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JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

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CONTENTS

EDITORIAL

Should Improved Diagnosis and Management of Infectious and Tropical Illness in Bangladesh is Essential in the New Millenium

The health scenario in South East Asia including Bangladesh is similar. The important groups of health conditions in the region causing enormous health burden are still various infections and tropical illnesses. The most frequent causes of death recorded in public health facilities are acute respiratory illness, poisoning and diarrhoea among all age groups. Some other examples of significant public health problems prevalent in Bangladesh are: tuberculosis, dengue, typhoid fever, leishmaniasis, malaria, amoebiasis, filariasis, leptospirosis, viral encephalitis including Japanese B encephalitis, viral hepatitis, animal bites like dog bite resulting in fatal rabies, snake bite etc. (DGHS, 2001). All these illness are tropical health problems and has enormous influence of geographical, socio-cultural, economic and other factors requiring a region specific approach of management and prevention. The policy of management and prevention should therefore be based on locally generated evidence. A number of control programs in the country are functioning with reputation for the control of diseases like ARI, diarrhoeal illness and tuberculosis (TB). The disease burden due to illness prevalent and peculiar for the country mentioned above are huge and its economic impact is immeasurable and sustained. The population affected by such conditions in our country are mostly rural and less educated. Poor health awareness and poor primary heath care coverage are characteristics of such population. The sufferers are often either the bread earners in the family, or children and pregnant women. Prevention of morbidity, disability and mortality from such illness will have great economic impact and will be in line with current global commitment and trends in planning for the marginalized population for poverty alleviation should now focus much more attention on these group of illness in Bangladesh conforming with the Millennium Development Goal. Poor people should not be left behind. Lack of modern safer vaccines or modern TB treatment only due to poverty should not be acceptable (World Bank unpublished paper, 2002).

With the advancement of scientific knowledge and technology the diagnosis, treatment and prevention of tropical health conditions are improved significantly so that appropriate application can change the health scenario of the affected population. Until recently it was unheard that immunological and genetic diagnosis for such conditions like malaria, leishmaniasis, leptospirosis, snake bite will be available with low cost (Sehgal S C, et al 1999. One can now diagnose malaria and Kala azar efficiently in minutes, one can treat these conditions with less duration and cost that could not be predicted even a decade before (WHO, 2001). Oral treatment of Kala azar is now reality by using Miltefosine. Immunodiagnosis of snake bite by venom detection kit using ELISA method to qualitatively detect nanogram quantities of venom from wound site or urine is now part of a community based management those who can afford (White 1995). Early diagnosis of muscle paralysis and effective treatment by artificial respiratory support can prevent innumerable deaths from pesticide poisoning (Senanayake, 1999). Similarly, numerous deaths could be prevented following neurotoxic snake bite by artificial respiratory support which is the cornerstone of management along with antisnake venom. Post-exposure prophylaxis by genetically engineered anti rabies vaccine has found to be effective in prevention of a very deadly fatal illness causing few thousand deaths in the country (WHO 1992).

With this minimum background of evidence based information question arise when people in need in Bangladesh will get the benefit of science for the ailments of their common conditions of important public health priority. Creation of health manpower is a pre condition for the application of scientific knowledge in a particular sector. Bangladesh College of Physicians and Surgeons has taken a timely step for starting Fellowship in Infectious Disease and Tropical Medicine. Earlier the government has

initiated a long over due post graduation in Tropical Medicine in at least one medical college of the country which prove the eagerness to create trained health manpower to tackle tropical health conditions. We should welcome these initiatives. It would be required to include the agenda of improvement of diagnostic microbiology, management options including intensive care monitoring and follow up in the key health institutions of the country so that such man power acquire the requisite appropriate skills conforming with other similar advanced centres.

Facilities of diagnosis, treatment and prevention of such infections and tropical illness should be available throughout the country for application, training and research for creation of evidence in country perspective. Such measures have also been taken in countries of the region such as Thailand, India and Sri Lanka which can be taken as an example for immediate adoption (Eddleston et al, 2002). Assistance from donors could be sought, collaboration for research and development could be requested, and more and more interaction among the scientists of the region for technology transfer are urgently required. Facilitation and encouragement from the part of government will have a positive impact in such a move. It would be too late not to take action promptly now. So that we can provide benefit of evidence based science to our poor population in time of their urgent health problems.

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ORIGINAL ARTICLES

Validity of Tourniquet Test in Dengue Haemorrhagic Fever

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Summary :

Context

It is believed that dengue haemorrhagic fever can be detected clinically by positive tourniquet test (TT) at an earlier stage differentiating it from dengue fever (DF). However, our observation is different. Therefore we hypothesized that TT does not have enough validity to separate dengue hemorrhagic fever (DHF) from DF.

Objective

To determine the validity of TT in differentiating DHF from DF by using sensitivity, specificity and predictive values.

Patients and Setting

Seventy patients (DHF=40 and DF=30) of dengue syndrome from dengue special wards of the Shaheed Suhrawardy Hospital were enrolled in this study for validity analysis of TT. It was done by using Riva Rocci cuff daily from the day of admission till discharge or appearance of a positive result. All diagnoses were done clinically. However, for DHF, evidence of plasma leakage (PCV and USG) was considered essential.

Results

Mean age of the patients was 26 years (range 11-70 years). Forty (57%) patients were male. Analysis yielded a sensitivity of 42.5%, specificity of 70 % and positive and negative predictive values of 65.4 % and 47.7 % respectively.

Conclusion

In DHF, TT is a less informative test than the usual presumption. It has got less validity specially in terms of sensitivity and negative predictive values.

(J Bangladesh Coll Phys Surg 2003; 21 : 123-127)

Introduction

Febrile illness due to dengue virus infection encompasses three distinct clinical presentations: undifferentiated fever (UF), dengue fever (DF) with or without haemorrhage and dengue haemorrhagic fever (DHF) with or without shock[1]. These are termed 'Dengue Syndrome' as a whole. Initially, in the `Febrile phase' (2-7 days) the three presentations show essentially similar features and cannot be differentiated. However, in the 'Afebrile phase' (2-3 days) DF and DHF can be differentiated.

The earliest feature that clinically distinguishes DF from DHF in dengue syndrome is the tourniquet test (TT).[2-4] Later, as the disease (DHF) progresses, plasma leakage, clinically evidenced by effusions, circulatory failure and shock may develop[2-4]. This is never present in DF and thus recovery in DF is rather uneventful as compared to DHF. Also it is mandatory for the patient to have haemorrhagic features to make a diagnosis of DHF along with other features[2]. However, DF may or may not show features of haemorrhage. We considered that if the probable DHF patients could be predicted by positive TT early, it would profoundly assist in their better management and thereby improve outcome.

While encountering dengue patients in the special dengue wards at Shaheed Suhrawardy Hospital (SSH) the authors had a different experience. It was apparent that TT was positive in many of the DF patients that interfered with its early detection. Many of the DHF cases were also not TT positive. However, the national guideline for diagnosis emphasizes TT positivity in DHF[1] but does not mention clearly

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whether TT is positive in DF[2]. Similar experience was reported by other investigators in different parts of the world over the last decade[5-11]. So, the question of validity of TT in differentiating DHF from DF was raised and it was hypothesised that, TT was not sensitive and specific enough to differentiate DHF from DF.

Materials and method

This study was carried out at the Shaheed Suhrawardy Hospital, Sher-e-Bangla Nagar, Dhaka. It is a 375-beded multi-specialty general hospital of Dhaka with both out- and in-patient facilities and averagely equipped pathology, biochemistry and imaging departments. During the dengue outbreak period of 2002, a 100-bed special dengue ward was set up.

This study was done in this special dengue ward in October 2002. The subjects were the patients who were admitted there with suspected dengue syndrome from outpatient (OPD) and emergency departments. As the study involved no intervention or use of patient's personal information, consent was obtained verbally. After admission, history was taken and clinical examination was performed daily till discharge. All data were recorded on a pretested form.

An initial training was given to the observers, which consisted of adequate exposure to the 'National Guidelines for Clinical Management of Dengue Syndrome' and other World Health Organization guidelines[2,12,13] to ensure accurate independent diagnosis of dengue syndrome. Adequate skill in TT method and interpretation of results were also assured. Level of agreement between the observers on the interpretation of TT and diagnosis was not assessed, however, they were supervised closely.

A standard method was employed in performing TT[14]. A Riva Rocci cuff was used as a tourniquet and inflation pressure was raised to midway between systolic and diastolic blood pressure of the patient and kept for five minutes. At the end of five minutes the cuff was deflated and a 2.5 cm diameter area one centimeter below the cubital fossa was observed for petechae. A count of 10 or more petechae was considered positive for the test. TT was performed *daily* on each of the patient till either TT became positive or patient was discharged.

Laboratory tests done were, haemoglobin (Hb) level, total count of white blood cells (TC), differential count of white blood cells (DC), total count of platelets (PC), packed cell volume (PCV) and erythrocyte sedimentation rate (ESR) by Westergren method *and* ultrasonogram (USG) of lower chest and whole abdomen, as suggested in 'National Guidelines for Clinical Management of Dengue Syndrome'[15]. For the cut off levels between normal and abnormal, standard haematological values were used[16]. Considering the constraints of the laboratory services of the hospital at least two records of blood tests were considered logical and appropriate during the patients' hospital stay. Ultrasonogram (USG) of lower chest and whole abdomen was done once at the height of clinical suspicion for pleural effusion and or ascites.

Diagnosis was done in two steps. Initially, after history taking, clinical examination and baseline laboratory examinations, a preliminary diagnosis was made. A final diagnosis was established before discharge. The reason for this is that patients of DHF may show features of some or all of the four DHF grades as the disease evolves before recovery. Despite all efforts, the diagnosis remained as 'suspected' due to failure of confirmation of diagnosis by serology or virus isolation. Diagnosis was based on high index of clinical suspicion and the `National Guidelines for Clinical Management of Dengue Syndrome'[2].

Data were analyzed by using SPSS (version 10.07) statistical package. Analysis was done on patients with complete history and required laboratory test results. Patients with other febrile illness, in whom diagnosis other than dengue syndrome was confirmed later, were excluded from the analysis. Usual causes of incomplete data were inability of the patients to generate history due to failure to recall, incomplete hospital records, early patient requested discharge and failure of laboratory to provide reports due to manpower constraints. A total of four cases were excluded from analysis due to one or more of the reasons mentioned above. Continuous variables were expressed as mean and standard deviation (SD), median and compared by Student's t test. Skewed variables were log transformed (base 10) and analyzed. Geometric mean was used for description

of such data. Categorical variables were expressed in number and percentage and compared by Chi-square (c^2) test. Cut-off points for level of significance (P value) was considered to be less than or equal to 0.05. Validity analyses were done by calculating sensitivity, specificity and positive and negative predictive values using standard methods[17].

Results

A total of 70 patients were described. Among them 40(57%) were males. Patients had an average (geometric mean) age of 26 years with duration of fever of 5.6 days and hospital stay of 4.2 days. Thirty (43%) patients were diagnosed as DF and 40 (57%) as DHF at discharge. Baseline features of the DF and DHF groups are shown in Tables I - II. The DF and DHF patients were similar (P > 0.05) in baseline features except for chest pain (P = 0.007), petechae (P = 0.024) and abdominal distention (P = 0.003). These features were more in the DHF patients. The DHF patients also had longer mean duration of stay in the hospital than the DF patients (P = 0.007). Grading of DHF cases done at discharge is shown in detail in Table-III.

The results of TT in DF and DHF are shown in Table IV. The TT result does not vary significantly between DF and DHF (c^2=1.147, df =1, P=0.28). The validity analysis is done within the spectrum of dengue syndrome, which assumes DF as "non-diseased" and DHF as "diseased". We observed that TT is poorly sensitive (45%) and gives low negative predictive values. However, its specificity (70%) is relatively high.

