithelium showed marked hyperplasia. No malignant cell detected (Photo-2). VDRL test and smear test for Donovan bodies were negative. There was no clinical or radiological evidence of tuberculosis in the lungs

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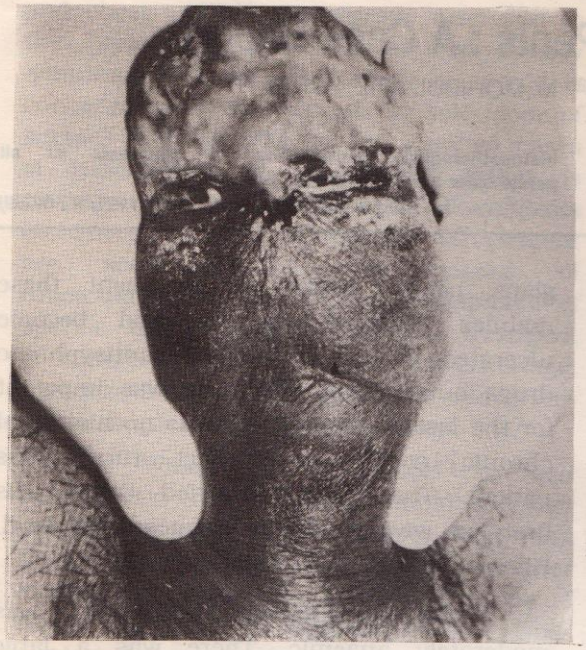

Fig 1 : *(Showing penile ulcer)*

Fig 2 : *Wedge biopsy from penile ulcer edge showing granuloma (mag 110X)*

and genitourinary system. Then the patient was discharged with antitubercular drugs (Rifampicin 600 mg/d, Ethambutol 100 mg/d and INH 300 mg/d).

There was only a minium reduction of size of the ulcer. And the penis remain stiff as before and troublesome oozing continued. After two months patient was re-admitted and partial amputation of the penis was done. Rifampicin and INH continued for next 7 months.

Now the patient is all right for the last two years after operation.

Discussion:

Tuberculosis of the penis may be primary or secondary depending upon absence or presence of tuberculous lesion elsewhere in the body[1-3]. Primary tuberculosis may be transmitted by sexual contact with the infected partner or by infected fomites[3,4]. It might have been caused, in the past, by the process of sucking as a means of attaining haemostasis after ritual circumcision in jewish children[2]. Secondary tuberculosis may be from haematogenous spread from coexisting tubercular lesion or from infected periurethral gland from tuberculosis bacilli in the urine or extravsation of urine[3]. Tuberculosis of penis in most of the cases present as superficial ulcer in the glans or shaft of the penis but it may also present as subcutaneous nodules without skin ulcer (tuberculous cavernositis)[3-4]. In the present case there was an extensive ulcer involving glans penis as well as there were subcutaneous nodules in the shaft.

Tuberculosis of the penis may be confused, in the early stage, with syphilis or granuloma inguinale and in the late stage, with the carcinoma of the penis[2,5].

Ideal diagnostic procedure is combination of histopathological examination and culture. But as culture from ulcerated lesion is often unhelpful, so histopathological examination is most reliable[2].

Depending upon case to case wide range of treatment are avaiable—antitubercular therapy alone; apmputation of penis; diathermy or ultraviolet rays followed by anti tubercular therapy;or antitubercular therapy followed by reconstructive surgery. In this case, before considering amputation we have considered age of the patient, impotency for 5 year, chronicity of the disease, unfavourable response with antitubercular therapy alone, and demand of patient for amputation.

Suspicion and early diagnosis can save mutilating steps in many patients.

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College News

Examination News:

Results of FCPS Part I, FCPS Part II and MCPS Examinations held in July, 1991 are given below:

299 candidates appeared in FCPS Part I Examination held in July, 1991 of which 11 candidates came out successful. Subjectwise results are as follows:

subject	Number appeared in theory examination.	Number qualified for viva-voce.	Number Passed
Medicine	46	8	2
Surgery	81	13	2
Obst. & Gynae.	51	5	1
Paediatrics	44	7	0
Ophathalmology	26	9	3
ENT Diseases	12	2	1
Psychiatry	5	1	0
Anaesthesiology	14	7	0
Radiology	7	2	1
Radiotherapy	2	0	0
Pathology	9	3	1
Physical Medicine	2	0	
TOTAL	299	57	11

