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JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

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CONTENTS

EDITORIAL

Acute Appendicitis : Triumphs & Tragedies

Acute appendicitis is the commonest global abdominal surgical emergency. It's incidence is low in high dietary fibre consumers. It is rarely encountered before the age of two years, reaches its peack during second and third decades and then declines,but no age is immune. Perforation of the inflammed appendix is encountered commonly at the extremes of age. Although considered one of the most elemental of general surgical disease process, it's notoriously inconsistent presentation regularly confounds the diagnostic acumen of even the most experienced surgeons. More than a century have passed since Reginald Haber Fitz coined the term "appendicitis " in his famous paper " Yet appendicitis continues to be a paradox"[1]. Perforated appendicitis resulted in the fatality of the Lewis & Clerk expedition and the significance of this disease has been well known since Charles McBurney's famous study on appendicitis presented before the New York surgical society in 1889[2,3] . However though the surgeons have been confronting acute appendicitis for more than a century its diagnosis remains elusive till today. Even if some doyens can diagnose the disease more accurately the fact is in most of the cases it is the novice who are to take the responsibility of decision making and their decision may be wrong in about 50% of cases [4]. The diagnostic accuracy rates vary in various involved patient population. Negative misdiagnosis rate is low in young male while the figure is much higher in females of child bearing age and children. Though the misdiagnosis rate is comparatively low, very high rate of perforation of appendix reflects the difficulty of diagnosis in old age. Diagnosis is also difficult during pregnancy and may result in both maternal and foetal mortality[4,5].

Life time risk of appendicitis is 5-20% with perforation rate 17-20%[6]. Surgeons resolve the dilemma of right iliac fossa pain mimicking acute appendicitis either by observation until clinical diagnosis is clear or by immediate operation. As the incidence of perforation is more or less proportional to the duration of the disease process traditional teaching has encouraged the surgeons to operate rather than observe even if the diagnosis is doubtful.This teaching has been challenged by some study[6]. The diagnostic aid introduced uptil now could not yet render this orthodox surgical teaching obsolete.

Universally practiced leukocyte count reveals elevated count along with elevated neutrophil in many series. Leukocytosis is also seen in patients having pain in right iliac fossa due to other causes. Also perforated appendix may show normal blood count. Thus the universally practiced sensitive test has lost its specificity & believed to have limited diagnostic value[7].

Plain abdominal radiography has proved as a non-specific, insensitive and inaccurate investigation, mentioned here only to discourage its use[8].

Barium enema examination is advocated to visualise barium filled normal appendix. Nonvisualisation is taken as obstructed appendix due to inflammation. Both of this statements have got falacy and the investigation has essentially limited role in the diagnosis of acute appendicitis in the era of ultrasonography and CT scanning[8].

Barium swallow examination has claimed 95% accuracy for diagnosis of acute appendicitis in children , needs further evaluation[9].

CT scanning can diagnose accurately the advanced cases of acute appendicitis but not the early cases [8]. Inflamed appendix is visible where as normal appendix is invisible on ultrasonography. Appendicolith or fluid surrounding the organ confirms appendicitis. Sensitivity and specificity of the investigation is highly satisfactory in the hands of the experts. Also this test can exclude other surgical, gynaecological and obstetrical diseases .But it is an operator dependent investigation[8].

Laparoscopy differentiates normal and inflamed appendix or identifies the signs of inflammation in the right iliac fossa when no other pathology could be

detected to account for appendicitis. Nonvisualisation of appendix acts as an indirect sign and might guide to the diagnosis. This investigation can also diagnose other causes of intra abdominal pathology and thus can reduce misdiagnosis[10].

A pre - programmed computer could diagnose acute appendicitis accurately if accurate data input from the history & physical examination could be provided[8].

Radioisotope scanning is highly sensitive but unreliable in women and there is need to exclude gynaecological disease by other methods[8].

Increased leukocyte or pus in peritoneal aspirate and lavage is a reflection of acute appendicitis. Gynaecological infection and mesenteric adenitis may also cause leukocyte rich peritoneal fluid . Negative lavage may exclude all three conditions[11].

In clinical diagnostic scoring patient's signs, symptoms and white blood cell count are given various scores. Patient who scores above a certain figure is considered to have appendicitis. The sensitivity and specificity of the investigation has been claimed around 90%. Misdiagnosis rate is also claimed to come down to 30% [8]. But it needs further evaluation.

In the new millennium despite the introduction of so many sophisticated investigations ,appendicitis still remains as a clinical entity and an ongoing diagnostic challenge. None of the available investigation possesses high degree of sensitivity, specificity and accuracy and hence could not provide marked difference in perforation and misdiagnosis rate. Thus a surgeon confronting a patient suspected to have acute appendicitis is wedged between the Schylla of perforation and Charybdus of negative appendecectomy. Therefore the likely aim of the surgeon shall be prevention of perforation of the appendix at any cost. To achieve this goal surgeons have created a surgical security zone which allows to accept certain percentage (15-30) of misdiagnosis rate with indemnity. To overcome this equivocal situation there is no substitute of skilled interviewing of the patient or attendent and eliciting the physical signs very rightly to make a relatively accurate diagnosis. So the clinical judgement still over-rules the laboratory aid when diagnostic dilemma prevails. Emphasis on the diagnostic aids at the expense of clinical evaluation will be bound to diminish the quality care of the patients with acute appendicitis.

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(J Bangladesh Coll Phys Surg 2004; 22 : 40-41)

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ORIGINAL ARTICLES

Cardiac Complications in Haemodialysis Patients with Special Reference to Diabetic (NIDDM) Subset

A A S MAJUMDER

Summary

This cross-sectional study was done to see the prevalence of cardiac complications in maintenance haemodialysis with special attention to diabetic subset along with a yearly follow-up upto two years. Thirty patients were selected, 76.6% (n=23) were non-diabetic and 23.3% (n=7) were diabetic; mean age was 44.36 (±12.62) years; duration of MHD was 19.53 (±18.9) months; systolic BP 162.83 (± 19.32) mmHg: diastolic BP 90.10 (±9.77) mmHg; number of IHD 36.6% (n=11); Hb 6.25 (±1.33) gm/dl. On echocardiogram, 80%(n=24) lead concentric hypertrophy and 20% (n=6) eccentric hypertrophy; LV mass index (LVMI) was 246.32g/m^2 in male (n=17) and 209.11 (±53.98) g/m^2 in female (n=13). When compared between diabetic and non-diabetic subset, significant difference was seen between age of 58.42 (±12.48) years vs 40.08(±9.24) years (p<0.05); presence of IHD 71.45% vs 26.08% (p<0.05); mean duration of HD 35(±8.42) months vs 51.09 (±21.44) months (p<0.05) in the surviving patients, on second year. Fifteen patients died at the end of two years followup and in 60% most likely cause was cardiac disease. When living and deaths were compared a significant difference was seen in interventricular septal thickness (IVST) 13.7(±1.6)mm vs 16:521(±3.3) mm (p<0.05); LVMI 170.81 (±30.07)g/m^2 vs 230.6(+-2l.9) g/m^2 (p<0.05); duration of 49.27(±19.66) months vs 32.26(±19.73) months (p<0.05) and mortality 57% vs 47%. We may conclude that cardiovascular complications are highly prevalent in MHD patients; diabetic patients are of older age group with increased incidence of IHD probably had caused decreased dialysis survival with increased mortality subsequently: LVMI is an independent predictor of mortality in both diabetic and non diabetic groups.

(J Bangladesh Coll Phys Surg 2004; 22 : 42-48)

Introduction

Haemodialysis (HD) is the main mode of renal replacement therapy (RRT) in end stage renal disease (ESRD) patients worldwide. United States Renal Data System (USRDS) shows the distribution of dialysis modalities where 80% are on HD[1]. In Bangladesh, HD also plays the major role in RRT[2]. Heart diseases are remarkably prevalent in dialysis patients at the start of therapy for ESRD. In a study of a cohort of 433 ESRD patients in whom dialysis therapy has been started, only 21% patients were found to be normal on echocardiograrn[3]. There is also increased incidence of atrial and ventricular dysrhythmias and complex ventricular ectopics in dialysis population[3]. European Dialysis and Transplant Association (EDTA) reports that the patients receiving RRT have a 16-19 fold increased risk of myocardial ischaemia and infarction when compared with age-sex matched population without renal failure[4]. Cardiac complications are major cause of death in dialysis patients consisting of about 40%[5].

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Canadian Organ Replacement Registry (CORR) in 1990 annual report mentioned that 44.7% dialysis patients died of cardiovascular diseases[6]. USRDS confirmed that 50% of total death in dialysls patients is due to cardiovascular diseases[7].

Diabetes mellitus is a common cause of ESRD and in turn is also a risk factor for cardiovascular diseases. In some countries, diabetes mellitus is the leading cause of ESRD.

United States Renal Data System (USRDS) 1 9 9 3 report shows that 34.3% of the total ESRD patients are diabetic. As for Bangladesh, a study shows 24% of MHD patients are diabetic[8]. Diabetic patients have a higher mortality rate, three times more than that of other ESRD patients[5]. Cardiovascular disease is the most common cause of death[5]. Blindness, coronary artery disease cerebrovascular and peripheral vascular diseases, are the common causes of morbidity and mortality. For these reasons diabetics were excluded from RRT in sixties and early seventies. But due to increased number of diabetic population and improvement in RRT more and more patients with diabetes are included in replacement therapy.

Echocardiogram has been proved to be a sensitive/ non-invasive tool for assessing left ventricular performance at an early stage. Left Ventricular Hypertrophy (LVH) in normal population is shown to be an adverse prognostic indicator[9-10]. Left Ventricular Hypertrophy (LVS) is also shown to be an independent prognostic factor in survival of ESRD patients[11] Echocardiography provides information about left ventricular geometry, contractility, and systolic-diastolic dysfunction. The type of hypertrophy (concentric/eccentric) can also be determined. So, emphasis of echocardiogram is given in this study.

This cross-sectional study was undertaken to assess the prevalence of cardiovascular complications in HD patients with special reference to diabetic subset and to find out the prognostic impact of echocardiographic determinants in both diabetic and non-diabetic patients.

Materials and Method :

Institute of Post Graduate Medicine & Research (IPGM&R). Dhaka is a tertiary referral Institute. All modalities of RRT are provided in this center. The study was done by the Department of Nephrology and the Department of Cardiology. More than 100 patients are treated annually in Nephrology department of which 24% are diabetic[8]. Out of 60 undergoing HD at the time of this study (for a variable period of time), 30 were selected. The study was done in the year 1996 and a follow-up was made at the end of '96 and '97 to evaluate the mortality. In second year there was 4 dropouts two transplanted: two discontinued). Of the 30 selected, seven were diabetic and 23 non-diabetic. Diabetic subset represented the similar percentage of the total patients currently treated. All were noninsulin-dependent diabetic (NIDDM) and did not need any hypoglycaemlc agent for controlling diabetes. Their medical records indicate that most likely cause of renal failure was diabetic nephropathy.

Patients were on haemodialysis for at least three months to avoid immediate complications of haernodialysis like - disequilibriuim syndrome and also patients were to be adjusted on this modality of treatment.

Exclusion criteria were acute renal failure, bed ridden patient, abnormal higher psychic function, taking drugs like steroids, sympathomimeties, suffering from chronic infection (TB. kala-azar. malaria etc.), decompensated liver disease, malignancies and patients with-dysrhythmias, old myocardial infection coronary angioplasty or bypass graft.

All the patients were hypertensive except one and was on multiple antihypertensives. None was getting erythropoietin for correction of anaemia.

All patients were dialysed through forearm arterio-venous fistula (AV fistula) 4 - 5 hours session about 2-3 times a week. Dialysis was done by using hollow fibre dialyser with a surface area of 1.1 sq. meter. Acetate dialysate was used at a rate of 500 ml/minute. Blood flow was 180-200 ml/ minute. Water was used after reverse osmosis.

Patients BP (supine) was recorded after evaluating the recordings of pre-dialysis BP over a period of one month. Cardiovascular status was evaluated by taking detailed clinical history and examination. Echocardiogram was performed after a mid-week haemodialysis whenever possible.

Echocardiogram was done by using M - mode and 2D echocardiography. Data were measured as criteria set by ASE[12]. Basic measurements included left ventricular internal diameter in diastole (LVIDd) and in systole (LVIDs), posterior wall thickness of left ventricle (LVPWT), interventricular septal thickness (IVST) and fractional shortening (FS). Left ventricular mass index (LVMI) was calculated by using following formula[13].

LVMI (g/m^2) =1.04 [$(LVIDd + IVST + LVPWT)^3$ - $(LVIDd)^3$)] - 13.6/ body surface area.

Left ventricillar end-diastolic volume index (LVEDVI) was calculated by the formula[14] : LVEDVI

$$ml/m^2 = \frac{7.0}{2.4 + LVIDd} (LVIDd)^3 \text{ body surface area}$$

In case of death, cause of death was concluded from medical records.

Definitions of terms:

Left ventricular, hypertrophy : LV mass index > $131 gm/m^2$ in males and >I00 gm/m^2 in females[15].