Table-I

Baseline features of continuous variables of patients of dengue haemorrhagic fever and dengue fever

Variables	DHF (n=40)		DF (n=30)		P**
	Number	Mean (SD)	Number	Mean (SD)	
Age in years*		29.8		23.4	.06
Fever duration in days*		5.7		5.6	.85
Reported peak body temperature (home) in ° F *	32	103	20	102.6	.16
Pulse per minute	39	85.5 (11.8)	29	83.9 (11.1)	.58
Systolic blood pressure in mm Hg *	39	104.2		102.9	.68
Diastolic blood pressure in mm Hg	39	71.5 (9.8)		71.2 (9.6)	.87
Body temperature (hospital) in °F *	38	99.6		99.2	.26
Total count of WBC *	13	5,732	12	6,918	.29
Neutrophil DC in % *	14	54.6	12	58.1	.40
Lymphocyte DC in %	13	35.5 (7.3)	12	36.1(8)	.84
ESR, Westergren 1 hour *	10	14.2	09	12.4	.72
Haemoglobin in gm/di	13	12.7(l.7)	12	12.9 (2.2)	.88
PCV in %	17	39.1(5.5)	15	39(5.1)	.95
Total count of platelet per cumm *	38	60,646		54,338	.49

DC: differential count; ESR: erythrocyte sedimentation rate and PCV: packed cell volume

* Geometric mean

** Student's t test

Table II

Baseline features of categorical variables of patients of dengue haemorrhagic fever and dengue fever

Variables	DHF n=40 Number (%)	DF n=30	P*
Myalgia	37(92)	27(90)	0.71
Arthralgia	32(80)	19(63)	0.12
Retro-orbital pain	22(55)*	23(77)	0.80
Headache	35(87)	24(80)	0.39
Rash	18(45)	15(50)	0.68
Nausea	38(95)	28(93)	0.77
Vomiting	35(87)	21(70)	0.07
Loss of appetite	40(100)	28(93)	0.10
Sore throat	10 (25)*	11(37)	0.32
Cough	15 (37)*	15(50)	0.43
Diarrhoea	12(10)	13(43)	0.25
Breathlessness	19(47)	8(27)	0.08
Chest pain	39 (97)	13(43)	0.01
Generalized weakness	30(75)	27 (90)*	0.38
Dizziness	28(70)*	16(53)	0.19
Drowsiness	04(10)	03(10)	1.00
Abdominal pain	30(75)	14(47)	0.15
Abdominal distention	27(67)	09 (30)*	0.01
Ciprofloxacin use	10(25)	07(23)	0.87
NSAID use	14(35)	06(20)	0.17
Bleeding Manifestations:			
Gum bleeding	17(42)	11(137)	0.62
Sub-conjunctival bleeding	09(22)	08(27)	0.69
Hematemesis	08(20)	06(20)	1.00
Melaena	16(40)	14(47)	0.58
Epistaxis	02(5)	01(3)	0.73
Per-vaginal bleeding	05(31)	02(15)	0.27
Petechae	16 (40)**	05(17)	0.02

* One missing value
** Two missing values
Chi-square test

Table-III

Grading of dengue haemorrhagic fever patients at discharge (n = 40)

Diagnosis	Number (%)
DHF 1	02 (2.8)
DHF 2	29 (14.4)
DHF 3	8(11.4)
DHF 4	1(1.4)

DHF indicates dengue hemorrhagic fever

Table IV

Validity indices of TT (n=40)

Sensitivity of TT	42.5
Specificity of TT	70%
Predictive value of a positive TT	65.4
Predictive value of a negative TT	47.7

Discussion :

This study was done with the hypothesis that positive TT is not more prevalent in DHF in comparison to DF, although existing belief and established guidelines on dengue virus infection suggests differently. The result of this study is consistent with this hypothesis. Available information reveals that no such studies have been done on this issue in Bangladeshi population.

The baseline statistics of the study population are similar to other previous findings[5-11]. DHF and DF patients, baseline features of the sample are also similar. So the group of patients in this study are not largely different from the patients of other dengue studies conducted elsewhere. Wali *et al* reported that a negative TT might not sufficiently exclude a diagnosis of DHF. They have also emphasized the need for re-defining the clinical criteria for the diagnosis of DHF, particularly DHF grade l. 'Cao *et al* concluded that TT differentiated poorly between DHF and DF[6]. Gomber *et al*[5] commented TT as a less sensitive marker of DHF[7]. Though there are studies reporting the increased positivity of TT in DHF, their numbers are much less. Richards *et al* reports 100% positive TT in 23 DHF children in the first outbreak in Irian Jaya, Indonesia in 1997[18]. However, it may be

argued that their study patients were aged from 1-12 years and sample size was small. Other authors also suggest the possivility of TT posivity in DF[19].

Positive TT is thought to be early sign of increased capillary fragility and a part of abnormal haemostasis in dengue patients[2-4,20]. Other haemostasic defects that gradually develop are easy bruising at venepuncture site, thrombocytopenia, impaired platelet function, consumptive coagulopathy and disseminated intravascular coagulation. However, the study patients did not show TT possibvity despite their development of other signs of haemostatic defect (Table-II). It is argued that, if positive TT is a sign of covert haemorrhage[14] then it should not have any preference towards DHF cases than DF with haemorrhage, and all or most DHF cases should have positive TT.

Despite all sincere *efforts* this study had several limitations. All the DHF patients might not have been tested for TT at DHF stage-1. The diagnosis that has been used were not confirmed by serology or virus isolation and may be a source of possible bias also. Because this study was done in an outbreak situation, external validity of its results is also not beyond question.

It may be concluded that, TT is not sufficiently sensitive and predictive values are low. It should be considered less informative in isolation. TT should be used judiciously along with other criteria of dengue syndrome. As small sample size precludes firm conclusion, further studies with large sample size are warranted.

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Epidemiological, Clinical and Biochemical Profile of Hepatitis E in Hospital Admitted Adult Patients

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Summary:

This is a prospective study of viral hepatitis E patients admitted in the medicine department at the 200 bedded Hospital Narayanganj to study the epidemiological, clinical and biochemical profile of hepatitis E in relation to area of residence and seasonal variation. A total of 80 adult patients of viral hepatitis were admitted in the year 2002. The prevalence of hepatitis E was 55% among the viral hepatitis cases. Young male adults between 16-35 years of age were predominantly affected. The mean age was 25.70 years. Infection of HEV occurred predominantly in the months of March to May (62.5%) Most of the patients (68.18%) lived in the urban area and the majority were from lower socioeconomic strata (85%). The students (36.36%), labourer and industrial worker (22.72%) and the house wives (18.18%) were mainly affected. Most common presenting complaints were yellow colouration of eyes and urine (100%), nausea (95.45%), anorexia (90.90%) fever (72.72%) and arthalgia (65.90%). Physical findings include clinical jaundice (100%), fever (72.72%), hepatomegaly (68.18%). Features of cholestasis were observed in nine (20.45%) patients while bleeding occured in one (2.27%) patient. Majority of the patients (43.18%) had serum bilirubin in the range of 6 -10.9 mg/dl.The mean ALT level in HEV positive cases was 452 Iu/L, over a range of 102-3450. Prothrombine time was prolonged in eight out of 30 patients in whom it was done. Only one patient died from fulminant hepatic failure giving the mortality rate of 2.27%.

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Introduction:

Hepatitis E virus (HEV) is an enterically transmitted calici-like virus that has been implicated in huge outbreaks of hepatitis in developing countries. It also causes substantial number of sporadic cases in endemic areas and is responsible for much morbidity and mortality. This burden tends to fall on poor areas since the virus which is predominantly waterborne, is most prevalent where sewage disposal facilities are inadequate and water supplies are unsafe. This disease was first recognised from an epidemic hepatitis in Kashmir, India in 1978[1]. HEV infection is a common disease in Indian subcontinent, Asia and Africa.

In developing countries where all types of viral hepatitis prevail, a preponderance of HEV infection has been observed[2]. The magnitude of enterically transmitted viral hepatitis is yet to be explored in Bangladesh. Data from two teaching hospitals reveal that enterically transmitted hepatitis is endemic in the country[3], attaining an exaggerated proportion in rainy season, specially in the flood-affected areas.

Narayanganj is a district town situated in the South-eastern part of Bangladesh. Being an industrial and commercial town, migrant labourer come in large numbers from Northern and Southern parts of Bangladesh. The town has a population of two million with approximately half being migrant labourer. They live in slums, in overcrowded rooms with no piped, treated water supply. The risk of transmission of oro-faecal infections to humans is very high. The present study was undertaken to study the epidemiological, clinical and biochemical profile of hepatitis E in hospital admitted adult patients.

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Material and methods :

This is a prospective study conducted in the year 2002, in the department of Medicine at 200 bedded Hospital, Narayanganj. Patients are referred by their physicians to come to this specialized hospital for treatment. This hospital serves patients from Narayanganj, Munshiganj and Chandpur districts. these patients come from a variety of socioeconomic backgrounds and therefore were not exposed to the same type of sanitary conditions.

A total of 80 adult patients who presented with symptoms of acute hepatitis, between January 2002 - December 2002, and who satisfied the clinical criteria for participation is this study were admitted in the medicine wards of this hospital. The diagnosis

of hepatitis presumed to be of viral origin was made when the following criteria were met: (i) presence of a typical prodrome of viral hepatitis in history and clinical observation with jaundice; (ii) no past history of liver disease or jaundice; (iii) biochemical evidence of hepatocellular injury, such as increased serum bilirubin, asperate aminotransferase, and alanine aminotransferase levels; and (iv) no other cause for the development of liver dysfunction or jaundice. (v) absence of ascites, cirrhosis and bile duct obstruction. Patients with a history of alcohol intake, exposure to drugs, or blood transfusion were excluded from this study. Patients data were collected in prescribed forms containing history, clinical findings, available laboratory data and socioeconomic background.

Sera collected from the patients were analysed to establish liver dysfunction and the viral cause of hepatitis. Assessment of liver function test included estimation of serum total bilirubin, asperate aminotransferase, and alanine aminotransferase, alkaline phosphatase.

For the detection of the viral marker, serum samples of patients were screened for anti HAV immunoglobulin (Ig)M and hepatitis B surface antigen (HBsAg). For the detection of HEV infection, all sera were tested for anti HEV IgM using enzyme-linked immunosorbent assay (ELISA) as reported previously[4]. Patients who were found IgM positive for HEV were taken as case of hepatitis E and were further analysed in respect of age, sex, occupation/social groups, clinical features, biochemical and serological profile.

Results:

During the year 2002, a total of 80 adult patients of viral hepatitis were admitted in the medicine wards of the 200 bedded Hospital, Narayanganj. Hepatitis E infection was responsible for 55% (44/80) of viral hepatitis. 26 (59.09%) patients were male while 18 (40.91%) were female (Table-I) and their mean age was 25.7 years (range 16-45 years). Forty (90.90%) patients were in the age group of 16-35 years. Table-II shows the monthly distribution of the viral hepatitis cases those found positive for anti HEV IgM. Although viral hepatitis occurred throughout the year, there is significant rise in the number of cases in the months of March to May (62.5% of viral hepatitis and 68.8% of hepatitis E).

Table-I

Age and sex distribution of hepatitis E cases (n=44)

Age (in years)	Male	Female	Total (%)
16-20	6	3	09 (20.45)
21-25	8	2	10 (22.72)
26-30	7	6	13 (29.54)
31-35	3	5	08 (18.18)
36-40	1	1	02 (04.54)
41-45	1	1	02 (04.54)
Total	26	18	44 (100)

Table-II

Monthly distribution of hepatitis E (n=44)

Month	Number of viral hepatitis cases	HEV (%)
January	02	01 (50.00%)
February	04	02 (50.00%)
March	20	13 (65.00%)
April	17	10 (58.82%)
May	13	07 (53.84%)
June	10	06 (60.00%)
July	04	02 (50.00%)
August	03	01 (33.33%)
September	03	01 (33.33%)
October	02	01 (50.00%)
November	01	00 (0.00%)
December	01	00 (0.00%)
Total	80	44 (55%)

On analysis of demographic data, 68.18% of patients with hepatitis E infection were residents of urban areas and rest (31.82%) were from villages close to the outskirts of town (Figure-1). Table III describes the social and occupational groups affected by HEV infection. Largest number (36.36%) occurred in students while 22.70% occurred in labours and industrial worker, 18.18% (8/44) in house wives and 13.63% in businessmen.