101 candidates appeared in FCPS Part II Examination in different subjects. List of candidates who satisfied the board of examiners is as follows :

Roll No.	Name	Graduated from	Specility
7	Dr. Md. Azharul Hoque	Rangpur Medical College Sy	Internal Medicine
15	Dr. Shekhar Battacharjee,	Sylhet M. A. G. Osmani Medical College	Internal Medicine
30	Dr. Pradip Kumar Dutta	Chittagong Medical College	Medicine
35.	Dr. Md. Rafiqul, Islam	Mymensingh Medical College	Medicine
38	Dr. Md. Shahid Karim	Dhaka Medical College	Surgery
39	Dr. Rabindra Kishore Nath	Dhaka Medical College	Surgery
52	Dr. Md. Jahangir Kabir	Rangpur Medical College	Surgery
56	Dr. Md. Taibur Rahman	Rangpur Medical College	Surgery
61	Dr. Mukul Kumar Das	Sir Salimullah Medical College	Surgery
63	Dr. Jashim Uddin Ahmed	Chittagong Medical College	Surgery
67	Dr. Ranjit Ranjan Roy	Chittagong Medical College	Paediatrics
68	Dr. Chowdhury Yakub Jamal	Chittagong Medical College	Paediatrics
69	Dr. Kazi Abdul Mannan	Chittagong Medical College	Paediatrics

Roll No.	Name	Graduated from	Specility
71	Dr. Md. Rafiqul Islam	Chittagong Medical College	Paediatrics
73	Dr. Md. Lutfor Rahman	Dhaka Medical College	Paediatrics
74	Dr. Mohammad Azizul Hoque	Mymensingh Medical College	Paediatrics
75	Dr. Md. Nazrul Islam	Mymensingh Medical College	Paediatrics
76	Dr. Md. Iqbal Bari	Rajshahi Medical College	Paediatrics
82	Dr. Showkat Jahan	Dhaka Medical College	Obst.&Gynae
83	Dr. Mahbubur Rahman Chow.	Dhaka Medical College	Ophthalmology
84	Dr. Md. Abdul Awal Mia	Mymensingh Medical College	Ophthalmology
85	Dr. Deen Mohd. Noorul Huq	Mymensingh Medical College	Ophthalmology
86	Dr. Md. Shamsul Haque	Mymensingh Medical College	Ophthalmology
90	Dr. Wahiuddin Mahmood	Chittagong Medical College	Anaesthesiology
95	Dr. A. H. M. Mestafizur Rahman	Sylhet M.A.G. Osmani M. College	Psychiatry
96	Dr. Mohammad Zillur Rahman	Dhaka Medical College	E. N. T.
100	Dr. Zuberul Islam Chowdhury	Sylhet M.A.G. Osmani M. College	Radiology

110 candidates appeared in MCPS Examinations in different subjects; List of candidates who satisfied the board of examiners is as follows :-

Roll No.	Name	Speciality
20	Dr. Md. Ruhul Amin	Surgery
22	Dr. A. K. M. Shamsul Alam	Surgery
30	Dr. Md. Idris Ali	Surgery
34	Dr. Hasan Ashraf	Paediatrics
47	Dr. Rokeya Khatun	Obst. & Gynae.
48	Dr. Nazma Begum	Obst. & Gynae.
50	Dr. Shamima Chowdhury	Obst. & Gynae
54	Dr. Hazera Khatun	Obst. & Gynae.
58	Dr. Selina Begum	Obst. & Gynae.
66	Dr. Sultana Begum	Obst. & Gynae.
68	Dr. Amal Chandra Paul	Obst. & Gynae.
69	Dr. Md. Mahbub Alam	Obst. & gynae.
72	Dr. Md. Nazmul Hoque	Ophthalmology
82	Dr. Nilufar Sultana	Anaesthesiology
84	Dr. Md. Shamsuzzoha Sirker	Anaesthesiology
93	Dr. Md. Moniruzzaman	Clinical Pathology
94	Dr. Ikramul Islam	Clinical Pathology
96	Dr. Md. Abdul Hakim	Radiology
98	Dr. K. M. Altaf Hossain	Dental Surgery
99	Dr. Abul Kalam Azad	Dental Surgery
100	Dr. Khandker Iftekharuddin Ahmad	Dental Surgery
101	Dr. Jalal Uddin Mahmood	Dental Surgery
107	DR. S. A. Aleem	Psychiatry
110	Dr. Abdul Momin	Dermatology & Venereology