Concentric hypertrophy: in response to pressure overload, hypertrophy with normal cavity volume[16].

Eccentric hypertrophy : in response to volume overload. hypertrophy with LV dilatation (cavity volume > 90ml/m^2)[16].

Systolic dysfunction : Fractional shortening 25% or less.

Cardiac mortality: Death recorded as due to myocardial infarction. or from other cardiac causes[17].

Statistical analysis

Results are expressed as mean ± SD. Students unpaired 't' test was used to see the level of significance between groups. Chi square (x^2) and "Z" tests were also used. P value <0.05 was considered significant.

Results :

Number of patients were thirty. 17 were male, 13 female and ratio was 56.7:43.3 (Table-I). Mean age was 44.36 (±12.62) years. Cause of renal failure was chronic glomerulonephritis 70% (n=21). diabetic nephropathy 23.3% (n=4) and 6.6% (n=2) were due to hypertension and obstructive uropathy. Mean duration of haemodialysis was 19.53 (±18.90) montbs at the beginning of the study with a range of 3-68 months. Systolic BP was 162.83 (±19.32), diastolic BP 91.10(±9.77) mmHg and mean pressure was 116.51 (±15.11) mmHg. Ninety seven percent (n=29) were hypertensive and were on multiple antithypertensive medications. 36% (n=11) were indentisied clinically as having angina (IHD) and they were on different nitrate preparations continuously or on demand. Congestive cardiac failure (CCF) was noted (both left and/or right) In 26.6% (n=8). Mean Hb was 6.25gm/dl.

Echocardiographic measuremeanis showed that all the patients had LVH (Table-II).

Table-I

Baseline choractertstics of the patient population

Total number of patients	-	30
Age (years)	-	44.36 (±12.62)
Male	-	17
Female	-	13
M/F Ratio	-	56.7 : 43.3
Renal Disorder		
Glomerulonephritis	-	70%
Diabetic nephropathy	-	23.30%
Hypertensive nephrosclerosis	-	3.30%
Obstructive uropathy	-	3.30%
Duration of Haemodialysis (months)	-	19.53 (±18.90)
Blood pressure (mm of Hg)		
Systolic	-	162.83 (±19.32)
Diastolic	-	91.10 (±9.77)
Mean pressure	-	116.4 (±15.11)
Hypertensive	-	97%
Antihypertensive medication	-	97%
Angina (IHD)	-	36.6%
Congestive cardiac failure (CCF)	-	26.6%
Haemoglobin (gm/dl)	-	6.25 (±1.33)

Table-II

Echocardiographic abnormalities in the patient position

LVIDd (mm)	50.56 (±7.50)
LVIDs (mm)	34.83 (±6.70)
LVPWT (mm)	14.F0 (±3.00)
IVST (mm)	F5.10 (±-2.90)
LVEDVI (ml/m^2)	74.32. (±23.90)
LVMI (g/m^2)	
Male	246.32 (±71.61)
Female	209.1 (±-53.98)
FS %	31-46 (±6.78)%
Left ventricular hypertrophy	100%
Concentric	80%
Eccentric	20%
Systolic dysfunction	20%
Pericardial effusion	10%

Concentric hypertrophy was seen in 80% (n=24) and eccentric hypertrophy in 20% (n=6).. Systolic dysfunction was seen in 20% (n=6). Of these patients, five had concentric hypertrophy one had eccentric hypertrophy. None of the patients having CCF had systolic failure. Some of the patients having CCF or systolic dysfunction also belonged to IHD group. Calculated LVMI in male was 246.32 ±71.61) g/m^2 and in female 209.11 (±53.98) g/m^2 and all were well above the normal range defined for sex and body surface area.

Diabetic subset had a mean duration of DM for 14.28 (±3.94) years and hypertension for 10.10 (±6.25) years. But in non-diabetic group, as most were diagnosed at the stage of severe renal failure, no definite history of duration could be noted. When the diabetics and non-diabetics were compared (Table- III) significant difference in age and IHD was seen. Out of 30 patients, 20 patients survived in second year. Sixty continued dialysis and 4 discontinued. Of the16 patients, 5 were diabetic and 11 non- diabetic. At the end of second year significant difference in duration of dialysis was seen between these two groups.

Of the 30 patients, 15 died at the end of two years follow up, Four of them were diabetic. The overall mortality was 57% vs 47% between diabetics and non-diabetics. Patients surviving and dead were compared at the end of second year of the study (Table –IV). In dead females, there was higher value of IVST and LVMI in dead males, there was tendency of increased LVMI. All the eight patients with CCF died. Follow up at the end of second year showed difference in duration of dialysis in both of these groups: 49.27 (±19.66) years vs 32.26 (±19.72) years.

Table-III

Comparison between diabetic and non-diabetic patients

Variable	Diabetic (7)	Non diabetic (23)	p value
Age	58.42 (±12.48)	40.08 (±9.24)	< 0.05
Duration of HD			
• Starting	13.28 (±7.46)	21.43 (±20.90)	NS
• End of First year	24.57 (±8.54)	33.22 (±21.70	NS
• End of Second year	35.00 (±8.42)	5F.09 (2F.44)	<0.05
Systolic BP	161.42 (±13.45)	16F (±20.08)	NS
Diastolic BP	90.00 (±7.63)	90.00 (±8.86)	NS
Mean pressure	112.42 (±7.00)	114(±11.95)	NS
Hb	07.13 (±1.37)	06.35 (±1.30)	NS
IHD	71.45	26.08	<0.05
LVIDd	53.14 (±9.50)	49.56 (±6.90)	NS
LVEDVI	83.92 (±32:90)	72.58 (±20.40)	NS
LVMI	269.6 (±92.30)	233.14 (±60.45)	NS
FS%	31.00 (±7.50)	31.50 (±6.50)	NS

Table-IV

Comparison between living and those who died

Variable	Living (11)	Dead (15)	P value
Age 47.45 (±12.75)	45.4 (±11.03)	NS	
Starting HD duration	25.27 (±19.66)	19.53 (±19.14)	NS
Total HD duration	49.27 (±19:66)	32.26 (±19.73)	<0.05
Mean pressure	113.27 {±13.30)	114.6 (±8.30)	NS
IHD	27.20	46.60	NS
Hb	6.63 (±90)	6.44 (±1.50)	NS
LVPWT	13.45 (±2.20	15 (±3.60)	NS
IVST	13.7 (±1.60)	16.53 (±3.30)	<0.05
LVIDd	51.9 (±7.60)	49.09(±8.10)	NS
LVEDVI	78.74 (±26.70)	70.69 (±22.60)	NS
LVMI (female)	170.81 (±30.00)	230.60 (±21.90)	<0.05
LVMI (male)	245.37 (±87.49)	265.50 (±68.90)	NS
FS%	30.9 (±6.40)	32 (±7.50)	NS

Cause of death was not confirmed by autopsy but careful evaluation of history regarding death and medical records was done-to point out the most likely cause. Of the 15 dead patients, nine died of CVS complication 60%; 2 from CVD (13%) and four from other cause (27%) like irregular dialysis, fistula failure etc.

Discussion:

The strict selection criteria of study population was aimed at minimizing the effects of other factors on cardiovascular system and to observe only cardiovascular complications in haemodialysis population of end stage renal disease patients. The variable duration of haemodialysis with a wide range (Table-I) also enabled us to observe different cardiac complications of different dialysis time period. Hypertension has been reported to be present in 75-80% ESRD patients[18]. In this study it was present in 97% of the patients and all were on anti- hypertensive therapy. Suspected causes of hypertension in general are volume overload, elevated angiotensin II, uraemic toxins, sympathetic overactivity etc. By effective dialysis, majority patients show control of pressure by removal of excess body fluid and sodium. It is recommended that the BP should be at 140/90mmHg or below[19]. Increased BP with increased mean pressure in the study patients indicated increased peripheral resistance which in turn may be due to angiotensin and sympathetic overactivity.

The haemoglobin level among the patients was quite low. Anaemia is a common finding in renal failure. It contributes to hyperdynamic circulation, left ventricular hypertrophy, left ventricular dilatation and ischaemic heart disease. In HD patients chronic blood loss during dialysis procedure is an added reason along with low erythropoitin activity. Effective therapy with recombinent erythropoictin improves haemoglobin level and reduces left ventricular rnass[20]. None of the patients was getting erythropoietin but received multiple units of blood transfusion which is usually not sufficient for restoration of normal haemoglobin level.

Congestive cardiac failure was present in about one-fourth of the patients. As these patients had left ventricular hypertrophy with normal systolic function, the possible cause of cardiac failure was diastolic dysfunction resulting from ischaemic heart disease[21].

In 20% of the patients, systolic dysfunction was evident in the advance of previously described features of uraemic cardiomyopathy. It may be assumed that coronary insufficiency may be the cause of systolic dysfunction in these patients.

Left Ventricular Hypertrophy (LVH) is present in 30%-80% patients of end stage renal failure as is shown by several studies[22]. This hypertropy may be concentric, eccentric or asymmetric. Concentric hypertrophy is the commonest. It is mainly due to pressure overload and eccentric variety is due to volume overload[23]. Volume overload may result from anaemia, AV fistula and fluid retention. In these-patients, majority had concentric hypertrophy. Probably hypertension played a dominant role in them.

It has been described by different authors that in ESRD patients, hypertension is not the only factor for LVH. Hypertrophy may progress in dialysed patients even when they become normotensive. In contrast, following renal transplantation, LVH may regress even if hypertension persists: So it is postulated that other than hypertension, anaemia, fluid overload, uraemic toxins (like PTH), A-V fistula all may contribute to development of LVH in different proportions in different patients.

In the series, patients of diabetic subset were of older age group and incidence of IHD was also significantly more in this group. Usually onset of NIDDM occurs in older age and diabetic nephropathy develops over a period of 10-30 years of diabetes. These patients developed ESRD after a mean interval of 14-16 years. Patients of primary cause of renal failure may present at any age. So there is significant age difference. The diabetic patients were also hypertensive. Diabetes, hypertension and older age all contributed to increased incidence of ischaemic heart disease. Thus in the diabetic subset IHD is more common than in non-diabetic patients. There is significantly lower duration of HD in diabetics in the second year (Table-III) of our followup. This is because death of diabetic patients during different dialysis duration. Previous reports also showed reduced survival in diabetics on dialysis compared to those with other primary renal disorders. It is about 60% of the non-diabetics[24].

When living and dead are compared, significant increase in septal thickness was found in the later group; LVMI was also increased in this group. Increased LVMI in the dead in comparison to the living made this is an important prognostic marker influencing the mortaltiy as majority died of cardiac and vascular causes. Increase in LV mass as an adverse prognostic factor has been shown by several studies in both renal and non-renal patients[9,10,25]. Silberberg et al showed that LVMI >125g/m^2 had a significantly lower survival than those who had LVMI <125g/m^2[,11]. It has been reported that in patients of ESRD with diabetes, LV hypertrophy is an additional risk actor for mortality and morbidity[26]. This is in conformity with this study as there is 57% mortality in diabetics vs 47% in non-diabetics at the end of second year of this study. In this series all the patients who developed clinically overt failure succumbed to death indicating that clinically overt heart failure is also a predictor of mortality. This is supported by some previous studies[5].

The limitation of this study was that the sample size was small. Other cardiovascular risk factors (lipid profile, coagulation factors etc.) were not evaluated and diastolic function of the left ventricle could not be assessed.

It may be concluded that cardiovascular complications are highly prevalent in patients on haemodialysis. LMVI is an important prognostic marker. Heart failure is a predictor of mortality. Diabetic subset is of higher age group with increased incidence of clinically evident ischaemic heart disease which has affected their survival. It is advisable that echocardiography should be performed at an early stage and then at regular intervals.

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Risk Factors For Recurrent Febrile Convulsion

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Summary:

Febrile convulsion is the most common form of convulsions in children. Parents are usually concerned by the risk of recurrence. The aim of this study was to determine the incidence and risk factors for recurrence. All children between four months and six years, admitted to Dhaka Shishu Hospital with febrile convulsion during July 2001 to June 2002, were enrolled in this study. There were 95 cases of which nine were lost in follow-up; therefore results were analyzed for 86 cases. Eighty seven percent patients were between four and 18 months of age and 63.95% below 12 months. Male-female ratio was 2.07: 1. Generalized convulsion occurred in 95.34% cases. Seizure duration of less than 15 minutes was in 83.72% cases. Family history of febrile convulsions was reported in 20.93% cases. Recurrence within year of follow up occurred in 33.72% of the patients. Factors associated with recurrence were - first episode of convulsion before six months, which was true for 24.13% in recurrence vs. 5.26% in non-recurrence group (P<0.05) and clusters of convulsions within 24 hours of first attack, which was 48.27% vs. 26.31% in recurrence and non-recurrence group respectively (P<0.05).