Fig-1: *Urban versus rural distribution of HEV cases*

Table-III

Occupational / social group that predispose to hepatitis E

Occupational/ social group	Number of HEV (%)
Labours/industrial worker	10 (22.72)
Students	16 (36.36)
House wives	08 (18.18)
Service	04 (09.09)
Business men	06 (13.63)
Total	44 (100)

Table IV shows the symptoms and signs of 44 patients with HEV infection. The most common complaints were yellow colouration of eyes and urine (100%), nausea (95.45%) anorexia, (90.90%), fever (72.72%) and arthralgia (65.90%). Clinical Jaundice were present in 44 patients (100%). Hepatomegaly was observed in 68.18% patients with an average liver size of 4 cm below the costal margin (range 2-10). Feature of cholestasis (deepening Jaundice, itching, scratch marks) were noticed in 20.45% cases of HEV while bleeding manifestation (haematemesis and melaena, gum bleeding) occurred in only one (2.27%) case.

Table-IV

Clinical feature of hepatitis E infection

Symptoms and signs	Number (%)
Anorexia	40 (90.90)
Nausea	42 (95.45)
Vomiting	26 (59.09)
Malaise	28 (63.63)
Abdominal discomfort	20 (45.45)
Fever	32 (72.72)
Arthalgia	29 (65.90)
Clinical Jaundice	44 (100.00)
Hepatomegaly	30 (68.18)
Cholestasis (itching, scratch marks)	09 (20.45)
Bleeding manifestation	01 (02.27)
Total	44 (100)

Table V describes the level of serum bilirubin (mg/dl) found on admission of HEV patients. Largest number of patients (43.18%) had serum bilirubin in the range of 6 - 10.9 mg/dl. Two patients (4.54%) were found deeply Jaundiced with serum bilirubin above 20 mg/dl. Serum alanine aminotransferace (ALT) level found on admission of patients with HEV are shown in table VI. Twenty two (50%) patients had serum ALT level in the range of 40-400 units/L, while eight patients (18.18%) had ALT above 1000 units/L, and the maximum level recorded was 3450 units /L.

Table-V

Serum bilirubin level in hepatitis E cases (n = 44)

Serum bilirubin range (mg/dl)	Number (%)
2 - 5.9	15 (34.09)
6 - 10.9	19 (43.18)
11 - 14.9	05 (11.36)
15 - 19.9	03 (06.81)
$\geq$ 20	02 (04.54)

Table-V1

Serum alanine amino transferance level in hepatitis E cases (n = 44)

Serum ALT level range (Units / L)	Number (%)
40 - 200	12 (27.27)
201 - 400	10 (22.72)
401 - 600	06 (13.63)
601 - 800	04 (09.09)
801 – 1000	04 (09.09)
> 1000	08 (18.18)

Table VII describes the rate of anti HEV IgM (cut off rate were 0.338-0.430). Seventeen (38.63%) patients had anti HEV IgM in the range of 3-3.9.

Table-VII

Anti HEV IgM level among the hepatitis E cases (n = 44)

IgM level (OD)	Number (%)
5 – .9	06 (13.63)
1 – 1.9	07 (15.90)
2 – 2.9	09 (20.45)
3 – 3.9	17 (38.63)
4 – 5	05 (11.36)

Discussion:

Epidemic and sporadic viral hepatitis is a common health problem in Bangladesh. This leads to morbidity and mortality in a large number of population[5]. There are epidemics of viral hepatitis almost twice in every year in the months of March to May and September to October. Hepatitis E was first recognized during an epidemic of hepatitis in Kashmir Valley in 1978[1]. It is an ecologically determined disease with faecal contamination of drinking water. It exists as sporadic hepatitis with periodic occurrence of epidemic. More than 50% of acute viral hepatitis in all age groups in Kashmir is due to HEV. Similar findings have been reported from Delhi[3] which are comparable with finding of this study. Data gathered by the South East Asia Regional office of WHO shows that hepatitis E is wide spread in several countries of this region and may account for up to 90% of all sporadic cases of acute viral hepatitis[6,7].

Outbreaks have been described throughout rural and urban parts of India, South-east Asia, China and Russia[8,9,10]. No outbreaks have been described in developed countries, presumably because water supply and sanitary systems are satisfactory[11]. Hepatitis E is a water borne disease. Contamination of water sources can occur following heavy rains, flooding and recession of flood waters, from sewage pipes, open drains, crowded living conditions with unsafe water supply and disposal of human waste, as in refugee camps and rapidly growing urban slums[12]. HEV affects young adult population (15-40 years)[12] but has a lower attack rate in children and elderly. This disease is transmitted orofaecally and should affect children more than adults. It is possible that hepatitis E in children manifests as anicteric disease and is not apparent clinically[12]. In this study, HEV infection was responsible for 55% of viral hepatitis patients. It occurred predominantly in the months of March to May and young adults in the age group of 16-35 years (90.90%) were mainly affected.

Narayanganj is an overcrowded city with many urban slums and low lying areas. Flooding and overflowing of sewers is common following monsoons. The sewage system laid many years back is old and always leaking. Acute icteric disease is similar to those of hepatitis A. The onset of jaundice is usually accompanied by malaise, anorexia, abdominal discomfort and enlargement of liver. About two thirds of the patients develop fever and complain of arthralgia. The diagnosis of HEV until recently was done by exclusion of hepatitis A, B, and C and epidemiological features. However, several methods have been developed like immune electron microscopy, immune flurescence method, polymerase chain reaction and enzyme linked immuno sorbent assay. All the tests are used as a research tool except the last one. ELISA kits are now commercially available. IgM antibody to HEV is a single serum sample is diagnostic of acute HEV hepatitis[5]. Liver function tests are suggestive of hepatocellular necrosis. The serum bilirubin and transaminase rise are similar to that of acute hepatitis B. Transaminase

elevation is monophasic, however cholestasis is a predominent feature in 9 (20.45) patients and should be differentiated from large bile duct obstruction .

In general, the disease runs a benign self limited course. The overall mortality rate is usually low (.5-3.0%) except in pregnant women who have a case fatality of about 20-25% from fulminant hepatic failure[13]. Women in the third trimester of pregnancy are specially vulnerable[14]. The reason for this high mortality is not yet clear. Although Hepatitis E was found in 18 female patients, only two were pregnant and none died in this study. The possible explanation could be that pregnant women with viral hepatitis are being referred by general physician to nearby Institute of Child and , Maternal Health and Hospital, Matuail, a tertiary care hospital.

A follow up study of 304 patients four years after the Delhi epidemic revealed no evidence of chronic liver disease[15]. Recent evidence suggests that human with sub-clinical HEV infection and animals may represent reservoirs of HEV. Hepatitis E virus is found in both wild and domestic animals; thus, HEV is a zoonotic virus. The viruses isolated from pigs in the United States or Taiwan are closely related to human HEV found in those areas. The close genetic relationship of the pig and human virus suggests that pig may be a reservoir of HEV.

The control of HEV depends upon improvement of sanitation, proper sewarage disposal and supply of safe drinking water. Besides, mass education and public awareness is the cornerstone for control of HEV infection.

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Symptomatic Improvement & Echocardiographic Changes in Patients with Coronary Artery Disease (CAD) Treated by Percutaneous Transluminal Coronary Angioplasty (PTCA)

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Summary :

This prospective study was carried out in the department of Cardiology, Bangabandhu Sheikh Mujib Medical University (BSMMU) and Combined Military Hospital (CMH), Dhaka, Bangladesh during the period of March 1999 to October 2000.The study was designed to see the symptomatic improvement and echocardiographic change in patient with CAD receiving interventional treatment. Fifty-five consecutive patients with CAD were included and divided into two groups according to final treatment they received. Among the 55 patients, 40[72.73%(35 male and 5 female)] were in stent group (group A) and 15[27.27%(14 male and 1 female)] in PTCA group (group B). Repeat PTCA, PTCA on grafted vessel and bailout stenting was excluded from the study. Mean age of the two groups were 50.85±6.81 years and 46.60±6.45 years respectively (P<0.01). Myocardial infarction was the commonest clinical diagnosis (56.37%), followed by unstable angina (23.63%) and chronic stable angina (20%). Before intervention, 43(78.18%) patients were symptomatic (functional class II and III) and 12(21.82%) were asymptomatic (class I). No significant difference was observed between the two groups regarding clinical presentations (P>0.05). At 6 months follow up, there was significant improvement of symptoms (35.42% were symptomatic & remaining 64.58% became asymptomatic), and echocardiographycally there were improvement in wall motion and LV systolic function were observed (P<0.05). In conclusion, it was clearly evident that the result was better in term of symptom relief and functional status in both the stent group and PTCA group only.

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Introduction :

CAD is the leading cause of death and disability world wide[1] and it is increasing in South Asian countries including Bangladesh[2-3]. PTCA is currently an accepted method for treatment of patients with CAD. In Bangladesh, PTCA is practiced since 1987 and PTCA with stent from 1997[4]. In 1953 Elder & Hertz of Sweden began to use ultrasonoscope to examine the heart[5]. Feigenbaum described the techniques in 1963[6]. In Bangladesh echocardiography was introduced in the National Institute of Cardiovascular Disease in 1981. Much development has occurred in echocardiography as a non-invasive tool for defining left ventricular performance. Many studies have been carried out in different countries in this field regarding changes in echocardiographic profile and symptoms after PTCA. This study was conducted to see left ventricular function, wall motion change as well as symptomatic improvement of patients receiving interventional treatment.

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Method :

This study consisted of fifty-five consecutive patients selected in CMH, Dhaka during the period of March1999 to October 2000, and grouped into two groups (Group A and Group B) according to final treatment they received. In group A, 40 patients were included who underwent PTCA with stent and in group B, 15 patients who underwent PTCA only. The study population consisted of patients with chronic stable angina (CSA), unstable angina (UA) after stabilization of symptoms, myocardial infarction and those who had evidence of provocable ischaemia on exercise tolerance test (ETT). But bailout stenting, repeat PTCA and PTCA on grafted vessel were excluded from the study. The study was carried out under standard procedure. The specific criterion for

intervention was at least 70 percent stenosis. In addition to angiography, baseline clinical characteristics, patients symptoms, basic investigations were noted. Echocardiography(2D & M-mode and Colour doppler) were done to see functional improvement and wall motion changes and to exclude congenital and valvular diseases in all patients before the procedure and at sixth month follow-up.

Statistical analysis: All the parametric values are expressed as mean±one standard deviation (mean±SD) and non-parametric values as percentage(%). Unpaired Students 't' test, Chi-square, and Fisher's exact test were used where applicable. A probability (P) value of <0.05 was considered as significant.

Results :

A total of 55 consecutive patients were included in this study and divided into two groups, group A & group B. Group A included 40(72.72%) patients treated with stent and Group B included 15(27.28%) patients treated with PTCA only. The mean age of group A was 50.85±6.81 years and in group B was 46.6±6.45 years; male patients were significantly higher in both the groups (P<0.01). MI was the commonest clinical diagnosis followed by UA and CSA in both the groups and no significant difference was observed in two groups(P>0.05)[table-I]. Before intervention 32(80%) patients in group A and 11(73.33%) patients in-group B were symptomatic (functional class II & III) and no significant difference were observed regarding clinical presentations (P>0.05). But at 6-months follow-up 12(33.33%) in group A and 5(41.67%) in group B were symptomatic and significant improvement of symptom in both the groups was observed (P<0.001)[table-II]. Majority of the patients had positive ECG for IHD in both the groups (90% Vs 86.67% respectively) and ETT was positive in all cases. But at 6- month follow up there was significant improvement in ECG and ETT (P<0.001)[table-III]. Majority of patients had hypokinetic wall motion in both the groups (60% Vs 53.33%) before intervention. During follow-up there was improvement in wall motion in both the groups which was significant (P<0.05)(44.45% Vs 41.67%). Most commonly involved wall was LV anterior wall followed by posterior wall. Most of the patients had ejection fraction in between 40-49%, followed by >50% and < 40% but no significant difference was noted in between groups (P>0.05). But at 6-month follow-up there was significant improvement of LV ejection fraction (P<0.00!)[table-IV].

Table - 1

Baseline clinical characteristics of the study population (N=55)

Characteristics	Group-A(n=40)	Group B(n=15)
1. Age (years) (mean±SD)	50.85±6.81	46.60±6.45
2. Sex (No./ %)		
Male	35(87.5)	14(93.33)
Female	5(1.25)	1(6.67)
3. Height (cm) (mean±SD)	166±2.63	160.13±6.23
4. Weight (kg) (mean±SD)	63.90±5.07	65.53±5.94
5. BMI (kg/m2)	23.21±1.73	25.51±0.86
6. Risk factor (No./ %)		
Smoking	28(70.0)	10(66.7)
Hypertension	24(60.0)	8(53.3)
Diabetes Mellitus	14(35.0)	4(26.67)
Dyslipidaemia	26(65.0)	9(60.00
F/H of IHD	16(40.0)	3(20.0)
7. Clinical diagnosis(No./ %)		
Chronic stable angina	8(20.0)	3(20.0)
Unstabe angina	10(25.0)	3(20.0)
Myocardial infarction	22(55.0)	9(60.0)

Table-II

Symptoms of study patients

Variables	Before intervention(N=55)		After intervention(N=48)	
	Gr-A(n=40)	Gr-B(n=15)	Gr-A(n=36)	Gr-B(n=12)
Asymptomatic				
CCS class-I	8(20.0)	4(26.67)	24(66.67)	7(58.33)
Symptomatic				
CCS class-II	20(50.0)	8(53.33)	6(16.67)	3(25.0)
CCS class-III	12(30.0)	3(20.0)	6(16.67)	2(16.67)

Tabel - III

Baseline investigation of the study population (N=55)

Investigations	Before intervention(N=55)		After intervention(N=48)	
	Gr-A(n=40)	Gr-B(n=15)	Gr-A(n=36)	Gr-B(n=12)
ECG(No/%)				
Positive for IHD	36(90.0)	13(86.67)	27(75.0)	10(83.33)
Negative for IHD	4(10.0)	2(13.33)	9(25.0)	2(16.67)
ETT(No/%)				
Positive	40(100)	15(100)	30(83.33)	11(91.67)
Negative	0	0	6(16.67)	1(8.33)

Table-1V

Echocardiographic profile of patients before & after intervention

Variables	Before intervention(N=55)		After intervention(N=48)	
	Gr-A(n=40)	Gr-B(n=15)	Gr-A(n=36)	Gr-B(n=12)
1. RWMA:				
Normal	16(40.0)	7(46.67)	20(55.55)	7(58.33)
Hypokinetic	24(60.0)	8(53.33)	16(44.45)	5(41.67)
Akinetic& Dyskinetic	0	0	0	0
2. Wall involved:				
LV anterior wall±IVS	14(58.33)	5(62.5)	10(62.5)	3(60.0)
LV lateral wall	3(12.5)	1(12.5)	2(12.5)	1(20.0)
LV posterior wall±IVS	7(29.16)	2(25.0)	4(25.0)	1(20.0)
3. Ejection fraction(%)				
<40	6(15.0)	3(20.0)	2(5.55)	1(8.33)
40-49	20(50.0)	8(53.33)	16(44.45)	6(50.0)
≥50	14(35.0)	4(26.67)	18(50.0)	5(41.67)
4. Other abnormalities	0	0	0	0

Discussion :

Fifty-five consecutive patients with CAD who underwent intervention with PTCA with and without stent were included in this study and were divided into two groups. Group A included 40 patients who underwent PTCA with stent and group B included 15 patients who were treated with PTCA only. In this study, symptomatic improvement and echocardiographic profile of the patients were analyzed before and at six month follow-up who were treated successfully with PTCA with and without stent. During follow-up, 4 patients from group A and 3 from group B were missed. Among the 55 patients, males were 87.5 & 93.33 percent, and females were 12.5 & 6.67 percent in-group A and B respectively. Sex distribution was not statistically different between the two groups (P>0.05). The mean (±SD) age of the two groups were 50.85±6.81 years and 46.60±6.45 years respectively. Similar sex and age distribution was reported by Rahman et al[7] . Study patients had clinical diagnosis of MI (55vs 60%), UA (25vs 20%) and CSA (20vs20%) in group A & B, respectively. Majority of the patients were symptomatic (functional class II and III) before intervention (80vs 73.33%) in group A & B, respectively. At 6-month follow-up, symptomatic patients were significantly less (33.33%vs 41.67%) in group A & B, respectively (P<0.05). This is similar to the other studies.

Before intervention, majority patients had hypokinetic wall motion in both the groups (60%vs53.33% respectively) and at six months follow-up there were improvement in wall motion in both the group which was significant (P<0.05) (44.45% vs 41.67% respectively). Most of the patient had ejection fraction in between 40-49%, followed by >50% and < 40% but no significant difference was noted in between groups(P>0.05). But at six month follow-up there were significant improvement of LV ejection fraction(P<0.001). This finding is comparable with other studies[10,11].

Conclusion :

This study showed that there was significant improvement of symptoms as well as improvement functional status and wall motions in patients who were treated with PTCA.

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Sonographic Evaluation of Intraventricular Haemorrhage in High Risk Newborn

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Summary :

Objective : To find out the prevalence of intraventricular haemorrhage in the newborn with low birth weight or birth asphyxia.

Design : This is a cross sectional prospective study.

Setting : Neonatal ward of Khulna Medical College Hospital for a period of 15 months between January 2002 to March 2003.

Subjects :All the high risk newborn admitted in neonatal ward were enrolled for this study. Low birth weight and perinatal asphyxia were regarded as high risk factors.

Main outcome variables : Detection and grading of intraventricular haemorrhage, Relation of intraventricular haemorrhage with low birth weight and perinatal asphyxia.

Results : Seventy one out of 118 cases were asphyxiated baby. Intraventricular haemorrhage were found in 21 (17.8%) children of different birth weight. Among the 37 normal birth weight baby with perinatal asphyxia only 2 (5.4%) cases had intraventricular haemorrhage and[9] babies were extremely low birth weight (55.6%) suffering from intraventricular haemorrhage. Very low birth weight (18.8%) and Extreme low birth weight (23.1%) babies bore the brunt of the disease. Prevalence and severity of haemorrhage was inversely proportional to birth weight and directly proportional to perinatal asphyxia.

Conclusion : Low birth weight and perinatal asphyxia are evidently high risk factors for intraventricular haemorrhage and intracranial ultrasonography is an essential tool for its evaluation.

(J Bangladesh Coll Phys Surg 2003; 21 : 137-140)

Introduction :

Intracranial haemorrhage in newborn may result from birth asphyxia or trauma and rarely from primary haemorrhagic disturbance[1,2]. Haemorrhage due to trauma may be epidural, subdural or subarachnoid but that due to haemorrhagic disturbance is usually intracerebral. Haemorrhages in the ventricles usually occurs from prematurity or perinatal asphyxia without trauma[3,4].

Improvement in perinatal and neonatal care have increased the survival of high risk newborn which means there are more infants who are candidates to develop intraventricular haemorrhage (IVH) than 20 years ago[5]. The incidence of IVH increases with decreasing birth weight:- 60-70% in extreme low birth weight (ELBW) & 10-20% in very low birth weight (VLBW) baby. IVH is rarely present at birth because 80-90% cases occur within 3rd day of life and 10-20% within 7 days of life. Delayed haemorrhage may occur in 5-10% cases between 7-28 days of life[6,7]. IVH in the premature infant occurs in gelatinous subependymal germinal matrix which is highly vascular with poor tissue support. Predisposing factors for IVH include prematurity, hypoxic ischemic encephalophathy (HIE), respiratory distress syndrome and hypervolaemia[6]. IVH leads to infarction which later develops periventricular leukomalacia (PVL) due to necrosis of periventricular whitematter[5-7].

Scanning of brain in children can be done by several ways - ultrasonography, CT Scan, MRI & isotope scanning. In most of the cases ultrasonography is more sensitive than CT scan and less sensitive than MRI. Sensitivity in detecting NH by ultrasorography and CT scan is 83% and 39% respectively[8]. So before closure of fontanelle ultrasonography is easier, cheaper and acceptable method to diagnose IVH in children.

The awareness of the magnitude of IVH will help the clinician to predict the problem with high risk cases. The objective of this study was to find out the prevalence of IVH in the newborn with low birth weight or birth asphyxia.

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Patients & Methods :

This study was conducted for a period of 15 months between January 2002 to March 2003 in Khulna Medical College Hospital. All the newborn having admitted in the neonatal unit and meeting the inclusion criteria were recruited for this study. Inclusion criteria was at least one of the two risk factors- low birth weight (LBW) and perinatal asphyxia (PNA). Birth weight below 2500gm was LBW and below 1500gm was VLBW and below 1000gm was ELBW[9]. Perinatal asphyxia was determined by recording of APGAR score (<4) just after birth[4]. Babies dying within three days and having congenital cranial anomaly have been excluded from the study.

After thorough physical examination each baby record were taken in a proforma by the clinical assistant and reviewed by the consultant. These proforma were designed to get the basic information including gestational age, birth weight and perinatal factors such as maternal illness, mode of delivery etc.

Cranial ultrasound were done routinely in all patients on the third day the anterior fontanelle in standard coronal and parasagittal views. IVH was classified as grades I to' IV 'as proposed by Volpes[10.] Grade I Germinal matrix haemorrhage; Grade II- Small IVH occupying 10-50% of ventricles; Grade III - Large IVH occupying >50% ventricular area; Grade IV- IVH with periventricular echodensity. Sonography was performed by Siemmens Sonoline (SL-2) ultrasonography machine with 3.5 MHz & 5 MHz linear and sector probes respectively.

Data after collection were extrapolated in master sheet and then transferred into tables and figures using descriptive statistics.

Results :

A total of 118 cases were recruited for this study. Among them 71 were asphyxiated baby. Intra Ventricular Haemorrhage was found in 21(17.8%) cases of different birth weight.

Seventy one children with perinatal asphyxia were categorized into four groups according to birth weight (Table-1). Among 37 normal birth weight babies 2 (5.4%) developed IVH. Nine babies had extremely low birth weight (ELBW) and majority (55.6%) of them suffered from IVH. Table II shows forty seven children having low birth weight without perinatal asphyxia. There were 8 (17%) cases of IVH among them and most of them were VLBW & ELBW babies. Grades of haemorrhage in different level of birth weight and PNA are shown in Table-III. Highest number (7) of grade III & IV haemorrhage were found in ELBW infants and it was more frequently associated with PNA.

Table- I

IVH in asphyxiated babies (n=71)

Birth weight	No. of baby	IVH cases	Percentage
Normal	37	02	5.4
LBW	14	02	14.3
VLBW	11	04	36.4
ELBW	09	05	55.6
Total	71	13	18.3

(IVH in total asphyxiated Low birth weight-11/34=32.4%)

Table-II

IVH in nonasphyxiated small babies (n=47)

Birth weight	Number of baby	IVH cases	Percentage
LBW	18	02	11.1
VLBW	16	03	18:8
ELBW	13	03	23.1
Total	47	08	17.0

Table III

Grades of haemorrhage in newborn with risk factors (n=21)

Grade of IVH	Number of cases	Normal BW		LBW		VLBW		ELBW	
		-PNA	+PNA	-PNA	+PNA	-PNA	+PNA	-PNA	+PNA
I	02	0	1	1	0	0	0	0	0
II	06	0	1	1	1	1	1	1	0
III	07	0	0	0	1	1	2	1	2
IV	06	0	0	0	0	0	2	1	3
Total	21	0	02	02	02	02	05	03	05

(Grade III&IV haemorrhage in total VLBW-5/27=18.5%; Grade III&IV haemorrhage in total ELBW-7/22=31.8%)

Discussion :

Intracranial haemorahge is diagnosed on the basis of the history, clinical manifestation, transfontanel cranial ultrasonography, computed tomography and knowledge of the birth weight specific risk of the type of haemorrhage. Infants weighing under 1000gms are at high risk of IVH and should undergo ultrasonographic scan within first 3 days of life[11,12]. The ultrasound scan can also detect cortical haemorrhagic infarction, cystic lesion of PVL, cortical atrophy and the progression of post haemorrhagic hydrocephalus[12,13]. CT scan can be done for those suspected infant in whom USG failed to reveal intracranial haemorrhage or infarction.