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Introduction:

Febrile convulsions occur in young children when there is rapid increase in their body temperature. It is defined as epileptic seizures that are provoked by fever of extra cranial infective origin and occur in children aged between six months and five years[1,2]. It may, however, occur from four months up to six years of age[3,4]. Febrile convulsion occur in 25% of all children below five years of age, making it the most common form of seizures in children[5]. It was as high as 9% in Japan and 15% in Mariana Island[6]. If a child has had a febrile convulsion, he or she is prone to more. About four out of ten children who had febrile convulsion will get them again at some stage, although the risk factors differs greatly from child to child[7]. A child is four times more likely to have a febrile convulsion if either parent was affected in their childhood. Children who have their first febrile convulsion before the age of one year has a 50% chance of further seizures[8]. Parents are psychologically traumatized by seeing their child developing the very first febrile convulsion. They become quite concerned about the likelihood of child death during an attack and means to prevent it. Therefore, they often become obsessed about the risk of recurrence and possibility of development of epilepsy in the future. This study was carried out to know the incidence of the recurrence and its risk factors among the children of this country.

Materials and methods:

All children between four months and six years admitted to Dhaka Shishu Hospital with a history of first or recurrent attack of febrile convulsion during July 2001 to June 2002, were included in a prospective study to determine the recurrence rate and the risk factors for recurrence. A study physician who collected the data examined all patients and was supervised by a consultant paediatrician. The data collected include: age at first onset of convulsion, sex, duration of fever, duration of convulsion, description and recurrence of convulsion within 24 hours and family history of convulsion. In addition to physical examination, developmental history, and pre- and perinatal events were also recorded. Lumber puncture along with other

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routine investigations were performed for first attack of convulsion. All patients were followed up every three months for one year. Nine cases were excluded from the study, as follow up could not be maintained due to lack of proper address.

Enrolled patients were divided into two groups according to history of recurrence and the results were statistically analyzed between recurrence and non-recurrence group by doing Z test and finding of P values.

Results:

A total of 95 patients were included in the study, nine cases were excluded. Out of 86 (95 - 9) cases 10 (11.62%) were below six months of age at onset, 45 (52.32%) between six and 12 months, 20 (23.26%) between 13 and 18 months and 11 (12.79%) were above 18 months (Figure-1). Fifty eight patients were male and 28 female (ratio 2.07: 1). Febrile convulsions were generalized in 82 (95.34%) cases and partial in four (4.65%) cases. In 72 (83.72%) cases the duration of seizure was less than 15 minutes and in rest (16.28%) it was more than 15 minutes. More than one attack within 24 hours of onset of fever was found in 29 cases (33.72%). Family history of febrile convulsion was reported in 18 (20.93%) cases.

Recurrent febrile convulsions occurred in 29 (33.72%) cases. Male female ratio in both recurrence and non-recurrence group were similar (Table-1). In recurrence group onset of first convulsion occurred at four months of age in five (17.24%) cases, in contrast to one (1.75%) in non-recurrence group (P<0.05). In 21 (72.41%) cases of recurrence group first attack of febrile convulsion occurred before one year of age and it was true for 3·a (59.65%) in non-recurrence group (P>0.05). In 14 (48.28 %) of recurrence group frequent convulsions occurred within 24 hours of first onset but in non-recurrence group it happened in 15 (26.32%) cases (P< 0.05).

None of the patient had any neurodevelopmental abnormalities.

Table –I

Risk factors for recurrence in patients with febrile convulsion (n=86)

Risk Factors	Children with recurrence (n=29) No (%)	Children without recurrence (n=57) No (%)	P-value
Age at onset:			
4 months	05 (17.24)	01 (1.75)	P<0.05*
5 months	02 (6.90)	02 (3.51)	P>0.05
4 months & 5 months	07 (24.14)	03 (5.26)	p<0.05*
6 months - 12 months	14 (48.28)	31 (54.39)	P>0.05
13 months - 18 months	06 (20.69)	14 (24.56)	P>0.05
19 months - 24 months	00	00	
>24 months	02(6.90)	09 (15.79)	P>0.05
Sex:			
M: F	20: 9(6897: 31.03)	38: 19 (66.67: 33.33)	P>0.05
Types of convulsion:			
Generalized	27 (93.10)	55 (96.49)	P>0.05
Focal	02 (6.90)	2(3.51)	P>0.05
Clusters of convulsion at first attack	14 (48.28)	15(26.32)	P<0.05* Duration of feve
at onset:			
<24 hours	15 (51.72)	30 (52.63)	P>0.05
>24 hours	14 (48.28)	27 (47.37)	P>0.05
Duration of convulsion:			
<15 minutes	25 (86.21)	47 (82.46)	P>0.05
>15 minutes	04(13.79)	10 (17.54)	P>0.05
Family history of convulsion	06(20.69)	12 (21.05)	P>0.05

* Statistically significant

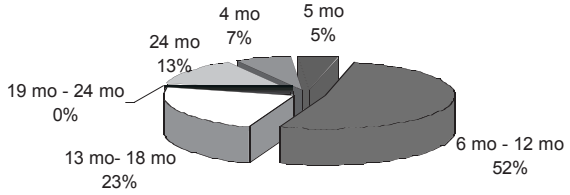

Fig.- 1 : *Age wise distribution of patients with febrile convulsion (n=86)*

Discussion:

Febrile convulsions have long been recognized, but only in recent years more fully understood. Hippocrates, writing in the fourth century BC, described such a convulsion, clearly differentiating it from rigors and breath holding attacks. He noted that both generalized and partial seizures can occur, and realized that there was a strong association with age, high fever and a precipitating infection[8]. Children between the ages of six months and five years are affected; most are at the younger end of the age range. According to Consensus Development Panel it can occur as early as four months of age[3]. In this study in 64% cases it occurred before 12 months of age. Febrile convulsions are found age dependent with a similar distribution curve in several studies[9] including the present study (Figure-I). It was also found that in 10 (11.62.%) cases the subjects were less than six months old, and it was reported less than 06% in the literature[9]. In this study the males outnumbered the females and this finding is similar to those of previous studies[10,11]. For some reasons boys are more likely to be affected than girls.

Simple febrile convulsions occur four times more than complex febrile convulsions[12]. The findings of current study conforms to that. In this study generalized convulsion occurred in 82 (95.35%) cases and partial seizure was found in 4.65% cases. Duration of seizure of less than 15 minutes was found in 83.72% patients and in 16.28 % it was more than 15 minutes. Bessissco et al found seizure duration of less than 15 minutes in 92% cases and more than 15 minutes in 8% cases[9]. Long lasting seizure more than 30 minutes were reported with variable incidence from 18 to 35%[7,13,14,15]. In some studies it was shown that in 70-75% cases the most long lasting seizure was of seven minutes during initial seizures[16,17]. In this study 33.72% had more than one convulsion during 24 hours of first attack. It is reported to be 14% and 16% in two other studies[9,18] . Duration of fever was less than 24 hours in 52.33% cases in this study whereas it was found 75% in another recent study[9].

This study showed that 21 % of patients had family history of febrile convulsion. Different studies found similar incidence (17-22%) among siblings[6,9,19]. Aicardi and Chevrie found an incidence of 31% in first degree relatives[16,20]. The emperic risk for further offspring in a family with one affected child is approximately 10%. Children may inherit the tendency to suffer from febrile convulsion from their parents[21,22]. If either parent suffered from febrile convulsion in childhood, the risk of the child getting it rises by 10 to 20 percent. If both parents and their child have at some point suffered a febrile convulsion, the risk of another child getting it rises by 20 to 30 percent[23]. Febrile seizures are 2-3 times more likely in family members of affected children than in the general population[24]. Most studies suggest a dominant mode of inheritance with reduced penetration and variable expression[24,25,26].

Recurrence of convulsion occurred in 29 patients (33.72%) within a year in this study. Approximately 25 to 37 percent of patient with febrile convulsion will get at least one recurrence[7,27,29,29]. In this study out of 29 cases of recurrent febrile convulsion, five (17.24%) had first episode of convulsion at four months of age and out of 57 in non-recurrence group only one (1.75%) had the episode at this age ($P<0.05$). Recurrence for infants of below one year was 38.18% and above one year 25.81%. The risk of recurrence for infant below one year was 50% and above one year 28% in other studies[8.30.31]. No significant gender difference was found for recurrence, although the boys outnumbered the girls in both recurrence and non-recurrence group. Male to female ratio was 2.2: 1 and 2: 1 in two groups. Patients with more than one attack of convulsion within 24 hours of first onset had recurrence in 48.28% compared to 26.32% in non-recurrence group ($P < 0.05$). Bessissco et al, in their study, found that patients with cluster onset had recurrence in 44% compared to three percent ($P=0.00$) in those without recurrence[9]. Other factors like duration of fever,

duration of scizure, family history of' convulsion were not found to be significant in this study.

This study showed that one third of the patients with febrile convulsion had recurrence and risk factors for recurrence were onset of first febrile convulsion before six month of age and clusters of convulsion within 24 hours of first onset. Patients should, therefore be, properly counselled about the recurrence of febrile convulsion and their immediate management in all cases with particular emphasis to those who has risk factors for recurrence.

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Estimation of Gestational Age by Foetal Biparietal Diameter in Bangladesh

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Summary:

This cross sectional study was done with the objective to prepare a chart to estimate gestational age by biparietal diameter (BPD) measurements from 13 to 41 weeks menstrual age of the foetuses in Bangladeshi population. So far the western charts of gestational age determination from biparietal diameter that were prepared on Caucasian population are being used here. But measurements among eastern population were found to be smaller than the western by various studies. This study was therefore designed to create local population standard for foetal BPD measurements. It was found here that BPD measurement of 23 mm predicted gestational age of 13 weeks, 61 mm predicted 25 weeks, 81 mm predicted 32 weeks, 87 mm predicted 36 weeks and 92 mm predicted 40 weeks.

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Introduction:

Estimation of gestational age by ultrasonography (USG) has now-a-days become an essential part of maternal antenatal care, especially in patients with uncertain dates or verification of dates for patients who are scheduled for elective cesarean section and induction of labour, to avoid preterm newborn. Ultrasound provides accurate dating for gestational age.

Therefore, it is now used widely by the obstetricians and has become one of the major tools to evaluate the growth of developing foetus. Multiple studies have found that accurate assessment of gestational age in the second trimester is possible with the measurement of foetal biparietal diameter[1,2,3]. But the size and growth of Bangladeshi foetuses is less than the reference population whose charts are being used so far in this country[4,5,6,7,8,9].

In a developing country like Bangladesh, maternal malnutrition and consequent low birth weight of the neonate is quite common. In many such cases a timely and early delivery becomes mandatory. A discrepancy of two weeks can be critical for the survival of an infant who has to be delivered early because of some antenatal complication[10].

Therefore, studies are required to create foetal biometry charts of the proposed population as standard. With this objective this study was done to develop a chart to estimate gestational age by biparietal measurement using ultrasonography.

Materials and method:

This cross sectional study was done over a period, starting from March, 1999 to September, 2001.The work was done in a private diagnostic center located in the city of Dhaka

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A total number of 797 consecutive healthy gravid women were studied. Their age ranged between 18 and 30 years.

Inclusion criteria for the study population included regular menstrual history, accurate recalling of last menstrual period (LMP), antenatal care starting before 20 weeks, singleton pregnancy and no oral contraceptive taken three months prior to LMP. Exclusion criteria were maternal malnutrition and any major maternal systemic disease like hypertention, diabetes, severe anaemia, heart or chronic lung disease, foetal anomalies, oligo and polyhydramnios, direct occipito-anterior or posterior position of foetal head, when the proper plane of biparietal diameter (BPD) measurement was not possible to obtain, and when the head shape was not optimum, uterine anomalies like bicornuate uterus or large fibroids, bad obstetric history and substance abuse.

The study group consisted of a cross section of serially scanned healthy gravid women. All patients underwent a complete sonographic examination including measurements of the foetal BPD, femur length (FL) and abdominal circumference (AC) using standard methodology. All measurements were obtained in millimeters (mm). Each patient was studied only once.

All study subjects were Bangladeshis, staying in Dhaka but belonging to different districts of Bangladesh. They were of the same race. Patients of Caucasian and Mongol race were excluded. Majority of the patients belonged to the middle class.

The sonographic examinations were performed with a real-time ultrasound system Aloka SSD 900 of Japan. The probe used was a 3.5 MHz curvilinear transducer. Electronic calipers or digitizer capable of measuring up to 1 mm were used for the measurements. The biparietal diameter (BPD) is the maximum diameter of the foetal skull at the level of the parietal eminences[11]. The correct transverse axial section of the head

demonstrated the following features on ultrasonography : oval shaped head, falx cerebri anteriorly and posteriorly only, cavum septum pellucidum anteriorly in the midline and choroid plexus in the atrium of each lateral ventricle.