Intraventficular haemorrhage is the principal form of intracranial haemorrhage occuring most commonly in preterm infants and the term infants with hypoxic ischaemic encephalopathy[1,9]. Other predisposing factor for IVH include respiratory distress syndromes, pneumothroax and hypertension[11]. Immature blood vessel in the highly vascular periventricular area may be subjected to various forces that together with poor tissue support predispose the premature infant to IVH[9]. PNA in term infants results in intracranial congestion, fluid leak, capillary permeability, endothelial injury and exudation of blood in ventricles[9].

In our study, we got 21 cases of IVH which represents 17.8% of the total neonates. The IVH in term baby with PNA was 5.4%. LBW with PNA suffered (32.4%) more than LBW alone (17%). In Bangladesh Institute of Child & Mother Health, Ahmed et al found 12.9% of IVH in a random sample of 253 newborn[6].

Presently, improvement in perinatal and neonatal care have contributed a reduction in the overall incidence of IVH in VLBW infants from 40-50% in 1970 to 20-25% in 1990. However this decline has been compromised by the increased survival of ELBW infant. Murphy et al observed an overall incidence of 22% IVH in VLBW infants[3]. In Bangladesh the prevalence of LBW is 45-48% and nearly one third of these are VLBW and ELBW baby which should make the figure of IVH quite high[14]. But an incidence of 17.8% is not that alarming in this context. Several reasons may be assumed for this. Some potential cases had not been included in this study. Secondly, USG detection was presumably low as it was done by an imaging expert and not by a neonatologist. In developed countries like UK, cranial ultrasound has become a tool used by paediatrician who are familiar with US appearance of normal variation, developmental anomaly and different pathology[15].

We have found that less the birth weight more severe was the haemorrhage and that Grade III & IV haemorrhage was mostly found in VLBW (18.5%) and ELBW (31.8%) infants which is consistent with other observation[16]. The study report from Australia revealed 32% severe IVH (Grade III & IV) in VLBW children and mean gestational age of those infants were 26±1.9 weeks[3]. We could not determine the gestational age for unreliable history of the mother.

In conclusion, intracranial ultrasonographic scan of high risk babies is an essential tool for evaluation of

IVH and the prevalence of haemorrhage is inversely proportional to the birth weight and directly related to perinatal asphyxia. Further large scale study can be done in this field avoiding the present limitations.

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REVIEW ARTICLE

Typhoid Fever in Children – An Update

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Introduction :

Typhoid fever (enteric fever) is a systemic infection caused by the bacterium, gram negative bacillus, *Salmonella enterica* serotype typhi. It was an important cause of illness and death in the overcrowded and poor sanitary urban conditions of the United States and Europe in the 19th century [1]. Today most of the burden of the disease occur in the developing world, where sanitary conditions remain poor[2].

Typhoid is endemic in many parts of the world where there is inadequate water supply and poor hygiene[3]. It is an important public health problem in Indian subcontinent including Bangladesh[4-6]. Annual incidence of 198 per 100,000 in the Mc Kong Delta region of Vietnam[7] and 980 per 100,000 in Delhi, India[8] have recently been reported. According to the best global estimates there are at least 16 million new cases of typhoid fever each year, with 600,000 deaths[9]. The introduction of chloramphenicol for treating typhoid fever by T Woodward in 1948 heralded the era of modern treatment of typhoid fever which has transferred a severe debilitating and often fatal disease into a readily treatable condition[10,11]. The clinical response to the drug has provided fairly uniform effect throughout the world namely rapid improvement in the patients' general condition followed by defervescence within 3-5 days[11]. This pattern of response become the gold standard against which the performances of subsequently introduced antibiotics have been measured". The wide scale emergence of *S. typhi* strains resistant to chloramphenicol during the 1970s compelled the search for a suitable alternative agent[11]. The emergence of resistance to chloramphenicol and other antimicrobial agents has been a major setback in the treatment of typhoid fever[12].

The present article is intended to provide the recent development of typhoid fever in children as a whole, so that the paediatricians of the country can gather recent knowledge regarding typhoid fever in children.

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Epidemiology

Typhoid fever (enteric fever) is a common infectious disease of the tropical world of which 80% of these cases occur in Asian countries[13]. It may occur sporadically or in epidemics. The organism may survive for many weeks in sewage but in fresh water 90% or more will die in less than four weeks[3].

Typhoid fever is a waterborne and foodborne disorder[2,8]. It is usually transmitted by ingestion of food or water contaminated by faecal matter or urine of carriers excreting S *enterica* serotype typhi[2]. It is a sporadic disease in developed countries that occurs mainly in returning travelers, with occasional point-source epidemics[14]. In endemic areas, identified risk factors for the disease include - eating of food prepared outside the home, such as ice cream or flavoured iced drinks from street vendors[15,16], drinking contaminated water[17], having a close contact or relative with recent typhoid fever[15,18] poor housing with inadequate facilities for personal hygiene[19] and recent use of antimicrobial drugs[15]. Regarding contamination of food two hypotheses have been proposed by some authors, a) by food handlers who are asymptomic carrier *of Salmonella typhi*[20]. b) by irrigation of fruit and vegetables with sewage contaminated, water[21,22]. A case control study revealed a significant association between disease and drinking unboiled spring water[23]. The implication of spring water was supported by the presence of faecal contamination in the spring water source[23].

The illness has a marked seasonality with a peak during the summer months[16]. In a Bangladeshi study[24] isolation rate of S. *typhi* was highest in monsoon and summer seasons and lowest in winter months.

The age incidence of typhoid fever varies widely in different studies in various countries. Some studies say that the highest incidence of typhoid fever is in children in the 8-13 years age group[10,25]. By others, the incidence of typhoid fever peaks between five and 12 years. In Malaysia, the average age at presentation

was 91.3 (range 6-159) months[27]. In a Taiwan study, out of 71 children diagnosed to have typhoid fever 83% were aged 5-15 years[28]. A study in Bangladesh revealed that the majority (54.5%) of S. typhi isolates were from children in first two years of life[24]. Typhoid is common and significant cause of morbidity between one and five years of age[8]. Vertical intrauterine transmission from an infected mother may lead to neonatal typhoid, a rare but severe and life threatening illness[29].

Pathogenesis

The infectious dose of S. enterica serotype typhi in volunteers varies between 1000 and one million organisms[30]. In the small intestine the bacteria adhere to mucosal cells and then invade the mucosa. After penetration the invading microorganisms translocate to the intestinal lymphoid follicles and the draining mesenteric lymph nodes and some pass onto the reticuloendothelial cells of the liver and spleen[2]. The salmonella organisms are able to survive and multiply within the mononuclear phagocytic cells of the lymphoid follicles, liver and spleen[31]. At a critical point which is probably determined by the number of bacteria, their virulence and the host response, the organisms are released from this sequestered intracellular habitat into the blood stream[2]. The incubation period is usually 7-14 days. In the bacteraemic phase the organism is widely disseminated commonly to liver, spleen, bone marrow, gall bladder and Payer's patches of the terminal ileum. Organisms excreted in the bile either reinvade the intestinal wall or are excreted in the faeces[2].

Count of bacteria in patients with acute typhoid fever indicate a median concentration of one bacterium/ml blood (about 66% are inside phagocytic cells) and about 10 bacteria/ml of bone marrow[32,33]. Typhoid induces systemic and local humoral and cellular immune responses but these confer incomplete protection against relapse and reinfection[2].

Clinical presentation

Most of the patients who present to hospitals with typhoid fever are children or young adults from 5-25 years of age[1,34,35]. The onset of bacteraemia is marked by fever and malaise. Patients typically present to hospital towards the end of first week with the symptoms of fever, influenza like symptoms with chills, a dull frontal headache, malaise, anorexia, nausea, poorly localized abdominal discomfort, a dry cough and myalgia with few physical signs[1,34,36,37]. Initially the fever is low grade but it rises progressively and by the second week it is often high (39°-40°C) and sustained [2,3].

Along with fever gastrointestinal symptoms like vomiting, abdominal pain, loose motion are common presentation in children with typhoid fever[3,29,38,39]. Convulsions may occur in children under five years of age[37]. Patients may present with the features of pneumonia or meningitis. Occasionally, they may present with frank neuropsychiatric symptoms resembling catatonic schizophrenia which is more frequent in Africa and Indian subcontinent[2,3]. Patients may be severely agitated, delirious or obtunded but complete stupor or coma is infrequent[2]. These neuropsychiatric presentations range from 10-40% among hospitalized patients with typhoid in lndonesia[40,41] and Papua New Guinea [42] but less than 2% in Pakistan[36] and Vietnam[43].

On examination relative bradycardia is considered as common but not a constant feature[2]. In a study it was noticed in 12 out of 21 patients of typhoid fever [39]. A few rose spots are present in less than 50% cases and some reported in 5-30% of cases[2]. They usually occur on abdomen and chest and rarely on back, arms and legs. A coated tongue, tender abdomen and hepatosplenomegaly are common. In one study hepatosplenomegaly was the most common physical sign observed in children with typhoid fever followed by abdominal tenderness[28]. Another study on typhoid fever in children showed hepatomegaly in 85.3% and splenomegaly in 27.5% cases[27]. In some Bangladesh studies it is shown that palpable liver was present in 58% and palpable spleen was present in 33%[6] cases and hepatosplenomegaly was present in 44.1% cases[44]. Diarrhoea, abdominal pain, distension, coated tongue all are common findings in Bangladesh studies[6,44].

Complications

Complications occur in 10 - 15% of patients and particularly occur in patients who have been ill for more than two weeks[2]. Typhoid ileal perforation is a major problem in developing countries and causes a

high mortality[45]. Some studies revealed that intestinal (usually ileal) perforation is the most serious complication, occurring in 1-3% of hospitalized patients[46,47] . A Nigerian study showed the overall perforation rate of 10.3% in children with typhoid fever[48]. Some authors found that gastrointestinal bleeding is the most common complication occurring in 10% of patients[2]. Another study showed 20.3% of bleeding in the form of haematochesia[48]. Complications of typhoid fever may involve any system of the body[3]. Though isolated cerebellar ataxia or nephritis is rare in enteric fever, it may occur in the second week[13]. Common complications related to urinary tract are cystitis, pyelitis and pyelonephritis but glomerulonephritis is uncommon in enteric fever[13].

Relapse and long term carriers

Relapse may occur in 5-10% patients usually two to three weeks after the resolution of fever[2]. Following the acute disease, some patients continue to excrete the organism in stool and less commonly in urine[3]. Up to 10% of convalescing patients with untreated typhoid excrete S *enterica* serotype typhi in the faeces for up to three months and 1-4% become long term carriers excreting the organism for more than one year[2].

Antimicrobial resistance

Emergence of drug resistance in enteric fever is a major concern for the clinicians. The rapid spread of multi drug resistant (MDR) typhoidfever has posed a great challenge for the treatment of these cases worldwide[49]. Since 1987, MDR strains of *Salmonella typhi* resistant simultaneously to ampicillin, chloramphenicol and trimethotrim- sulphamethoxasole have caused epidemics of severe typhoid fever in Asia and Africa[50]. The first case of chloramphenicol resistant *Salmonella typhi* in Bangladesh was reported in 1982[51], later MDR strains were also isolated in Bangladesh[52,53]. In 1992, a study in ICDDRB showed that 44.1% strains of S. typhi were resistant to first line drugs viz, ampicillin, Chloramphenicol, cotrimoxasole[54]. Another study from Bangladesh Institute of Child Health in 1994 showed that among 236 strains of S. *typhi,* 66.5%, 72.9% and 78.8% were resistant to ampicillin, cotrimoxazole and chloramphenicol respectively[55].

Two intervention studies in Institute of Postgraduate Medicine and Research (IPGMR), Dhaka, Bangladesh showed 39%[5] and 86%[6] *Salmonella typhi* resistant to multi drugs. Some recent studies showed that 80% of salmonella isolates were resistant to amoxycillin, chloramphenicol and cotrimoxazole but all were sensitive to ciprofloxacin and ceftriaxone[38]. Similarly, ampicillin, chloramphenicol, cotrimoxasole resistance was 46%, 39.3% and 44.8% respectively in another study[56]. Surprisingly all the S. *typhi* and S. *paratyphi A* isolates were resistant to the three first line antibiotics (ampicillin, chloramphenicol and cotrimoxazole). All strains with decreased sensitivity to ciprofloxacine showed resistance to ofloxacin and, ciprofloxacin[57]. Now a days, S. *enterica* serotype typhi resistant to ciprofloxacin is not uncommon. In the year 2000, nine strains were detected as ciprofloxacin resistant, which might be influenced by misuse and overuse of ciprofloxacin in the treatment of typhoid fever[58]. Some recent studies pointed out the re-emergence of sensitivity to the first line antibiotics. A study showed significant resistance to ciprofloxacin (55.5%) and to ceftriaxone (44%) and the results pointed towards the re-emergence of sensitivity to chloramphenicol[59]. In another study in the UK showed that 50% *S.typhi* isolates were resistant to chloramphenicol ampicilin and trimethoprim and 33% showed decreased sensitivity to ciprofloxacin[60]. Majority of those patients had recently returned from Indian Sub-Continent particularly Pakistan, India, Srilanka and Bangladesh. All strains with decreased sensitivity to ciprofloxacin were fully sensitive to cephalosporin like ceftriaxone or cefotaxime[60]. Due to significant decrease in isolation of MDR strains some study suggests that cheaper and effective first line of antibiotics may re-emerge as drugs of choice for treatment of typhoid fever in Bangladesh[50]. In a Bangladeshi study yearly analysis of resistance among the strains revealed a remarkable decreasing rate of resistant strains to multidrugs which were 46.5%, 29.7% and 17.8% in 1994, 1995 and 1996 respectively[61].

In an Indian study it has also been seen that 91.6% strains of S.typhi were sensitive to chloramphenicol in 1998 as compared to 71.9% in 1994, which appears that chloramphenicol may re-emerge as the drug of choice for treating typhoid fever[62].

Diagnosis :

The absence of specific symptoms or signs makes the clinical diagnosis of typhoid fever difficult. A fever without evident cause lasting more than one week should be considered typhoid until proved otherwise. Blood and bone marrow cultures both can be used for diagnosis of typhoid fever in children. Though the blood culture is considered as standard diagnostic method, bone marrow is more sensitive than blood culture because of lower numbers of micro organism in blood as compared to bone marrow[32,33]. Bacteria in the bone marrow of typhoid fever patients are less affected by antibiotic treatment than bacteria in the blood. Enteric fever is the only bacterial infection of human for which bone marrow examination is routinely recommended by some authors [35].

Although of limited use as a primary diagnostic test, a raised serum C-reactive protein (CRP) may still have a place as one of a range of features that facilitate assessment of a febrile child in a typhoid endemic area. Choo et al showed serum CRP was highest in culture positive (n=108) children with enteric fever and reflects the immune response to the infection in that group[63].

The ELISA-Ty test has a high reliability for the detection of typhoid fever in children based on the finding of a degree of diagnostic sensitivity as high as 95.45% and 90.91% for IgM and IgG respectively and a diagnostic specificity as high as 93.33% for both IgM and IgG[64].

Application of a dipstick assay for the detection of S. *typhi* specific IgM antibodies on samples collected from S. *typhi* and samples collected from S. *typhi* and S. *paratyphi* culture positive patients revealed the presence of specific IgM antibodies in 43.5%, 92.9% and 100% for samples collected 4-6 days and 6-9 days and >9 days after the onset of fever respectively[65]. The result can be obtained on the same day allowing a prompt treatment and no special laboratory equipment is needed to perform the study.

Currently, the laboratory diagnosis of typhoid fever is dependant upon either the isolation of *Salmonella enterica* serotype typhi from a clinical sample or the detection of raised titre of agglutinating serum antibodies against the lypopolysaccharide (LPS) (O) or flagellum (H) antigens of serotype typhi (the Widal test). The serological assays based on the detection of IgM antibodies against either serotype typhi LPS (ELISA) or whole bacteria (dipstick) had a significantly higher sensitivity than the Widal TO test when used with a single acute phase serum sample (P=or>0.007)[66]. These tests could be of use for the diagnosis of typhoid fever in patients who have clinical typhoid fever but are culture negative or in the region where bacterial culturing facilities are not available [66]. Though the gold standard for the diagnosis of typhoid fever i*s isolation of S. typhi* from blood, bone marrow, stool, urine or any other body fluid, in countries like Bangladesh isolation of organism is often jeopardized by lack of facilities or improper antibiotic use prior to culture. For this reason, laboratory diagnosis' of S *typhi* infection relies heavily on serological test, Widal test[67]. On the basis of cut off value for TO (1 :80) and TH (1 :160) and considering both the agglutinin equally important, sensitivity and specificity of the test were 89% and 97% respectively[67].

Treatment

For hospitalized patients, effective antibiotics, good nursing care, adequate nutrition, careful attention to fluid electrolyte balance and prompt recognition and treatment of complication are necessary to avert death[2]. Ampicillin, chloramphenicol and cotrimoxazole, the first line of drugs for the treatment of typhoid are loosing their efficacy and most of the organisms have developed resistance against these drugs[56]. Until 1950, these drugs were effective in patients with acute infection reducing complications and mortality but since then progressive resistance to one or two of these first line antibiotics have been reported and in 1989 resistance to all three first line antibiotics was observed in Pakistan, India, China and the Arabian Gulf [68-70]. There is strong evidence that fluoroquinolones like ciprofloxacin or ofloxacin are the most effective drugs for the treatment of typhoid fever and these drugs have proved in all age groups and are rapidly effective even with short course (3-7 days)[7I-74]. The fluoroquinolones are associated with lower rate of stool carriage than the traditional first line drugs[71-72]. The use of fluoroquinolones is warranted in the treatment of typhoid fever in some study[75]. Another study showed that the

fluoroquinolones were the most effective drug against multidrug resistant *Salmonella typhi*[76]. Some authors said that ciprofloxacin may not represent a reliable and useful option for treating MDR typhoid fever; ceftriaxone may be an effective alternative for such cases[49]. Some study results revealed that among several drugs defervescence time was least with ceftriaxone and greatest with amoxycillin in all cured children with typhoid fever[38]. Unfortunately, quinolone-resistant strains are often multidrug resistant and the choice of drug is limited to azythromycin and cephalosporins[2]. The third generation cephalosporins (ceftriaxone, cefixime, cefotaxime and cefoperazone) and azithromycin are also effective drugs for typhoid. Some authors opined that oral azithromycin once daily appears to be effective for the treatment of uncomplicated typhoid fever in children[77]. Even though the rapid spread of MDR typhoid fever is a great challenge for the treatment of these cases there are re-emergence of sensitivity to chloramphenicol[59]. Another study suggests that cheaper and effective first line antibiotics may re-emerge as drugs of choice for the treatment of typhoid fever in Bangladesh[50].

If no culture is available knowledge of the likely susceptibility from the available global data may be useful in treating typhoid fever in children[2]. Children with severe typhoid fever characterized by delirium, obtundation, stupor, coma or shock get benefit from the prompt administration of dexamethasone along with fluoroquinolone[2,3]. The dexamethasone should be given at an initial dose of 3 mg/kg by slow intravenous infusion over a period of 30 minutes followed by 1 mg/kg at the same rate every six hours for eight aditional doses[2,3]. High index of suspicion and prompt treatment is highly critical in the treatment of septicaemia in young children[78].

Case fatality

The average case fatility rate is less than1% but it varies considerably among different regions of the world [2]. Among hospitalized patients the fatality rate varies from less than 2% in Pakistan 36 and Vietnam[43] to 30-50% in some areas of Papua New Guinea and Indonesia[40,41]. Case fatality rate is highest among children under one year of age[34,36.37]. Poor outcome is usually related to delay in instituting effective antibiotic treatment[2].

Prevention

The provision of potable water, adequate sanitation and immunization are means to eradicate the disease[45]. In developing countries, reducing the number of cases in general population requires the provision of safe drinking water, effective sewage disposal and hygienic food preparation[9]. Mass immunization had been used successfully in some areas[79]. In developed countries identification of chronic carriers is now less important than previously. Most cases are the result of travel to endemic areas. Travelers in such areas need to take particular care with food and water[2]. Water for drinking should be boiled or bottled, food should be thoroughly cooked and ice cream should be regarded with suspicion[2]. The available Vi (sic) polysaccharide vaccines provide a reliable means in preventing a disease responsible for a significant morbidity and placing a heavy burden on health budgets[80]. The Vi based vaccine is suitable for children over the age of two years and has no serious side effects[2]. In areas where epidemic risk is high mass immunization should be considered during disasters or in refugee camps in combination with adequate provision of safe water and food[81].

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CASE REPORTS

Complete Rectal Prolapse along with Extrusion of Small Gut, A Case Report

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Summary:

A 60 year old man with history of reducible chronic rectal prolapse presented acutely with severe haemorrhage and spontaneous extrusion of whole small intestine through perineum by tearing pelvic diaphragm. Complete rectal prolapse is not an uncommon feature but prolapse of small intestine along with rectum is extremely unusual and has not yet been reported, to our knowledge.

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Introduction

Rectal prolapse is a condition in which mucous membrane & submucosa or even all the layers of the rectal wall descends through the anus & may be found in all ages and sexes. It may be partial or complete (procedentia) and may have acute or chronic presentation. But complete rectal prolapse associated with extrusion of the small gut is very unusual.

In Rectal prolapse the basic defect is deficit in one or more of the supporting structures of the rectum[1]. In chronic & habituated cases the pelvic diaphragm & anal sphincter becomes weak & lax[2].

Case History

A 60 years old Railway driver had habituated reducible complete rectal prolapse for 35 years and dysentery like symptoms for the same duration. But he had no history of chronic cough, straining for urination and no perineal surgery. On 16th November 2002, he developed severe acute episode of dysentery like symptoms, noticed per rectal bleeding and protrusion of gut through the perineum. He was immediately admitted to surgical Unit-2 of Rajshahi Medical College Hospital.

On Examination, the patient was found very pale, anxious and dehydrated. He was rolling on the bed. His clothing's and quilt were soaked with blood. His entire small intestine, the mesentery and rectum were found outside the perineum. The anal sphincter was characteristically patulous.

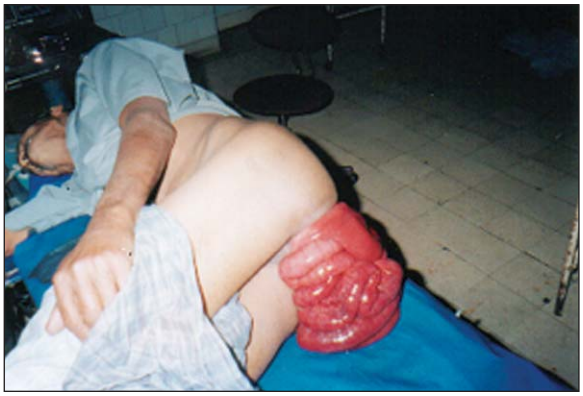

Fig.-1 : *Extrusion of the small gut with complete rectal prolapse. (Right lataral position).*

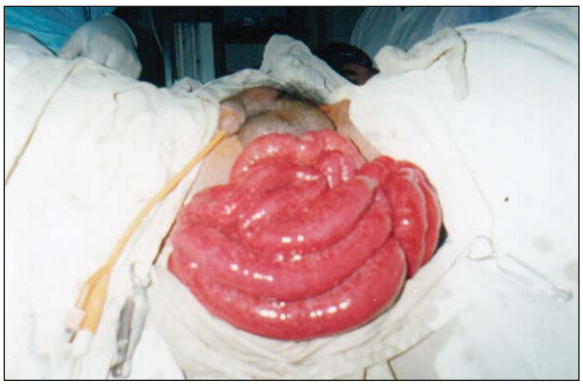

Fig.-2 : *Extrusion of the small gut with complete rectal prolapse. (Lithotomy position).*

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After immediate resuscitation with intravenous fluid and whole blood, he was taken for emergency operation. Two surgical teams approached the patient through abdominal and perineal routes.