Result

Gestational age by foetal biparietal diameter measurement:

Number of Patients	BPD (mm)	Gestational age (weeks)	2 Standard deviation (weeks)
15	23	13.0	1
	25	13.5	1
13	26	14.0	1
	28	14.4	1
19	30	15.0	1
	32	15.3	1
17	34	16.0	1
	36	16.5	1
18	37	17.0	1
	38	17.3	1
26	40	18.0	1
	41	18.2	1
26	43	19.0	2
	44	19.4	2
	45	19.7	2
23	47	20.0	2
	48	20.4	2
24	49	21.0	1.5
	51	21.3	2
15	53	22.0	2
	55	22.5	2
30	56	23.0	2
	57	23.4	2
24	58	24.0	2
	59	24.3	2
30	61	25.0	2
	63	25.3	2
31	65	26.0	2
	67	26.3	2
27	69	27.0	2
	70	27.3	2
28	72	28.0	2
	73	28.4	2
30	74	29.0	2
	75	29.4	2
32	76	30.0	2
	77	30.4	3
33	78	31.0	3
	79	31.3	3
33	81	32.0	3
	82	32.4	3
33	83	33.0	3
33	84	34.0	2
	85	34.4	3
34	86	35.0	3
34	87	36.0	3
34	88	37.0	3
35	89	38.0	3
	90	38.4	3
33	91	39.0	3
34	92	40.0	2
26	92	41.0	3

Total Patient = 797

This chart was prepared by regression analysis of the raw data obtained from 797 subjects. In this study the BPD measurements increased gradually from 13 to 28 weeks at 3 mm per week, then from 28 to 33 weeks it increased by about 2 mm per week, there after up to 40 weeks the increase was about I mm per week.

In this study, when dating a pregnancy between 13 to 26 weeks the predictive value decreases from ± 6 days to ±12 days in 95% of the population. The correlation of BPD with gestational age decreases in the third trimester and the predictive value decreases to ± 3 weeks or ± 14 to 23 days.

Discussion:

In the second trimester the BPD is the most widely accepted means of measuring the foetal head and estimating the gestational age[12]. Biparietal diameter is simple to measure than the other two measurements or parameters that involve the foetal head: corrected BPD and head circumference (HC)[13]. Areas and circumference are much less sensitive than diameter to change in shape but are more difficult to measure. The simplicity of the measurement process makes diameter measurement inherently more reliable[14].

All reports on the BPD have demonstrated it to be an accurate predictor of menstrual age before 20 weeks. A variability of ±1 week (2 Standard deviation) was demonstrated in a population of 1,771 patients with optimal menstrual histories seen between 14 and 20 weeks[15]. After 26 weeks the correlation of BPD with gestational age decreases because of the increased biological variability. The predictive value decreases to ± 3 weeks in the third trimester. The growth of the foetal skull slows from 3 mm per week in the second trimester to 1.8 mm per week in the third trimester[12].

In this study the BPD measurements increased gradually from 13 to 28 weeks at 3 mm per week, then from 28 to 33 weeks it increased by about 2 mm per week, there after up to 40 weeks the increase was about 1 mm per weeks.

In this study, when dating a pregnancy between 13 and 26 weeks the predictive value decreases from ± 6 days to ± 12 days in 95% of the population. Here also, like the above mentioned study, and also other studies the correlation of BPD with gestational age decreases in the third trimester and the predictive value decreases to ± 3 weeks or ± 14 to 23 days. Here it was found that BPD of 92 mm predicts 40 weeks gestational age, but in Kurtz study 92 mm predicts gestational age of 37 weeks 6 days, 96 mm predicts 40 weeks 2 days and 97 mm predicts 41 weeks[16].

This study provides a chart to determine gestational age from BPD measurements of the foetuses in Bangladeshi population. So far the measurements were dependent on western charts for gestational age determination. This study and the chart support the previous studies done on the similar population, and can be helpful for accurate dating of foetuses. However, more such studies are needed on bigger sample size.

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Childhood Nephrotic Syndrome : Rational of Management

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Summery:

We have analyzed the appropriateness of therapy of primary care physicians in 107 children with nephrotic syndrome referred to our institute from January 2001 to December 2003. Prednisolone was administered in adequate doses in 52 (54.73%), and for adequate duration in 40 children (42%). Adjunctive cyclophosphamide therapy was administered in the recommended doses in 72% of cases and duration in 34% of the cases. On evaluation of therapy it was observed that inappropriate treatment had been administered by 26% of the pediatricians, 72% of adult physicians and 81% of general practitioners. This study highlights the lacunae in the current state of knowledge amongst the primary physicians and highlights the need for creating greater awareness regarding the therapy of children with nephrotic syndrome.

(J Bangladesh Coll Phys Surg 2004; 22 : 57-60)

Introduction:

Nephrotic syndrome is an important chronic disorder in children. Minimal Change Disease (MCD) is the commonest histopathological variety[1]. This has an excellent response to steroids. Majority of the cases do not pose therapeutic problems and can easily be managed by the primary physicians if one adheres to the standard protocol[2]. Appropriate therapy helps in minimizing side effects. More over it has now been demonstrated that the adequacy of initial therapy effects the subsequent course of the illness[2,3]. The intensity of initial treatment may decrease the rate of subsequent relapses[4]. This study was done to see the appropriateness of therapy by primary physician in children with nephrotic syndrome in this country prior to their referral to tertiary care centres.

Materials and method:

The children with nephrotic syndrome referred to Bangabandhu Sheikh Mujib Medical University Hospital, Dhaka from the only public sector tertiary care center for paediatric nephrology January 2000 to December 2003 between one and 15 years of age. Children with systemic disease and congenital nephrotic syndrome has been excluded from the study. During this period 107 children with nephrotic syndrome were seen in this institute. The referring physicians comprised pediatricians, adult physicians and general practitioners. During initial evaluation apart from clinical exam detailed drug history was carried out to find out the appropriateness of therapy of the referring physicians, in terms of dose and the duration of prednisolone and cyclophosphamide. The currently recommended treatment protocol by the ISKDC for initial episode i.e. 2mg /kg (max 60mg) in two-three divided doses daily for 6 weeks, followed by 1.5mg / kg (max 40 mg), as a single morning dose on alternate day, for the next 6 weeks[9].

A relapse is treated with same drug in a dosage of 60mg/kg/m^2/ day till remission (documented for 3 days) followed by 40mg/m^2/alternate day x 4 weeks[3,5-8]. Cyclophosphamide administered in dose of 2mg/kg/day for 8 to 12 weeks in frequent relapsers and steroid dependent patients respectively was taken as standard for comparison[9]. Inappropriate therapy means any other regimen in terms of dose and duration beyond above mentioned protocols.

The name, address and qualifications of the referring physicians were duly recorded. Urine examination

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and blood biochemical investigations were done in all the patients. The diagnosis of nephrotic syndrome was based on the standard ISKDC criteria[10].

Kidney biopsies were carried out if:

i) the age was less then one and more then 10 years;.

ii) there is no response to four weeks steroid therapy;

iii) there is unusual clinical features (sustained hypertension, gross haematuria) or there is hypcomplementemia, elevated blood urea and creatinine.

iv) there is persistent microscopic haematuria; and before starting treatment with cyclosporin A.

Based on their subsequent response to adequate steroid therapy, these patients were categorized as frequent relapsers (FR), Steroid dependent (SD), initial non- responders (INR) and subsequent non- responders using standard case definitions[6].

Results:

Of 107 children there were 70 boys, 37 girls. The age of onset of disease was 4.2 years (range 1- 15 years), and the mean age at referral was 5.6 years (1.2 - 15years). The duration of followup at the institute ranged from one to 20 months (mean 16±42 months). Of the 107 children, 103 (96%), had received steroids prior to referral while 23(21°l0) had also been treated with oral cyclophosphamide. Of these, accurate details of previous therapy regarding duration and doses were available in 95 children. The distribution of these children based on the adequacy of dose and duration of the previous steroid therapy is shown in Fig 1. Only 52 (54.73%) children had received steroids in appropriate doses and 40(42%) had received therapy for appropriate duration prior to the referral.

In contrast inadequate therapy in terms of dose and duration had been given in 39 (41 %) and in 35(36.84%) children, respectively. Another three (3.1%) children had received excessive doses and 21 (22%) received therapy for prolonged periods. The common steroid side effects observed were Cushingoid appearance (67.6%), hypertension (26%), gastrointestinal symptoms (7.2%) and growth failure (6%).

None of them had shown haematuria following cyclophosphamide therapy (hemorrhagic cystitis). Of the 107 children, 23 had received adjunctive cyclophosphamide therapy prior to referral. In this group adequate doses were administered in 17 (72%) subjects, where the duration was optimum in only eight (34%) cases. Five (21.73%) children had received therapy for longer duration and three in excessive doses.

The qualified medical practitioners had referred all the children to us. General practitioners accounted for only 32.63% (n=31), while majority of children were treated by pediatrician 36.84% (n=35), and 30% (n=29)% adult physicians, prior to referral. The appropriateness of therapy of the referring physicians in terms of dose and to duration is depicted in Fig.-2. Of the 35 children in whom paediatricians were the primary physicians, treatment was appropriate in 26(74%), while nine (26%) had received inappropriate (inadequate /excessive) therapy. In contrast, appropriate therapy had been administered in only 19% cases by general physicians. Twenty one (72%) of cases treated by adult Physicians was inappropriate.

Children were classified according to their response to steroid therapy and infrequent relapsers constituted 33% of the cases. Frequent relapser comprised 23.75% of children while another 35% were steroid dependent. Initial non-responders and subsequent non-responders accounted for 5.6% and 03% respectively.

Kidney biopsy was done in 42 children. Minimal change disease (MCD) was the commonest histopathological lesion in the patient with idiopathic nephrotic syndrome, accounting for (62%) of the biopsied cases. The other common causes were focal segmental glomerulosceerosis (26%), membrano-proliferative glomerulonephritis (7.14%) and mesangial proliferative glomerulo nephritis (4.76%).

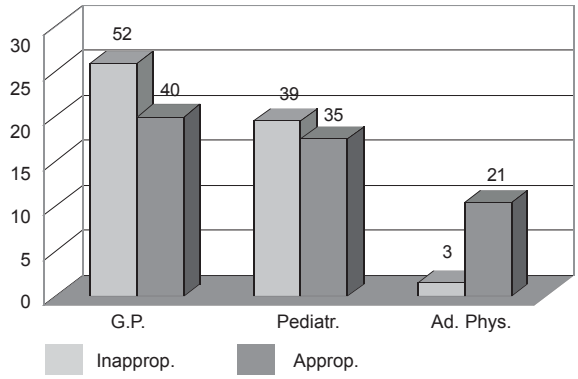

Fig.-1 : *Dose and duration of steroid therapy administered by primary physicians in children with Nephrotic Syndrome.*

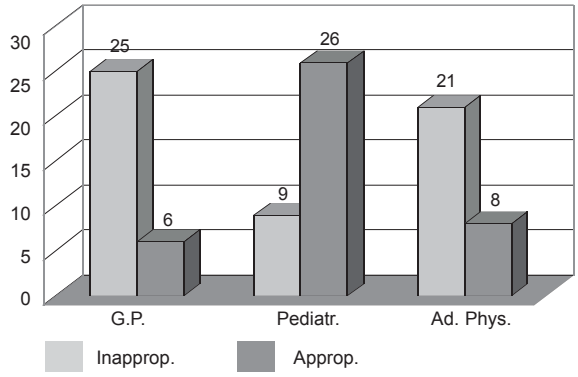

Fig.-2 : *Appropriateness of therapy of the primary physicians.*

Discussion

In children MCD predominates where other histological entities are rare[11]. Cortocosteroid are considered to be the drug of choice in MCD. In the ISKDC study it was found that by the end of eight weeks, there were only about 08% of children who did not respond to steroids[10]. The primary care physicians can easily manage majority of the cases. Steroids and cyclophosphamide are both potentially toxic drugs. Inadequate doses on the other hand may not only be associated with a lower response rate but also adversely effect the course of the illness.

Current evidence suggests that treatment of the initial episode influence the subsequent courses of the illness. The intensity of initial treatment may decrease the rate of subsequent relapse and long lasting subsequent remission[6,9]. Thus, it is imperative for the treating physician to adhere to the standard protocol.

The spectrum of idiopathic nephrotic syndrome in this series is similar to that reported from western countries[12].

Data regarding steroid therapy prior to referral shows only 54.73% of children had received steroid therapy in standard doses and 42% for adequate duration. Physicians had a tendency to use repeated courses of steroids in inadequate doses for shorter periods resulting in greater side effects and lower response rate. This could account for the fact that the majority of the patients behaved as steroid dependent. A large proportion of these referred patients could otherwise have been easily managed at the primary level had they been treated with appropriate regimens. Data regarding previous cyclophosphamide therapy was analyzed and it was found that therapy in recommended doses and duration had been given in 72% cases and 34% cases, respectively.

Three of them had been administered cyclophosphamide in doses exceeding the gonadotoxic dose (>304mg/kg)[13].

Qualified medical practitioners had referred all the children. Majority of them had been referred to us by general practitioners (32.63%) and (36.84%) pediatricians, respectively.

On evaluation of the therapy by different physician prior to the referral it was found that treatment was inappropriate by 81% general practitioners and 72% adult physician.

These finding highlights the lacunae in the current state of knowledge amongst the primary care physicians and underscores the need for greater awareness regarding the therapy of children with nephrotic syndrome. With this awareness one can decrease the frequency of drug related side effects and lower the frequency of relapses.