The exteriorized gut was cleaned with warm normal saline and reposited by simultaneous pulling and pushing action. Repair of ruptured pelvic floor, Thiersch's operation and pelvic loop colostomy were performed.

The postoperative recovery of the patient was uneventful. He was discharged on fifteenth postoperative day and advised for follow up and further requisite surgical manoeuvre.

Discussion :

Rectal prolapse may be congenital or acquired. Congenital form usually found in children and acquired found in adults and old ages[1]. In children it often commences after an attack of diarrhoea, as a result of severe cough or from weight loss and consequent diminution of fat in ischiorectal fossae[2]. The condition in adults is usually associated with third degree hemorrhoids. In female prolapse of the rectum is commonly associated with urogenital prolapse or after a gynecological operation. In male it may be due to excessive straining for passing urine due to stricture of urethra, BPH or Urinary bladder outlet obstruction or may develop after operation of fistula in ano.

The common complications of rectal prolapse are haemorrhage, ulceration, irreducibility and gangrene of prolapsed part. But extrusion of small gut by perforating the pelvic floor is very unusual; which was found in this patient

In this case anal sphincter & pelvic diaphragm were weak & lax. Moreover, severe acute attack of dysentery ruptured the weak pelvic diaphragm and cause the catastrophe of extrusion of small gut through the perineum.

Surgery in rectal prolapse can be performed by perineal or abdominal approach When ever indicated rectopexy is recommended, & can be performed by laparotomic, laparoscopic & transsacral methods[3,4] when the patient is elderly and very frail, a perineal operation is indicated.

In this case laparotomy was done to replace the extruded part of gut & to repair the pelvic diaphragm & to perform colostomy. Thiersch's manoeuvre was done to prevent immediate prolapse of rectum and to increase strength of anal sphincter and colostomy was performed to give rest to pelvic supports (pelvic diaphragm) and there by facilitate proper healing.

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Acute Intra Vascular Haemolysis After Coil Occlusion of Patent Ductus Arteriosus By Detachable Coil - A Case Report

NNF BEGUM, AU AHMED,

Summary :

A case of acute intra vascular haemolysis after occlusion of patent ductus arteriosus by a detachable coil in a 2 years old child is reported. The ductus size was 3 mm and it was closed with 8 x 3 mm coil. There was tiny residual shunt after deployment of coil. The patient developed haemolysis 6 hours after the procedure. She had massive haemolysis which required blood transfusion everyday. Parent refused to implant another coil for tiny residual shunt. After seven days of the implantation the coil was removed and the patient was referred to surgical team for PDA ligation.

(J Bangladesh Coll Phys Surg 2003; 21 :151-153)

Introduction :

Patent ductus arteriosus (PDA) is one of the commonest congenital heart lesion. The incidence of isolated persistent patancy of the ductus arteriosus has been estimated at 1:2000 to 1:5000 births, which is about 10 to 12 percent of all varieties of congenital heart disease[1,2]. Surgical treatment of PDA has long been established. Closures of PDA in catheterization laboratory have come into regular use in current practice. Several devices have been used including Rashkind PDA occluder Gianturco coil, detachable coils, cardio SEAL, umbrella device and Amplatzer PDA occluder. Complications' like embolization of coil, haemolysis, inability to release coil are often encountered[3,4,5]. We encountered only one case of haemolysis, in our experience in catheterization laboratory of Combined Military Hospital, Dhaka and coil was removed after 7 days of implantation. This patient also had glucose 6 phosphate dehydrogenase deficiency. This is the first case in Bangladesh with such complication and was managed successfully which led to writing this report.

Case Report :

Jannatun Nessa, a two and half years old girl was diagnosed as a case of patent ductus arteriosus (PDA) since 18 months of her age. She had history of recurrent respiratory tract infection (RTI) with poor weight gain. Her parent took her to a paediatrician for this reason and a continuous murmur was detected. She was referred to the paediatric cardiologist of Combined Military Hospital, Dhaka and her initial work up was done. Her chest X-ray showed mild cardiomegaly with plethoric lung fields. Electrocardiography (ECG) showed' left ventricular dominance. Two dimensional and Doppler echocardiography showed a PDA of 2.8 mm size, left to right shunt with peak pressure gradient (PPG) of 66 mm Hg across it. At the time of diagnosis her weight was 6 kg. She was treated with anti failure medicines for the time being to allow her to achieve body weight of eight kg and then to implant a coil inside PDA. Drugs were frusemide, digoxin and potassium. At the age of two and half years her weight reached nine kg and she got admission into paediatric cardiology unit for PDA coil occlusion. Pre catheterization work up were done, which included blood for hematological

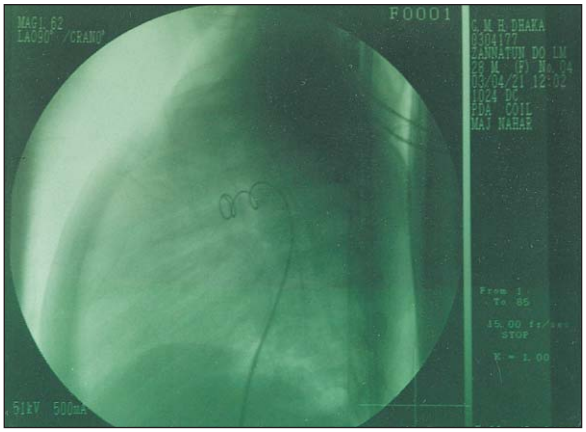

Fig-1 : *PDA Coil is delivered inside ductus with elivery system.*

Pediatric Cardiology Unit of CMH Dhaka.
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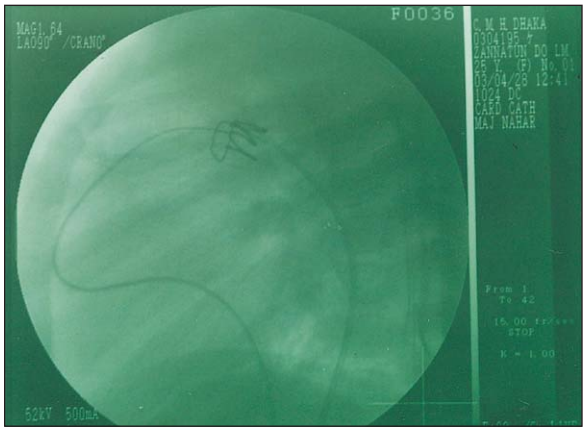

Fig-2: *PDA Coil is Snared with Snare Catheter in Order to take it out.*

profile, platelet count, urea, creatinine, electrolytes, prothombin time, partial thromboplastin time, blood grouping, HbsAg screening etc. All investigations were normal. On 24th April, 2003 Jannatun was taken into the cardiac catheterization laboratory of CMH Dhaka. Her aortogram showed a moderate size PDA, 3 mm in size. A cook detachable coil of 8x3 mm size was selected for her and it was implanted successfully. Total fluoroscopy time was 10 minutes. From 6 p.m of the same day she started passing high colored urine. The color 'darkened to almost black. Acute intra vascular haemolysis with haemoglobinuria was suspected. Blood sample was sent for Hb% (Haemoglobin percentage), Haematocrit, comment on peripheral blood film, serum billirubin, Coomb's test, G6PD (glucose-6-phosphate dehydrogenase), pyruvate kinase level etc. Urine was sent for free haemoglobin and RBC. G6PD was found deficient (90 mu/10^9 erythrocyte). All other investigations were within normal limits on first day.

Haemoglobin came down to 6 gm/dl on 3rd day of the procedure. Urine was positive for free haemoglobin on second day. She required blood transfusion on every day and billirubin was going up. On 3rd day serum billirubin was 6mg/dl. Echocardiography on 2nd day of procedure showed tiny residual shunt. Her father refused the plan to implant another coil for the residual tiny shunt. Because of severe anaemia and raised billirubin, PDA ligation and removal of coil was refused by cardiac surgeon. So removal of coil with snare catheter was planned. She was taken into the cardiac catheterization laboratory again on 31st April. Injection Heparin was given to her in previous 3 days and 'was stopped 12 hours before the procedure.

Procedure : 6 French (Fr) venous sheath was given to right femoral vein and 5 French arterial sheath was introduced to right femoral artery. A 5 Fr snare catheter was used to take out the coil. Aortic end of the ductus arteriosus was closed with a balloon to prevent eblolization of any thrombus to aorta. The pulmonary end of the coil was snared and it was loaded slowly inside the snare catheter. The catheter was then taken out of the sheath. The total fluoroscopy time for the procedure was 30 minutes. Within one hour of the procedure the patient started passing clear urine. She was then referred to the cardiac surgeon for PDA ligation and division. She is now waiting for surgery.

Discussion :

The clinical experience with Trans catheter closure of the PDA using coils has grown rapidly. Though conventional gianturco coil has complications like migration of coils into peripheral vessels, the detachable coil may have same technical benefits to reduce complications[6]. For small ductus single coil is enough but for moderate to large sized ductus simultaneous double or triple coil technique is used[6]. Trans catheter closure of the PDA using amplatzer duct occluder (ADO) is gaining popularity specially for the large ductus[7,8,9,10]. To overcome the drawbacks of various types of devices, both detachable cook coil and Amplatzer ductus occluder (ADO) were used in University Children's Hospital Slovania and 53 (98%) out of 54 patients had complete closure of the duct[11]. Major complications arising from coil occlusion are embolization, acute haemolysis, etc. Late coil migration due to thrombolysis after successful implantation of a coil was also reported[12]. Acute haemolysis after complete occlusion of PDA was reported in a patient[13]. That patient has mild pulmonary stenosis and mechanism of haemolysis was thought to be the presence of metallic spirals in the left pulmonary artery, just beyond stenosis. Simple balloon dilatation of the left pulmonary artery stenosis resulted in complete regression of haemolysis. Severe haemolysis was also

observed with the new Amplatzer duct occluder in one patient[14]. Temporary balloon occlusion of the aortic ampulla was performed 4 days after the initial procedure and haemolysis resolved. In our case cause of haemolysis was possibly the shunting of blood though tiny residual duct after coil occlusion which caused mechanical destruction of RBC. In this case glucose-6-phosphate dehydrogenase was deficient as well, but that was possibly a coincidence only. One study conducted in the Yorkshire heart center, UK detected haemolysis as an important complication following implantation of coil[15]. Haemolysis was detected in 3.6% of 137 procedures in their series and is most likely to occur in young patient with large duct. To avoid complications three things must be considered[16]. The maximum diameter of PDA should be approximately 3 mm, colour flow mapping must show complete obliteration of flow in the catheterization laboratory and the detachable coil system should be used to allow precise placement of coil[16].

Conclusion :

Acute haemolysis following coil occlusion of PDA is not a very uncommon complication. In most of the 'centers it was managed by implanting another coil to block residual shunt. In this case it was not possible as parent refused to place another coil. Removal of a coil from the site of implantation after 7 days of procedure, a challenging job was preformed successfully in cardiac center of CMH Dhaka, which was never reported in any other journal earlier.

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COLLEGE NEWS

(J Bangladesh Coll Phys Surg 2003; 154-157

Examination News :

FCPS Part I, FCPS Part II & MCPS examinations, of July, 2003 were held on Scheduled dates. Result of examinations were announced on the day examinations were held.

2437candidates appeared in FCPS part - I examination in various specialities. Amongst them 44 candidates came out successful. Speciality wise results are as follows :

FCPS Part I Examination

Speciality	No. of candidates appeared	No of candidates Passed
Medicine	659	11
Surgery	480	10
Paediatrics	304	12
Obst. & Gynae	512	02
Ophthalmology	92	01
Otolaryngology	68	00
Psychiatry	16	00
Anaesthesiology	46	00
Radiology	45	03
Radiotherapy	16	01
Dermatology & Venereology	74	03
Physical Medicine	15	00
Dental Surgery	51	00
Haematology	29	00
Biochemistry	03	00
Microbiology	13	00
Histopathology	14	01
Total 17	2437	44

FCPS Part II Examination :

237 candidates appeared in FCPS part II examination in different specialities. 70 candidates could satisfy the board of examiners and are declared to have passed the FCPS Part II examinations of Bangladesh College of Physicians and Surgeons held in July 2003. List of successful candidates are as follows :

Roll No.	Name of candidate	Graduated from	Speciality
004	Dr. Md. Shahedur Rahman Khan	Chittagong Medical College, Chittagong	Medicine
014	Dr. Md. Kabir Uddin	Sir Salimullah Medical College, Dhaka	Medicine
016	Dr. Md. Abdul Ali Mia	Mymensingh Medical College, Mymensingh	Medicine
018	Dr. Mohammad Jashim Uddin	Dhaka Medical College, Dhaka.	Medicine
019	Dr. Md. Abdus Sattar	Chittagong Medical College, Chittagong	Medicine
020	Dr. Hanif Mohammad	Chittagong Medical College, Chittagong	Medicine
029	Dr. Md. Toufiqur Rahman	Dhaka Medical College, Dhaka.	Medicine

Roll No.	Name of candidate	Graduated from	Speciality
032	Dr. Mohammad Masudur Rahman	Dhaka Medical College, Dhaka.	Medicine
033	Dr. A.F. M. Samsul Haque	Mymensingh Medical College, Mymensingh	Medicine
036	Dr. Anisul Awal	Chittagong Medical College,Chittagong.	Medicine
039	Dr. Md. Anwarul Kabir	Sylhet MAG Osmani Medicalo College	Medicine
041	Dr. Md. Fazal Karim	Dhaka Medical College, Dhaka.	Medicine
042	Dr. Abul Ehsan Md. Mohiuddin Osmani	Chittagong Medical College, Chittagong	Medicine
045	Dr. A.K.M. Murshed	Dhaka Medical College, Dhaka	Medicine
047	Dr. Md. Azizur Rahman	Dhaka Medical College, Dhaka	Medicine
050	Dr. Delwar Hossain	Mymensingh Medical College, Mymensingh	Medicine
052	Dr. Mufti Munsurar Rahman	Dhaka Medical College,, Dhaka.	Medicine
054	Dr. Md. Halimur Rashid	Sir Salimullah Medical College, Dhaka	Medicine
061	Dr. S.M. Rokonuzzaman	Mymensingh Medical College, Mymensingh	Medicine
066	Dr. Md. Monjurul Haque	Sir Salimullah Medical College, Dhaka.	Medicine
073	Dr. Md. Abdul Momen	Sylhet MAG Osmani Medical College, Sylhet	Surgery
079	Dr. Mohammad Noor-A-Alam	Jahurul Islam Medical College, Bajitpur	Surgery
082	Dr. Tohid Mohammad Saiful Hossain	Sylhet MAG Osmani Medical College, Sylhet	Surgery
088	Dr. Sadia Sajmin Siddiqua	Dhaka Medical College, Dhaka.	Surgery
090	Dr. Syed Alfasani	Sir Salimullah Medical College, Dhaka.	Surgery
094	Dr. Salma Sultana	Chittagong Medical College; Chittagong.	Surgery
096	Dr. Md. Tanvirul Islam	Sir Salimullah Medical College,, Dhaka.	Surgery
098	Dr. SM Shakhwt Hossain	Mymensingh Medical College, Mymensingh	Surgery
101	Dr. Md. Saidur Rahman	Mymensingh Medical College, Mymensingh	Surgery
106	Dr. Md. Mizanur Rahman	Mymensingh Medical College, Mymensingh	Surgery
109	Dr. Nashir Uddin	Sir Salimullah Medical College, Dhaka.	Surgery
112	Dr. Masuque Ahmed Mushfiqur Rahman	Chittagong Medical College, Chittagong	Surgery
117	Dr. Tapash Kumar Maitra	Chittagong Medical College, Chittagong	Surgery
121	Dr. Md. Hasan Jamal Fakir	Dhaka Medical College, Dhaka.	Paediatrics
135	Dr. Jesmin Sultana	Mymensing Medical College, Mymensingh	Paediatrics
139	Dr. Hasina Sultana	Dhaka Medical College, Dhaka.	Obst. & Gynae
144	Dr. Zakia Parvin	Mymensingh Medical College, Mymensingh	Obst. & Gynae
145	Dr. Afsana Raushan	Mymensingh Medical College, Mymensingh	Obst. & Gynae
149	Dr. Syeda Nazia Akhter	Sylhet MAG Osmani Modedical College	Obst. & Gynae
160	Dr. Homaira Shameen	Rajshahi Medical College, Rajshahi	Obst. & Gynae
161	Dr. Tabassum Ghani	Mymensingh Medical College, Mymensingh	Obst. & Gynae
164	Dr. Dilip Kumar Bhowmik	Sylhet MAG Osmani Medical College, Sylhet	Obst. & Gynae
165	Dr. Jannatul Ferdous	Dhaka Medical College, Dhaka.	Obst. & Gynae
167	Dr. Abida Sultana	Mymensingh Medical College, Mymensingh	Obst. & Gynae
169	Dr. Shahena Akter	Cittagong Medical College, Chittagong	Obst. & Gynae
173	Dr. Hena Rani Barua	Cittagong Medical College Chittagong	Obst. & Gynae
174	Dr. Eti Saha	Rajshahi Medical College, Rajshahi	Obst. & Gynae
177	Dr. Meherun Nessa	Sher-e-Bangla Medical College, Barisal	Obst. & Gynae
180	Dr. Sharmeen Mahmood	Chittagong Medical College, Chittagong.	Obst. & Gynae
181	Dr. Dilruba Zeba	Sher-e-Bangla Medical College, Barisal	Obst. & Gynae
184	Dr. Nargis Akhter	Institute of Post graduate Medicine & Research	Obst. & Gynae
187	Dr. Sohel Ahmed Khan	Sir Salimullah Medical College, Dhaka.	Obst. & Gynae
188	Dr. Sufia Sultana	Sylhet MAG Osmani Medical College	Obst. & Gynae

Roll No.	Name of candidate	Graduated from	Speciality
193	Dr. Jamila Begum	Sylhet MAG Osmani Medical College, Sylhet	Obst. & Gynae
197	Dr. Setara Binte Kasem	Sylhet MAG Osmani Medical College, Sylhet	Obst. & Gynae
204	Dr. Bazlul Bari Bhuiyan	Sylhet MAG Osmani Medical College, Sylhet	Ophthalmology
207	Dr. Sheikh Hasanur Rahman	Dhaka Medical College	Otolaryngology
214	Dr. Md. Abdus Samad	Dhaka Medical College, Dhaka.	Anaesthesiology
/220	Dr. Md. Rabiul alam	Mymensingh Medical College, Mymensingh	Anaesthesiology
222	Dr. Hasina Begum	Dhaka Medical College, Dhaka.	Anaesthesiology
223	Dr. Md. Mozaffer Hossain	Mymensingh Medical College, Mymensingh	Anaesthesiology
226	Dr. S.M,A. Al-Muid	Mymensingh Medical College, Mymensingh	Radiology
227	Dr. Naheed Rukhsana	Sir Salimullah Medical College, Dhaka.	Radiotherapy
228	Dr. S. M. Humayun Kabir	Sir Salimullah Medical College, Dhaka	Radiotherapy
/229	Dr. Yasmin Hossain	Dhaka Medical College, Dhaka.	Dermatology & V.
230	Dr. Fahmida Hafez	Institute of Post-graduate Medicine & Research	Physical Medicine
233	Dr. Md. Belayet Hossain	Rangpur Medical College, Rangpur	Haematology
/236	Dr. Mahjabeen Ara	Sir Salimullah Medical College, Dhaka	Biochemistry
237	Dr. Md. Mujubul Rahman Howlader	Institute of Post graduate Medicine & Research,	Conservative Dentistry

MCPS Examination :

275 candidates appeared in MCPS examinations in different specialities. 54 candidates could satisfy the board of examiners and are declared to have passed the MCPS examinations of the Bangladesh college of Physicians & Surgeons held in July 2003. The list of Candidates are as follows

Roll No.	Name of candidate	Speciality
006	Dr. Aloke Kumar Raha	Medicine
021	Dr. Saleh Mohammad Shahedul Islam	Medicine
023	Dr. Mohammad Abdul Malek	Medicine
025	Dr. Mohammed Harun-Or-Rashid	Medicine
034	Dr. Biswajit Datta	Medicine
039	Dr. Rajib Nayan Chowdhury	Medicine
043	Dr. Dr. Samsul Arefin Mohammad Masihuzzaman	Medicine
045	Dr. Biplob Kumar Roy	Medicine
046	Dr. Mustafizul Aziz	Medicine
055	Dr. Sambhu Kumar Mallick	Medicine
057	Dr. Sunil Kumar Biswas	Medicine
061	Dr. Kazi Shahnboor Alam	Medicine
067	Dr. Md. Azman Ali	Surgery
074	Dr. Md. Al Amin Salek	Surgery
079	Dr. Md. Abdul Mobin Chowdhury	Surgery
098	Dr. Md. Ibrahim Khalil	Paediatrics
101	Dr. Gulshan Ara Begum	Paediatrics
107	Dr. Mohammed Mahbubul Islam	Paediatrics

Roll No.	Name of candidate	Speciality
110	Dr. Farzana Islam	Paediatrics
113	Dr. Salma Jahan	Paediatrics
116	Dr. Md. Anwar Hossain	Paediatrics
118	Dr. Md. Abdul Hai Mia	Paediatrics
122	Dr. Selina Akhter	Obst. & Gynae
128	Dr. Betheeka Roy	Obst. & Gynae
148	Dr. Yasmin Shammi Ahmed	Obst. & Gynae
162	Dr. Beauty Paul	Obst. & Gynae
464	Dr. Farzana Banu	Obst. & Gynae
169	Dr. Most. Fatima Dolon	Obst. & Gynae
177	Dr. Nasima Shaheen	Obst. & Gynae
182	Dr. Begum Ainun Nahar	Obst. & Gynae
187	Dr. Nazlima Nargis	Obst. & Gynae
191	Dr. Md. Munirul Islam Khan	Ophthalmology
192	Dr. Md. Ismail Hossain	Ophthalmology
203	Dr. S.M. Mesbah Uddin Ahmad	Otolaryngology
210	Dr. Md. Abdul Mannan Shaikh	Anaesthesiology
211	Dr. Mostafa Nuruzzaman	Anaesthesiology
213	Dr. Md. Abul Kalam Azad	Anaesthesiology
214	Dr. Suraiya Shireen	Anaesthesiology
215	Dr. Subrata Kumar Mondal	Anaesthesiology
217	Dr. Md. Idris Ali	Anaesthesiology
221	Dr. A.N.M. Naushad	Anaesthesiology
225	Dr. Rafiqul Alam Khan	Anaesthesiology
226	Dr. Ashish Ranjan Roy Choudhury	Anaesthesiology
227	Dr. Sheikh Rukunuddin Ahmed	Anaesthesiology
231	Dr. Md. Harun Or Rashid	Anaesthesiology
232	Dr. Md. Taifur Rahim	Anaesthesiology
241	Dr. S. M Anwar Sadat	Dental Surgery
245	Dr. Md. Abdus Samad Al-Azad	Forensic Medicine
46'2	Dr. Razia Sultana	Clinical Pathology
263	Dr. Md. Monirul Hoque	Clinical Pathology
265	Dr. Md. Iqbal Karim	Clinical Pathology
268	Dr. Monwar Tarek	Clinical Pathology
271	Dr. Md. Monirul Huq	Clinical Pathology
274	Dr. Quamrunnisa	Clinical Pathology