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REVIEW ARTICLE

Results of Coronary Bypass Surgery in Diabetic Patient- A Review

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(J Bangladesh Coll Phys Surg 2004; 22 : 61-65)

Introduction

Coronary atherosclerosis is more prevalent among diabetic than non-diabetic patients. In addition in diabetic patients it is more extensive and takes an accelerated course.[1-3] The reported prevalence of diabetes among patients undergoing coronary artery bypass grafting (CABG) ranges from 7% to 20%.[1.4.5]

Diabetes mellitus is an established risk factor for the development of coronary artery disease. Epidemiological data from the Framingham study [6] have shown that diabetes is a major independent risk factor for cardiovascular disease even after adjusting for other confounding risk factors such as age, hypertension, hypercholesterolemia, and tobacco abuse. Further, the incidence of congestive heart failure and cardiovascular death are even higher in female diabetic patients than in male diabetics.[7] Diabetic patients have a higher incidence of two and three-vessel disease and a lower incidence of one vessel disease than do nondiabetic patients.[8] Diabetes has been associated with higher perioperative morbidity as well as decreased survival after coronary artery bypass grafting.[9-11]. The present article reviews the underlying causes of higher perioperative morbidity and mortality in diabetic patients and the current researches on this issue to address this problem.

Study-1

Lawrie et al (1986) underwent a long term follow up study of 1,434 patients with CABG done between 1968 and 1973, 212 of whom were diabetic. All patients underwent isolated saphenous vein aortocoronary bypass graft procedures on one surgical service. Of 212 diabetics, 87(41%) patients were receiving no drug treatment, 108(51%) patients were being treated with oral hypoglycemic agents, and 17(8%) patients were receiving treatment with insulin. There were no significant difference in the demographic profiles, number of major proximal coronary lesions, left ventricular function between the diabetic and nondiabetic groups. Perioperative mortality (30 day) showed no significant differences between the two groups especially due to cardiac causes, which accounted for 60% (9/15) of mortality in the diabetic patients and 70.9% (39/55) mortality in nondiabetics. The causes of late mortality were however significantly different between the two groups. There was excessive mortality from cardiac causes, stroke, and renal causes in the diabetic group particularly in the insulin-treated patients. The results of Kaplan-Meier analysis of survival probability indicated that the diabetics had significantly lower long-term survival probability than nondiabetics (p<. 05). This difference was observed also when patients were stratified according to preoperative left ventricular function (p<. 05). Diabetics receiving no drug therapy had survival identical to that of normal patients, while patients receiving insulin therapy had the worst survival[12].

Comment

The severity of diabetes mellitus before surgery influenced the long-term survival rates negatively after coronary by-pass surgery particularly in those patients who were taking insulin. The 30-day mortality between the diabetic and non-diabetic was however comparable.

Study- 2

Cohen et al performed a prospective national cohort study, which included patients who underwent CABG in 14 medical centers in Israel during 1994. The purpose of that study was to identify factors associated with 30-day mortality after CABG among diabetic patients, and to compare them with risk

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factors among nondiabetics. Multivariate logistic regression analysis was used to identify factors associated with a 30-day mortality in diabetic and nondiabetic populations. The results showed that crude mortality was 5.0% among diabetic patients (n=1,034) and 2.5% among nondiabetics (n=3,350; p< 0.001). Crude mortality rates by gender in nondiabetics were 2.3% for men and 3.5% for women (p=NS); among diabetic patients, crude mortality rates by gender were 3.7% for men and 8.3% for women (p=0.002). Multivariate logistic regression analysis identified female gender, 3-vessel disease, and left main disease as independent risk 30-day, post-CABG mortality unique to diabetic patients, left ventricular dysfunction was found to effect a greater risk among diabetic patients, whereas chronic renal failure was associated with greater risk among nondiabetics.[13]

Comment

In contrast to the previous study the study conducted by Cohen et al demonstrated that 30-day mortality is significantly higher in diabetics than their nondiabetic counterpart particularly in female diabetic patients, and patients with 3-vessel disease, left main disease and patients with left ventricular dysfunction.

Study –3

Thourani et al did a short and long term follow up study on 12,198 patients who underwent routine CABG operation between 1978 through 1993 at Emory university hospital. During the 16 years, 9,920 of these patients were classified as nondiabetic and 2,278 as diabetic according to the definition given by American college of cardiology. Compared with nondiabetic patients, the group with diabetes was older (62±10 years versus 60±10 years), comprised more women (31% versus 19%), had a greater incidence of hypertension (61% versus 44%) and previous myocardial infarction (51% versus 48%), had class III-IV angina more commonly (69% versus 63%), showed a higher incidence of congestive heart failure (11% versus 5%) or triple-vessel or left main disease (60% versus 50%), and had lower ejection fractions (0.54 versus 0.57) (all, p£ 0.05). Diabetic patients had a higher incidence of postoperative death (3.9% versus 1.6%) and stroke (2.9% versus 1.4%)(both, p£ 0.05), but not Q wave myocardial infarction (1.8% versus 2.9%). Diabetics had lower survival (5 years, 78% versus 88%; 10 Years, 50% versus 71%; both, p£ 0.05) and lower freedom from percutaneous transluminal coronary angioplasty (5 years, 95% versus 96%; 10 years, 83% versus 86%; latter, p£ 0.05), but diabetics did not have lower freedom from either myocardial infarction (5 Years, 92% versus 92%; 10 years, 80% versus 84%) or additional coronary artery bypass grafting (5 years, 98% versus 99%; 10-years, 90% versus 91%). Multivariate correlates of long-term mortality were diabetes, older age, reduced ejection fraction, hypertension, congestive heart failure, number of vessel diseased, and urgent or emergent operation.[7]

Comment

This study clearly established the higher preoperative risk factors, early and late morbidity and mortality in diabetics than their nondiabetic counterpart.

Study: 4

Magee M J et al compared the influence of diabetes on mortality and morbidity between off-pump CABG and CABG with cardiopulmonary bypass. A total of 9,965 patients, from January 1995 through December 1999, of whom 2,891(29%) had diabetes, underwent isolated CABG. Twelve percent (346 of 2,891) of diabetic patients and 12% (829 of 7074) of nondiabetic patients underwent CABG without cardiopulmonary bypass. Patients undergoing CABG without cardiopulmonary bypass compared with those having CABG with cardiopulmonary bypass had higher mean predicted mortalities (diabetic, 3.96% versus 3.72%, p=0.83; non-diabetic, 3.03% versus 2.86%, p=0.79). In nondiabetic patients, coronary artery bypass grafting without cardiopulmonary bypass provides an actual and risk-adjusted survival advantage over coronary artery bypass grafting with cardiopulmonary bypass (1.81% versus 3.44%, p=0.0127; risk-adjusted mortality, 1.79% versus 3.61%, p=0.007). The survival benefit of coronary artery bypass grafting without cardiopulmonary bypass was not seen in diabetic patients (2.89% versus 3.69%, p=0.452; risk-adjusted mortality, 2.19% versus 2.98%, p=0.42) Diabetic patients undergoing coronary artery bypass grafting without cardiopulmonary bypass had fewer complications, including decreased blood product use

(31.39% versus 58.4%, p=0.001), and reduced incidence of prolonged ventilation (6.94% versus 12.10%), p=0.005), atrial fibrillation (15.90% versus 23.26%, p=0.002), and renal failure requiring dialysis 10.87% versus 2.75%, p=0.036).[14]

Comment

The survival advantage in nondiabetic patients treated with coronary artery bypass grafting without cardiopulmonary bypass was not apparent in diabetic patients. Coronary artery bypass grafting without cardiopulmonary bypass in diabetic patients was nevertheless associated with significant reduction in morbidity.

Study: 5

Szabo et al in Sweden studied the early post-operative outcome and medium-term survival in diabetic and nondiabetic patients undergoing CABG.

A total of 2,779 consecutive patients undergoing isolated CABG during 1999 were studied, 19.4% of whom had diabetes mellitus. Demographic and peri-procedural data were registered prospectively in a computerized institutional database.

The diabetic group was younger and included a higher proportion women, and patients with hypertension, triple- vessel disease, unstable angina. They required a higher number of bypasses, and a longer cross clamp and cardiopulmonary bypass times. Intensive care of hospital stays were prolonged and for inotropic agents, hemotransfusions, and dialysis was higher in the diabetic group. Renal failure, stroke (4.3%versus 1.7%), mediastinitis, and wound infections were more frequently encountered. Thirty-day mortality was 2.6% versus 1.6%(p=0.15). Cumulative 5-year survival was 84.4% versus 91.3%($p < 0.001$).

Comment

Short-term mortality was acceptable in diabetic patients after CABG but they had increased postoperative morbidity in comparison with nondiabetic patients, particularly with regard to renal function, cerebral complications, and infections. Midterm survival was impaired in diabetic patients mainly because of a less favourable outcome of patients treated with insulin.[15]

Discussion

The above five studies clearly established the unfavorable outcome in terms of morbidity and mortality in diabetic patients than their nondiabetic counterparts. Several studies have been done to determine the underlying cause of unfavourable outcome of diabetic patients undergoing CABG compared to non-diabetic patients. Verma et al showed that increased endothelin-1 production in diabetic patients after cardioplegic arrest and reperfusion impairs coronary vascular reactivity. Hyperglycemia is a potent stimulus for endothelin-1 production. The coronary effluent release of endothelin-1 is higher in diabetic than in nondiabetic patients after cardiopulmonary bypass and reperfusion, diabetic coronary micro vessels respond to bypass and reperfusion with greater endothelin-1 mediated vasoconstriction and diminished nitric oxide-mediated vasodilatation, and these effects are attenuated by endothelin antagonism. The diabetic heart elicits an exaggerated response to ischemia-reperfusion, with altered neutrophil adhesion, endothelial dysfunction, myocyte contractility, oxidative stress, and myocardial energetics.[16]

Endothelin-1 is one of the most potent vasoconstrictors known and has been implicated in the development of a number of cardiovascular diseases, including congestive heart failure, pulmonary hypertension, endothelial dysfunction, atherosclerosis, and vasospasm.[16]

Verma et al in another study showed that cardiomyocytes might also produce endothelin-1, which might directly impair myocyte contractility by increasing intracellular calcium levels. This impairment of endothelium and cardiomyocyte could be antagonized by endothelin receptor blockers (BQ-123 and Bosentan)[17].

The mature 21 amino acid peptide endothelin-1, or ET-1, is synthesized from a 38 amino acid precursor, known as “big endothelin.”Big ET” is then converted to the biologically active ET-1 by an ET converting enzyme. The diverse physiologic actions of ET-1 appear to be mediated through two receptor subtypes, the ET_A and ET_B receptors.

The production of ET was first described in endothelial cells, but the synthesis of ET has now

been identified to occur in a number of cell types including smooth muscle cells and cardiac myocytes[18]. ET causes potent vasoconstriction of several vascular systems[18]. Potent constriction of vascular smooth muscle occurs primarily through binding of ET to the ET_A receptor and through several intracellular signaling events, increases calcium availability to the contractile elements. The ET_B receptor contributes to the regulation of vascular smooth muscle tone in several different ways. First, ET_B receptors located on endothelial cells mediate vasodilatation via the release of nitric oxide and prostacyclins. Second, this receptor subtype also exert vasoconstriction when located on the smooth muscle cells. Therefore contractile effect of ET depends mainly on the relative density of ET_A and ET_B receptors on smooth muscle cells and of ET_B receptors on endothelial cells. The vasoconstrictive effects of ET are more pronounced in arteries with atherosclerotic disease, ET amplifies coronary artery constrictions induced by nor epinephrine, serotonin.[18]

Whereas the vasoconstrictive effects of ET are widely recognized, activation of the ET_A receptor has direct effects on myocyte biology, including contractile protein interactions, inotropic state, protein expression, and electro physiology.

Fundamental intracellular events that have been reported to occur after ET_A receptor activation are the release or release and mobilization of intracellular calcium (Ca^{+2}) and intracellular pH changes[18]. Since ET_A receptor activation can cause increased release of intracellular Ca^{+2}, then activation of this receptor system after cardioplegic arrest and reperfusion would potentially exacerbate intracellular Ca^{+2} homeostasis and contractile function. An important sarcolemmal exchange system that directly influences intracellular pH is the Na^+/H^+ exchanger. Intracellular pH under normal ambient conditions is maintained relatively alkaline when compared with the environment; this is achieved through the transport of protons out of the myocyte. This exchange system has the capacity to correct an intracellular acid load during periods of ischemia through the acceleration of H^+ extrusion and intracellular accumulation of Na^+. This increased intracellular Na^+ can in turn, increase the exchange rate of the Na^+/Ca^{+2} exchanger with a subsequent accumulation of intracellular Ca^{+2}. Several studies have demonstrated that the Na^+/Ca^{+2} exchanger directly contributes to the increased influx and accumulation of Ca^{+2} during ischemia and reperfusion. Thus during early reperfusion, intracellular Ca^{+2} homeostasis could be further aggravated by the activation of the Na^+/H^+ exchanger[18].

A three to six fold increase in systemic levels has been documented to occur immediately after cardioplegic arrest and reperfusion. A number of clinical studies have demonstrated that increased ET levels persist well during the postoperative period. Potent and specific ET_A receptor antagonists, as well as combined ET_A/ET_B receptor antagonists, have been described. These new nonpeptide ET receptor antagonists are constructs with significant bioavailability, prolonged half-life, and high specificity. ET receptor antagonists have been successfully used in patients with pulmonary hypertension and heart failure. These ET receptor antagonists have been successfully used in several animal models of cardioplegic arrest and CPB. The ET receptor antagonist with greatest clinical profile to date is the nonselective antagonist bosentan. Specifically, the immediate administration of bosentan in patients with heart failure has provided favorable effects on systemic homodynamic and pulmonary hypertension.[18]

Conclusion

Increased endothelin release and activation is an important cause of unfavorable outcome in diabetic patients undergoing CABG, and the use of endotheline antagonist may be a novel approach to counteract this problem.

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CASE REPORTS

Diagnostic Dilemma of Pelvic Tuberculosis: Case Reports and Review of Literature on Clinical Presentations and Diagnosis

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Summary

Tuberculosis of the genital tract was diagnosed in six patients in the department of obstetrics and gynaecology, unit IV, at Dhaka Medical College Hospital from April 2002 to March 2003. All the cases presented with ascities, abdominal discomfort, ovarian mass, abdominal or pelvic pain, weight loss and were misdiagnosed as ovarian carcinoma. None had relevant past history. Tuberculosis was diagnosed on histological evidence in all six cases, three by endometrial biopsy and three from omental and peritoneal biopsy. As tuberculosis is common in tropical countries like ours it may be concluded that pelvic tuberculosis should be considered as a differential diagnosis of cases with pelvic mass and ascities.

(J Bangladesh Coll Phys Surg 2004; 22 : 66-70)

Introduction

Approximately one third of world population (some 1.9 billion) is infected with Mycobacterium tuberculosis[1]. The global case fatality rate is 23 percent but exceeded 50 percent in some African countries with high HIV infection (Human Immuno Deficiency Virus) rates. Over 95 percent of new tuberculosis (TB) cases and deaths due to the disease occur in developing countries and highest incidences are in Asia and Sub Saharan Africa[1]. Although TB is uncommon in developed countries, its prevalence, especially that of extra pulmonary tuberculosis is increasing world wide[2,3]. Demographic factors such as urbanization, increased travel and migration from endemic areas are contributing to the spread of tuberculosis world wide[4]. Increasing prevalence of HIV infection with TB being one of the opportunistic infection is a major factor in the tuberculosis epidemic in many regions particularly in Africa and Asia.

Globally some 900 million women of reproductive age are infected with Mycobacterium tuberculosis and at least 2.5 million develop active disease each year[5]. Tuberculosis is a leading cause of death among women of reproductive age and accounts for 9% of female deaths world wide [5]. Genital tuberculosis is nearly always secondary to a focus elsewhere in the body, usually in the lungs. Five to 13% of patients with pulmonary TB develop genital infection[6,7]. Genital tuberculosis appears to be an uncommon disease but its incidence is likely to increase as the tuberculosis epidemic progresses and therefore continues to be a problem for women[8]. The actual frequency of tuberculosis of the female genital tract cannot be determined accurately, as a large number of patients remain undiagnosed. At the same time it is difficult to diagnose because genital tuberculosis is often a disease with absent or few symptoms, and the symptoms are often nonspecific[9]. Moreover, presentations may be atypical mimicking the conditions like ovarian and endometrial malignancy, pelvic abscess and even carcinoma of cervix[10]. Over one year period from April 2002 to March 2003 six new cases of genital tuberculosis were diagnosed in the Maternity Unit-IV, Department of Obstetrics & Gynaecology, Dhaka Medical College Hospital . As illustrated in the cases below, the presentation of these patients were not characteristics and these created a diagnostic dilemma. Consequently, an array of investigations were carried out and often surgeries were performed before the diagnosis of TB was arrived at. None had a family or positive past history, and diagnosis were made on histological evidences. The details are summarized in tables.

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Case-1

A 24 years old lady of para-2 from a lower middle class family presented with lower abdominal pain and

gradual abdominal distention for one month along with anorexia, weight loss, low grade fever and sweating for one and a half months. Her menstrual cycle was regular except for the last menstruation which was heavy in flow and persisted for about 10 days. Pelvic examination findings were unremarkable.

Her erythrocyte sedimentation rate (E.S.R) was 65 mm during first hour. Chest radiograph was normal. Mantoux test was negative. CA 125 was 60 ku/L. Ultrasonography of lower abdomen revealed normal findings except presence of moderate ascites and a small right ovarian cyst (4.5X2.7 cm). Peritoneal fluid tapping revealed the ascites to be exudative. Diagnostic endometrial curettage was performed in the first day of menstruation which revealed presence of granulomas with epitheleoid cell. The tissue was positive for Ziehl-Neelsen staining but culture for Mycobacterium tuberculosis of the peritoneal fluid was negative. Antitubercular drugs were started and the clinical responses were good.

Case-2

A 33 years old lady of para-3 from a middle class family presented with dull aching abdominal pain for eight months, weight loss, anorexia and intermittent pyrexia together with menorrhagia for six months. Pelvic examination findings were unremarkable.

Her ESR was 80 mm during first hour. Chest radiograph revealed right sided mild pleural thickening. Mantoux test was positive. CA 125 was 317 Ku/L. Ultrasound examination of the abdomen and pelvis revealed irregular mass around the left ovary (supposedly inflammatory). Right ovary was normal with moderate ascites. Ascitic fluid was exudative in nature and cytological examination did not reveal any malignant cells. Diagnostic endometrial curettage was performed and revealed presence of granuloma with epitheleoid cells.

Both Ziehl-Neellsen staining and culture were negative for Mycobacterium tuberculosis. The patient responded well with antituberculous therapy.

Case-3

A 35 years old lady of para-3 from a poor family presented with intermittent lower abdominal pain for one year and feeling of a non tender mass in right lower abdomen, weight loss and anorexia for one and a half months. There was no history of menstrual disturbance. Abdominal examination revealed a small mass (5/7 cm) in right iliac fossa, non-tender with restricted mobility, firm in consistency and the margins were not clearly defined. A firm right sided adnexal mass was found on vaginal examination. Apart from a raised ESR of 70 mm in first hour, all other laboratory tests and skiagram of chest were normal. Ultrasound scan of the pelvis showed minimal ascites and right adnexal complex mass. The clinical suspicion of ovarian carcinoma guided laparatomy.

The operative findings were dense adhesions with the formation of an omental cake, greenish-brown ascitic fluid and multiple miliary seedlings studded over the omentum, peritoneum, liver and undersurface of diaphragm. The pelvic organs were matted and covered in dense adhesions. The fallopian tubes were found to be dilated and inflamed. Both ovaries were otherwise normal. Histological examination of omental and peritoneal tissue showed multiple granulomas with numerous Langhan's giant-cells; both Ziehl-Neelsen staining and culture were negative for Mycobacterium tuberculosis.

The patient was with antituberculous therapy with good clinical response when reviewed two months later.

Case-4

A 19 years old poor woman, married for two years with no history of child birth presented with pain and swelling in lower abdomen for one month, anorexia, marked weight loss, intermittent rise of temperature and lethargy for the same duration. She was amenorrhic for previous seven months. Previously, her menstrual cycle was regular. She was grossly emaciated and developed spikes of temperature and acute abdomen. Abdominal examination revealed a cystic tender mass of about 24 weeks pregnancy size. On vaginal examination the mass was found to be separated from uterus.

Her ESR was 58 mm during first hour. All hematological and biochemical investigations including CA 125 and X-ray chest were normal. Ultrasound examination of the abdomen and pelvis revealed a large mass (1O X 15 cm) possibly

mucinous cyst adenoma of ovary with huge ascites. A clinical suspicion of primary ovarian carcinoma guided lapartomy. The operative findings were dense adhesions with a large pseudocyst occupying the lower and upper abdomen. The cyst got ruptured at the time of dissection and a brownish purulent fluid came out. The pelvic organs were matted and covered in dense adhesions. Both tubes were dilated and distorted. Both ovaries were inflamed.

Histological examination of the omental and peritoneal tissue confirmed the diagnosis of granulomatous lesion tuberculosis. The patient responsded to antituberculous therapy.

Case-5

A 16 years old unmarried school girl presented with the complaints of distention of whole abdomen, rise of temperature and loss of body weight for two months. She had normal menstrual cycle with average flow. She used to take chocolate milk. On abdominal examination, her abdomen was hugely distended. No mass could be separately palpable. Her ESR was 40 mm in first hour. IgM for Mycobacterium tuberculosis was negative. CT scan suggested as a case of ovarian neoplasm. Ascitic fluid could not be aspirated. So, a decision was made for laparotomy. On laparotomy, the omentum and intestines were found matted together and there was a big pseudocyst. There were miliary seedling all over the peritoneal cavity. Pelvic organs were healthy. Biopsy was taken from the omentum and peritoneum of granulomatous tuberculosis. Four-drug anti tuberculosis regime was started and the patient responded well and she was completely afebrile within seven days following surgery.

Case-6

A 35 years old, divorced working woman from upper middle class family with history of a live pregnancy presented with complaints of evening rise of temperature for one month, weakness, loss of interest work, weight loss and menorrhagia for one cycle. On general examination, her temperature was 101°F. Abdominal and vaginal examination revealed no abnormality. Her ESR was 40 mm in first hour. Skiagram of chest was normal and lgM for Mycobacterium tuberculosis was positive. Diagnostic dilatation and curettage of endometrial cavity was done and histopathology proved tuberculosis. Four-drug anti tuberculer therapy was started and the patient showed improvement within a week.

Treatment regimen :

WHO recommended antitubercular drug regimen were suggested for all patients after histodiagnosis. Treatment was continued for six months according to following schedule

Tab. Rimstar 4FDC (each tablet contains Rifampicin 150 mg + Isoniazid 75 mg + Pyrazinamide 400 mg + Ethambutol 275 mg) - three tablets daily ½ hour before breakfast for first two months. Then Tab. Rimactazid 450 mg (each tablet contains Rifampicin 450 mg + 300 mg Isoniazid + Sodium Lauryl Sulphate) one tablet daily ½ hour before breakfast, continued for another four months.

Follow up

Response to treatment was assessed as subsidence of acute symptoms like pain and fever and development of a sense of well being and similar other indicators. The patients were followed up monthly for three months and then bimonthly.

Table-I

Summary of characteristics of patients with genital tuberculosis

		Numbers (total=6)	Percentage
Age in years	<20	2	33.34
	21-30	1	16.66
	31-40	3	50.00
Parity	0	2	33.34
	1	1	16.66
	2-4	3	50.00
Infertility		1	16.66
Menstrual	None	3	50.00
disturbances	Amenorrhoea	1	16.66
	Menorrhagia/ irregular cycle	2	33.34

Histopathological Examination of the specimens confirmed the diagnosis

Table-II

Summary of clinical presentations of patients with genital tuberculosis

Clinical findings	Numbers (total=6)	Percentage
Abdominal pain/distention	5	83.30
Weight loss	4	66.64
anorexia	5	83.30
Exudative ascites	5	83.30
Pelvic /adnexal masses	4	16.66
Pleural effusion	0	0
Swinging temperature	4	66.64
Lathargy	3	50.00
CXR		
-Normal	5	83.30
-Pleural thickening	1	16.66
Laboratory findings		
- Raised ESR	6	100.00
- Raised CA 125	3(3 not tested)	50.00
Mantoux test		
- Not tested	4	
- Positive	1	
Negative	1	

CXR - chest X-ray, ESR - erythrocytes sedimentation rate, CA carcinogenic antigen

Table-III

Means of diagnosis in patients with genital tuberculosis

Means of diagnosis of tuberculosis	Numbers (total=6)	Percentage
Operative procedure		
Endometrial biopsy	3	50.00
Laparotomy with biopsy (omental)	3	50.00
Operative findings		
Adhesion/frozen pelvis	2	33.32
Miliary seedling/nodules	1	16.66
Positive histological diagnosis	6	100.00
Positive cytoloigcal diagnosis	1	16.66

Discussion

Bangladesh is one of the highest tuberculosis prevalent country in the world. As tuberculosis of the female genital tract often manifest in an atypical manner, diagnosis may be missed if the entity is not considered in the differential diagnosis. Pelvic tuberculosis classically present with infertility, pelvic pain and poor general health with menstrual disturbances[11]. However, the above cases demonstrate unusual presentations of pelvic tuberculosis, which often leads to a diagnosis of pelvic malignancy. The diagnosis of tuberculosis was not suggested by history, physical examination or chest X-ray as it was found in previous reports[7,12,13,14]. Fortunately, three patients were suspected before laparotomy. In all six cases, anorexia, weight loss, abdominal swelling discomfort and ascitis were the common presentations. Pelvic mass with moderately raised CA 125 and ascitis lead to initial diagnosis of ovarian cancer, thus providing difficulties of making, diagnosis on clinical evidence. Infertility is said to be one of the common presentation of pelvic tuberculosis. [15,16]. In this series only one out of six cases had infertility of two years. This is similar to the finding of another study[12]. However other classical presentations of pelvic tuberculosis including abdominal or pelvic pain, poor general health were common among the women in this series[12,17,18].

Out of six cases, three had normal menstruation which is comparable with findings of two other studies which reported that almost 87.7%[9] and 78%[12] of the women with genital tuberculosis had normal menstruation. Only one women in this series had amenorrhoea and one presented with menorrhagia

Positive past history or detailed contact history may be helpful, but interestingly none of the patients had such histories. An Indian study reported 85% of the patients had positive history of tuberculosis[16]. Other studies reported only 10 to 25% association.[19,20]

All the women in this series belonged to reproductive age group, similar to the previous reports[12,13,17]. Age incidence varies between developed and developing countries. In developing countries pelvic tuberculosis is common between the age of 20 and 35 years whereas in developed countries, most patients are above the age of 40 years[12].

Endometrial biopsy were taken from three cases who presented with menstural disburbances, and evidence of endometrial tuberculosis was found in all these cases. Although endometrial biopsy can provide a diagnosis of genital tuberculosis with high specificity, its low sensitivity of at most 50% makes it unsatisfactory as a screening test[21].

In this small series both culture and staining have been inconsistent in diagnosing tuberculosis and the findings are consistent with that of another study[12]. All the cases were diagnosed on histological evidence of the diseases. Isolation of Mycobacterium tuberculosis from genital tract is the confirmatory evidence for the diagnosis of genital tuberculosis although histological diagnosis by demonstration of granulomas has been universally accepted[12,17,21]. This is because non-tuberculous causes of granulomas such as sarcoidosis, brucellosis and foreign body reaction can usually be ruled out. Tuberculin skin test is not a useful diagnostic aid and only suitable as screening test[22].

Pelvic tuberculosis should be considered in the differential diagnosis of all cases presented with ovarian masses and ascitis, specially where the disease is common. Correlation of history and clinical findings with laboratory aids may help to have an accurate diagnosis and would prevent unnecessary surgery.

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Obstructed Stammer's Hernia: A Rare Case Report

S AHMAD

Summary

A male of 20 years presented with features of acute intestinal obstruction of small gut. He gave history of similar attack for several times in the last 13 years. On laparotomy a portion of viable small gut was found to be herniated through a defect in the mesentery (stammer's hernia). Closure of the defect was done and postoperative period was uneventful.

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Introduction

Stammer's hernia (herniation through a defect in the mesentery) is a type of internal hernia and internal hernias are rare but are important cause of intestinal obstruction (0.2-0.9% of all cases)[1]. They are often undiagnosed before emergency laparotomy and not uncommonly lead to gangrene necessitating bowel resection of varying extent and this may contribute to high morbidity and mortality.

Case report:

A 20 years old male presented with pain in the abdomen, vomiting and gradually distending abdomen for two days. Pain was sudden, severe, central abdominal, colicky and gradually increasing in intensity. He vomited several times and the vomitus contained bile stained fluid. Abdominal distension was gradual. He gave history of similar type of attack in the past for several times starting at his seven years of age, which were managed conservatively. He had tachycardia (pulse- 140 beats/ minute, thready and of low volume), blood pressure was 80/60 mm of Hg. and body temperature was normal. Abdomen was distended, rigid, tender with presence of muscle guarding. Digital rectal examination revealed empty ballooned rectum. There was electrolyte imbalance (hypokalaemia, serum potassium : 3 mmol/1) and plain X-ray abdomen in erect posture showed features suggestive of small gut obstruction (Fig-1). After appropriate resuscitation a decision of exploratory laparotomy was made.

During exploration of abdomen haemorrhagic ascitic fluid came out and small gut was found to be distended. A portion of small gut was found to be invaginated through a defect (6cm x 3cm) in the lower end of the mesentery. The portion of small gut proximal to the obstruction was distented, thick walled and viability was doubtful (Fig-2). After decompression of the gut , warm mop application and increased flow of oxygen, viability returned. The anatomical defect in the mesentery was repaired & abdomen was closed in layers. Postoperative period was uneventful and patient was discharged on the ninth postoperative day.

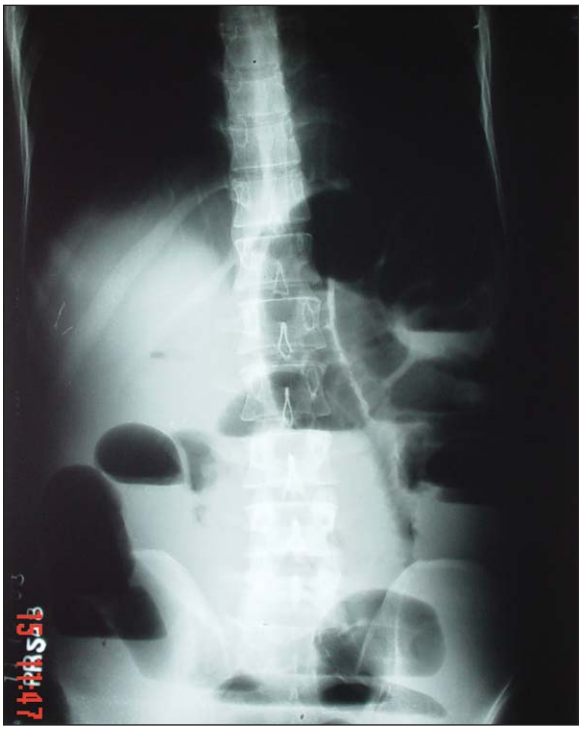

Fig-1 : *Plain x-ray abdomen in erect posture showing features of small bowel obstruction.*

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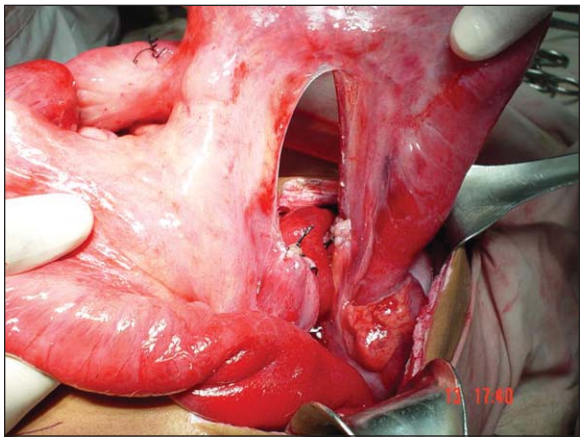

Fig-2 : *Peroperative photograph showing defect in the mesentery.*

Discussion:

Herniation of small bowel through a gap in the mesentery (Stammer's hernia) is a rare cause of small bowel obstruction and seldom diagnosed preoperatively partly because of unfamiliarity with this type of internal hernia. Since internal hernias are rare but important causes of intestinal obstruction (0.2-0.9% of all cases)[1], they are often diagnosed during laparotomy.

Internal hernias are often classified as developmental, congenital or acquired. By definition, developmental internal hernias cause obstructive symptoms in the absence of any previous intraabdominal interventions. Again herniation through a defect in the mesentery is not a true hernia rather it is internal prolapse since it does not have a sac.

The reported incidence of all internal hernias varies between 0.2 and 0.9% of the autopsies, 0.3-2% of parietal hernias and 0.01% of laparotomies[1]. Overall condition is more common in males (Male : female= 3:2). The age distribution varies widely but peak symptomatic incidence is in the fifth decade. The clinical symptoms of internal hernia may be intermittent and non specific making the diagnosis extremely difficult. Vast majority of patients (90%) present with features of acute intestinal obstruction which is often strangulating with evidence of established peritonitis(30-60%).

One should stress in the importance of plain radiological findings as diagnostic aids. A consistent intestinal gas imaging after some interval suggests the possibility of an internal hernia, specially when accompanied by a circular or oval defect of the gas shadow in the middle of the abdomen[2]. A serial abdominal X- ray study can be helpful. Diagnosis of internal hernia with CT is difficult[3]. Special attention should be given to the clustering of bowel loops, the mesenteric vessels and signs of small bowel obstruction.

The essence of good management is early intervention (laparotomy) as this is the only means of preventing gangrene of the bowel. The surgical treatment consists of reduction of hernial contents, resection of gangrenous bowel, (if, any), primary anastomosis and correction of anatomical defect.

The hospital stay, mortality (up to 30% in long series) and morbity depends on the presence or absence of bowel infarction. In the rare instances, when an internal hernia is discovered arter investigation of chronic symptoms, elective surgery is needed because of the pathogenic potential of this condition.

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Chordoma in the Nasopharynx- Reports of Two Cases

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Summary

Chordoma is a very rare bony tumour which arises from the notochordal remnants .Two cases of Chordoma in the nasopharynx were identified in the ENT Department of Bangladesh Medical College Hospital January 1992 to December 2002.Both the patients were old women. Tumour from nasopharynx were removed through the oral route. The Diagnosis was confirmed by the histopathology examination which showed chordoma. A full course of radiotherapy was given in both the cases and the patients improved rapidly.

(J Bangladesh Coll Phys Surg 2004; 22 : 73-75)

Introduction

Chordoma is a malignant bone tumour[1]. It arises from the embryonic remnants of the notochordal tissue. This tissue is normally situated within the vertebral bodies and interverteibral disc. It is found at any point along the axial skeleton. It is present mostly in the fifth and sixth decades of life and in both sexes equally. Fifty present of the chordoma arises from the sacrococcygeal area , 35% from the spheno-ocipital and rest from the cervico- thoracic spine [2]. It is very rare below 40 years of age. It takes about five to ten years to develop the symptoms. Memorial Sloan-Kettering Institute identified 53 cases of chordoma in the sacrum, 24 in vertebral bodies and three in the spheno-occipital region. A high percentage [8.4%] of primary malignant tumours were reported to the National Cancer Institute's Surveillence Epidemiology and End Results[SEER] as chordoma between 1973 to 1987[3].

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In Queen Mary Hospital, Hongkong, tumours in and around nasopharynx were identified and removed in 26 patients. Among them, 18 suffered from nasopharyngeal carcinoma, three had Chordoma, two had Schwanoma and one had adenocarcinoma and one had malignant fibrous histeocytoma[4]. Vollrath in Germany reported two cases of chordoma in the nasopharynx and categorized the chordoma as clival, cervical and sacrococcygeal[5].

Spheno-occipital chordoma may appear with nasal, paranasal or nasopharyngeal mass. It is hard on palpation and may create pressure symptoms. Multiple Cranial nerves may be involved. Chordoma is gelatinous and contains areas of haemorrhages. Microscopically it resembles normal notochordal tissue. Histologically the physeliferous cells are pathognomonic. The tumor in the spine destroys vertebral bodies and arches and can bulge into subdural space .

Clinical diagnosis is made by symptoms, signs and involvement of the surrounding structures and finally by the x-rays, CT scan, MRI and myologram. A soft tissue mass is essential to the radiological diagnosis, with a variable degree of destruction of the vertebrae. CT or MR Scans are invaluable for the demonstration of the extent of bone destruction and the extent of soft tissue mass[7].

Case reports

Case1:

Mrs. JB, 50 years of age, was admitted in ENT department of Bangladesh Medical College Hospital on 19.3.2001 with the complaints of nasal obstruction, feeling of a mass in the throat and occasional bleeding from the nose. She experienced

these problems since previous one year. She observed that something was coming out from behind the soft palate which obstructed her nasal passage. She had snoring during sleep. She also experienced bleeding from the nose three times in previous one year. A thorough clinical examination was done. X-ray of nasopharynx lateral view showed a mass in the nasopharynx completely obstructing the air passage. CT scan of base of the skull showed a tumour attached to the body of C1 and C2 vertebrae. All other routine investigations were normal. She was non-diabetic and non-hypertensive. Her renal and liver function tests were normal.

Excision of the tumour was done under general anaesthesia. The patient was put in tonsillar position by applying a sand bag below the shoulder. The tumour was firm on palpation. The soft palate was lifted up and the tumour was dissected from the body of the vertebrae. The bleeding was controlled by diathermy and a post nasal pack was applied. The specimen was sent for histopathology and was reported to be a chordoma.

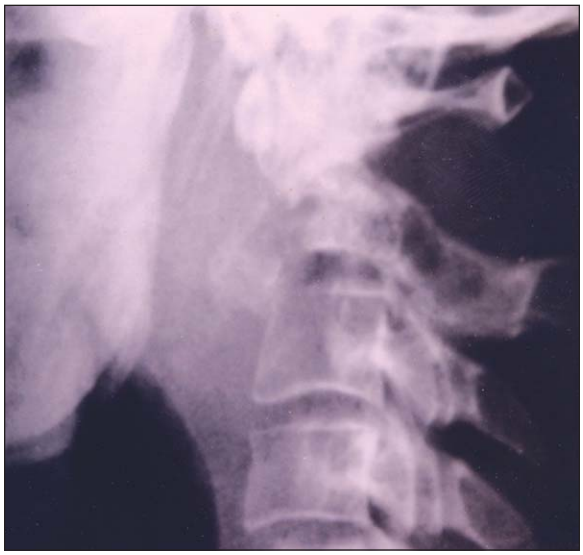

Fig.-1 : *Chordoma of 2nd cervical vertivra body of C2 is destroyed and large paravertileral mass is seen.*

Case- 2:

Mrs. DB, 45 years, was admitted in the ENT ward of Bangladesh Medical College Hospital on 24.10.1999 with history of nasal obstruction and occasional bleeding from nose for the previous six months. A thorough clinical examination and routine investigations were done. X-ray of nasopharynx lateral view showed a tumour in the nasopharynx. All other investigations were normal. She was non-diabetic and non-hypertensive. Her liver and kidney functions were normal.

Excision of the tumour was done through the oral route under general anaesthesia. The tumour was 2.5 x 3.5 cm in size and there was some erosion over the surface. The specimen was sent for histopathology and was reported to be chordoma. The wound healed smoothly within three weeks.

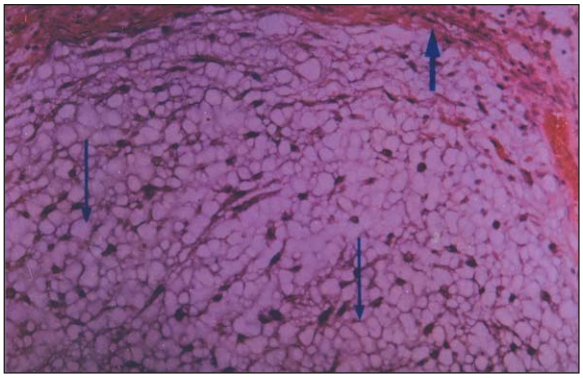

Fig.-2 : *Miroscopic picture of chordoma. the this arrow show large vacides with pale grey maleviales.*

Discussion:

Chordoma is a malignant bone tumour. It arises from the notochordal remnants. The ends of the spines are the most common site of its origin. They look like soft, gray coloured and multiloculated masses. Cervical chordoma frequently presents as a pharyngeal mass. Most of the tumour in the nasopharynx are thought to be carcinoma. But it must be distinguished from chordoma or chondrosarcoma. In case of carcinoma of the nasopharynx metastatic lymphnodes in the neck is an early presentation whereas chordoma has characteristic of late matastases. Invasion of spinal canal by chordoma may cause neurological complications. In one series the frequency of metastases was 43%[7]. The most common sites are the skin and bones but they may occur in any place in the body [8].Treatment of chordoma consists of complete resection of the tumour followed by radiation therapy.

German Society Vollrath compared the result of operation and radiation therapy and found that each alone fails because of the high recurrence rate. Hence they preferred combination of surgery & rediotherapy. One of his patients who was treated with both radiation therapy and surgery had a survival period of 14 years [9]. Thirteen patients were treated in the department of Radiotherapy, University of Cologne, Koeln, Germany histopathology revealed carcinoma arising from the nasopharynx; 8 chordoma 1, rabdomyosarcoma-1, chordrosarcoma-1 and haemangiopericytoma-2. All patients had repeated tumour resection or irradiation, hindering any further conventional fractionated radiotherapy or surgery [10]. But overall prognosis was poor.

Because of the location of chordoma in the base of the skull, removal of the tumour is very difficult and usually partial removal is done. Transcervical and transmandibular approach to the skull base can be employed in removing this tumour [11]. In Russian Academy of Medical Sciences, Moscow a transoral approach was used in two patients with tumours of the clivas, a chordoma and another chordosarcoma. Choice of the approach was based on data provided by clinical and radiographic examinations[12]. Cryosurgery with liquid nitrogen is occasionally used when complete removal of the tissue is not possible [13]. Orthopaedic surgeon should be included in the surgical team. Help of neurosurgeons is asked for when there is intracranial extension. Digital palpation is sometimes helpful in differentiating chordoma from other spinal tumours. X-ray of nasopharynx lateral view, CT Scan, MRI, FNAC and finally excision biopsy are essential requirement for accurate evaluation of chordoma. Vertebral angiography is helpful in demonstrating the tumours by vessel displacement, encasement and vascular staining [14].

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COLLEGE NEWS

(J Bangladesh Coll Phys Surg 2004; 22 : 76-78)

EXAMINATION NEWS

Result of FCPS Part-l, FCPS Part-II and MCPS Examinations held in January, 2004 were announced on the day, examination was held.

1704 candidates appeared in FCPS Part - I Examination in various specialitis held in January, 2004, among them 27 candidates came out successful. Speciality wise results are as follows

FCPS Part I Examination

	No. of candidates appeared	No Number of Candidates come out successful
Medicine	533	01
Surgery	315	07
Paediatrics	187	06
Obst. & Gynae	334	08
Ophthalmology	49	00
Otolaryngology	57	00
Psychiatry	12	00
Anaesthesiology	49	00
Radiology	30	02
Radiotherapy	08	01
Dermatology & Venereology	36	00
Physical Medicine	12	00
Dental Surgery	38	00
Haematology	24	00
Microbiology	07	00
Histopathology	13	02
Total-16	1704	27

FCPS Part-II Examination :

263 candidates appeared in FCPS Part-II Examination in different specialities. The following satisfied the board of examinar and declared to have passed the FCPS Part-II Examination of Bangladesh College of Physicians and Surgeons held in January 2004.

Roll No.	Name of candidate	Graduated from	Speciality
001	Dr. Md. Nor Uddin Tareq	Sir Salimullah Medical College, Dhaka	Medicine
018	Dr. Shamim Ahmed	Dhaka Medical College, Dhaka	Medicine
024	Dr. Shaikh Mohammad Hasan Mamun	Dhaka Medical College, Dhaka	Medicine
030	Dr. Mohammad Sohailul Islam	Chittagong Medical College, Chittagong	Medicine
046	Dr. Faruk Ahammad	MAG Osmani Medical College, Sylhet	Medicine
052	Dr. Mohammad Ibrahim Chowdhury	Dhaka Medical College, Dhaka	Medicine
053	Dr. Kazi Shalmoor Alam	Sir Salimullah Medical College, Dhaka	Medicine
055	Dr. Mohammad Musfiqur Rahman	Dhaka Medical College, Dhaka	Medicine
070	Dr. Mohammed Tanvir Jalal	Dhaka Medical College, Dhaka	Surgery

Roll No.	Name of candidate	Graduated from	Speciality
078	Dr. Jamal Ahmed Chowdhury	Chittagong Medical College, Chittagong	Surgery
090	Dr. Mohamad Masud karim	Rangpur Medical College, Rangpur	Surgery
109	Dr. Imtiaz Faruk	Sir Salimullah Medical College, Dhaka	Surgery
115	Dr. Md. Abul Kashem	Sir Salimullah Medical College, Dhaka	Surgery
135	Dr. Md. Ayub Ali	Mymensingh Medical College, Mymensingh	Paediatrics
147	Dr. Abdul Razzak Sikder	Chittagong Medical College, Chittagong	Paediatrics
155	Dr. Nazma Begum	Sir Salimullah Medical College, Dhaka	Paediatrics
160	Dr. Tanzeem Sabina Chowdhury	Bangladesh Medical College, Dhaka	Obstetrics & Gynaecology
162	Dr. Shahnaz Begum	Sir Salimullah Medical College, Dhaka	Obstetrics & Gynaecology
165	Dr. Suraya Ahmed Chowdhury	Sere-e-Bangla Medical College, Barisal	Obstetrics & Gynaecology
167	Dr. Mahbuba Khanum	Chittagong Medical College, Chittagong	Obstetrics & Gynaecology
168	Dr. Nahid Elora	MAG Osmani Medical College, Sylhet	Obstetrics & Gynaecology
173	Dr. Nasreen Akther	MAG Osmani Medical College Sylhet	Obstetrics & Gynaecology
175	Dr. Tazeen Sultana	Chittagong Medical College, Chittagong	Obstetrics & Gynaecology
181	Dr. Nasim Afza	Dhaka Medical College, Dhaka	Obstetrics & Gynaecology
185	Dr. Rashida Khanom	Mymensingh Medical College, Mymensingh	Obstetrics & Gynaecology
186	Dr. Quorrata Eynul Forhad	Rangpur Medical College, Rangpur.	Obstetrics & Gynaecology
189	Dr. Mariam Rabeya	Chittagong Medical College, Chittagong	Obstetrics & Gynaecology
194	Dr. Shoyela Shahnaz	Mymensingh Medical College, Mymensingh	Obstetrics & Gynaecology
195	Dr. Shefali Banerjee	Sere-e-Bangla Medical College, Barisal	Obstetrics & Gynaecology
197	Dr. Shahela Jesmin	Rajshahi Medical College, Rajshahi	Obstetrics & Gynaecology
209	Dr. Nazlima Nargis	Sir Salimullah Medical College, Dhaka	Obstetrics & Gynaecology
211	Dr. Sharmina Alauddin	Rajshahi Medical College, Rajshahi	Ophthalmology
214	Dr. Shamima Islam	Sere-e-Bangla Medical College, Barisal	Ophthalmology
219	Dr. Md. Ali Akbar	Dhaka Medical College, Dhaka	Ophthalmology
220	Dr. Md. Safiul Islam Prodhan	Rangpur Medical College, Rangpur	Ophthalmology
225	Dr. Dipak Kumar Nag,	Mymensingh Medical College, Mymensingh	Ophthalmology
231	Dr. Malcka Afroz	Chittagong Medical College, Chittagong	Otolaryngology
236	Dr. S. M. Fahmidur Rahman	Sere-e-Bangla Medical College, Barisal	Psychiatry
237	Dr. Sultana Algin	Chittagong Medical College, Chittagong	Psychiatry
238	Dr. Rubina Yasmin	Dhaka Medical College, Dhaka	Anaesthesiology
239	Dr. Bhahesh Chandra Mondal	Sir Salimullah Medical College, Dhaka	Anaesthesiology
241	Dr. Md. Torab Mollick	Sir Salimullah Medical College, Dhaka	Anaesthesiology
242	Dr. Abu Naser Muhammad Badruddoza	RUSSIA	Anaesthesiology
245	Dr. Niaz Ahmed	MAG Osmani Medical College, SyIhet.	Anaesthesiology
248	Dr. Sania Ahsan	Dhaka Medical College, Dhaka	Radiology
252	Dr. Md. Mukitul Huda	Mymensingh Medical College, Mymensingh	Radiotherapy
261	Dr. Md. Fakharuddin Bhuiyan	Mymensingh Medical College, Mymensingh	Haematology
262	Dr. Mimi Parvin	Dhaka Medical College, Dhaka	Biochemistry
263	Dr. Abdullah Al-Baki	Sir Salimullah Medical College, Dhaka	Microbiology

MCPS Examination :

208 Candidates appeared in MCPS Examination in different specialties. The following candidates could satisfied the board of examiners and are declared to have passed the MCPS examination of Bangladesh College of Physician and Surgeons held in January,2004

Roll No	Name of the candidate	Speciality
001	Dr. Mohammad Mahbubur Rahman Khan	Medicine
004	Dr. Abdul Ahad Mohammed Ryhan Uddin	Medicine
008	Dr. Abu Saleh Md. Badrul Hasan	Medicine
013	Dr. Md. Fazlul Karim	Medicine
018	Dr. Md. Mahbubul Alam	Medicine
023	Dr. A.K.M. Mijanur Rahman	Medicine
027	Dr. Md. Jamshed Alam	Medicine
035	Dr. Md. Sirajul Islam	Medicine
072	Dr. Najnin Umme Zakia	Paediatrics
076	Dr. Md. Abdul Awal	Obstetrics & Gynaecology
102	Dr. Noorjahan Begum	Obstetrics & Gynaecology
117	Dr. Rinku Rani Das	Obstetrics & Gynaecology
118	Dr. Sultana Afroza	Obstetrics & Gynaecology
129	Dr. Khaled Mahmud	Otolaryngology
137	Dr. Muhammad Chanchal Azad	Psychiatry
138	Dr. Md. Amirul Basher	Psychiatry
114	Dr. Mohammed Delowar Hossain	Psychiatry
148	Dr. Md. Zulfiker Ali Faruquee	Anaesthesiology
150	Dr. Md. Abdul Mannan Mia	Anaesthesiology
157	Dr. Khairul Anwar	Anaesthesiology
159	Dr. Pankaj Kumar Mohana	Anaesthesiology
160	Dr. Md. Mahbubur Rahman	Anaesthesiology
166	Dr. Muhammad Umar	Radiology
168	Dr. Khandaker Mahbub Hossain	Radiology
171	Dr. Abu Jafar Md. Shahidul Hoq	Dermatology & Venereology
172	Dr. K.M. Majedul Islam	Dermatology & Venereology
180	Dr. Shahida Khatun	Dental Surgery
181	Dr. Md. Enamul Haque	Forensic Medicine
197	Dr. Ajoy Roy Chowdhury	Clinical Pathology
203	Dr. Mamunur Rahman	Clinical Pathology
205	Dr. Md. Nurun Nabi	Clinical Pathology
207	Dr. Mostafizul Karim	Clinical Pathology

ANNUAL GENERAL MEETING:

The Annual General Meeting of the college for 2004 who held on February at the college premieres. A number of agenda along with audited report of income and expenditure of 2003-2004, and annual budget for 2004-2005 were placed before the meeting. Many important decision were adapted in the meeting along with approval of the audited report. Supplementary budget of the previous year and the annual budget of the next fiscal year were also approved.