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CONTENTS

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2. Parkin DM, Clayton D, Blook RJ, Massyer E, Fried HP, Iranov E, et al. Childhood Leukaemia in Europe after Chernobyl : 5 years follow-up. Br J Cancer 1996; 73 : 1006-12.

Chapter in a Book :

1. Phyllyps SJ, Whisnant JP. Hypertension and Stroke. In : Lurgh JH, Brennes BM, editors. Hypertension : Pathophysiology, diagnosis and management. 2nd ed. New York : Raven Press; 1995. p. 465-78.

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ORIGINAL ARTICLES

PRE AND POSTMENOPAUSAL CHANGES OF BONE MINERAL DENSITY: A COMPARATIVE STUDY DONE BY DUAL ENERGY X-RAY ABSORPTIOMETRY

M BEGUM[1], MI PATWARY[2], MA AHBAB[3], MH KHAN[4], AI CHOWDHURY[5], F BARI[6]

Abstract:

Background: Accelerated decline of bone mass occurs in women after the menopause, and might lead to excessive bone resorption and eventually to osteoporosis.[1] To find out the changes of bone mineral density (BMD) before and after menopause, the shown was undertaken.

Materials and Methods: This comparative study was conducted in the Department of Medicine, Sylhet M A G Osmani Medical College Hospital, Sylhet during July 2008 to June 2009. Forty postmenopausal women; monthly income and BMI matched 40 premenopausal women were selected according to inclusion and exclusion criteria. BMD of lumber vertebrae and femoral neck was determined using Dual energy x-ray absorptiometry (DXA) method (Norland XR 46, Pencil beam).

Results: The parity of the postmenopausal women was significantly higher than that of premenopausal women (6.9 ± 2.6 vs 3.1± 1.5; p<0.01). The body weight was significantly lower in postmenopausal women than that of premenopausal women (54.1 ± 8.3 kg vs 61.0 ± 9.7 Kg; p<0.01). The height was significantly lower in postmenopausal women than premenopausal women (148.2 ± 5.7cm vs 153.4 ± 6.4cm; p<0.01). The BMD was lower in postmenopausal women than premenopausal women in lumber vertebrae (0.68 ± 0.13 gm/cm^2 vs 0.94 ± 0.03 gm/cm^2; p <0.01) and also in femoral neck (0.63 ± 0.12 gm/cm^2 vs 0.84 ± 0.14 gm/cm^2; p < 0.01). A significant positive correlation was present between BMD and height (r=0.512; p<0.05); and weight (r=0.489; p<0.05); and a negative correlation between BMD and age (r=-0.408; p<0.05); parity (r=-0.456; p<0.05) and years since menopause (r=-0.350; p<0.05).

Conclusion: The BMD was significantly lower in postmenopausal women than that of premenopausal women and negative correlation was present between BMD and age, parity and years since menopause.

Keywords: *Menopause, Bone mineral density.*

Introduction:

Over the past 10 years, osteoporosis has emerged as a major clinical challenge for physician and patients, with regard both to its prevalence and to the morbidity and mortality of associated fracture.[2]

Osteoporosis affects an estimated 75 million people in Europe, United States of America (USA), and Japan.[3] Osteoporotic fractures occurs 1 in 3 women as well as 1 in 5 men over 50 years of age.[4-6]

The most important risk factor for bone loss in women is the menopause; Women loss about 50% of their trabecular bone and 30% of their cortical bone during the course of their lifetime, about half of which is lost during the first 10years after the menopause.[7, 8]

A 10% loss of bone mass can double the risk of vertebral fractures, and 2.5 times greater risk of hip fracture.[9] Their impact on quality of life can be profound as a result of loss of self-esteem, distorted body image, depression and activities of daily living.[10-14]

In an Indian study among women aged 30-60 years and low income groups showed the bone mineral

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density (BMD) at all the skeletal sites were much lower than that of developed countries.[15]

Bone mineral density in postmenopausal women was significantly lower than pre-menopausal women in a Bangladeshi study using a bone densitometer Single photon X-ray absorptiometry (SXA).[16]

Evidence suggests that many women who sustain a fragility fracture are not appropriately diagnosed and treated for probable osteoporosis. [17,18]

This study had been conducted to find out the changes of BMD before and after menopause with a view to detection of low BMD at an early stage gives an opportunity to intervene timely and will decrease the health hazard associated with it.

Material and Methods:

This cross-sectional comparative study was carried out among the patients attending in the Department of Medicine, Sylhet M A G Osmani Medical College Hospital and Centre for Nuclear Medicine and Ultrasound, Sylhet during the period from 1st July 2008 to 30th June 2009 with a view to find out the changes of BMD before and after menopause. For this purpose 40 participants aged between 51 to 70 years with body mass index between >18.5kg/m^2 to d" 30 kg/m^2 and non-smoker were included as case (Postmenopausal) and 40 BMI and socioeconomic status matched premenopausal women aged between 31-50 years were taken as control (premenopausal). Consecutive and convenient samples were collected. Secondary causes of decreased bone mineral density were excluded. Primary outcome variable was level of bone mineral density and secondary variables age, parity, height, weight and years since menopause.

Detailed history of women including age, parity, socioeconomic status, education, smoking was taken. Detailed menstrual history of the women were taken considering age of menarche, age of menopause, duration since menopause, type of menopause (natural or surgical) Women were enquired about any history of low back pain, height loss, recurrent fall, immobilization, any disease that known to affect bone metabolism at present or in past. Detailed history of medications was noted. Measurement of height (in meter), weight (in kg) and BMI (kg/m^2) were done in each patient. Systemic examination was done routinely. BMD was measured using DXA(Norland XR 46, Pencil beam) .

Statistical analysis

Data was processed and analyzed with the help of Statistical Package for Social Science (SPSS) software version 12. Mean and standard deviation were calculated for continuous data and percentage for categorical data. Unpaired t-test was done for comparison of continuous variable and Chi-square (χ^2) test for comparison of categorical variable to see the significance of difference. Relationship of variables was seen by multiple logistic regression analysis. P value <0.05 will be taken as significant.

Ethical issues

The institutional ethical committee of Sylhet M A G Osmani Medical College, Sylhet approved the study protocol before commencement of the study. Informed written consent of each participant was taken before enrollment.

Results

The mean parity was 6.88 ± 2.58 in the postmenopausal group and 3.10 ± 1.48 in the premenopausal (control) group (p < 0.01) (table-I).

In the postmenopausal group, 22 (55.0%) were illiterate, 12 (30.0%) had primary level, 4 (10.0%) had secondary level, 1 (2.5%) had higher secondary level and 1 (2.5%) had graduation or above in their education level; where as in the premenopausal group, 20 (50.0%) were illiterate, 10 (25.0%) had primary level, 4 (10.0%) had secondary level, 4 (10.0%) had higher secondary level and 2 (5.0%) had graduation or above in their education level (p=0.325) (table-I).

Eighteen (45.0%) had monthly income of less than 5000.00 taka, 12 (30.0%) had monthly income of 5000.00 to 7000.00 taka and 10 (25.0%) had monthly income of more than 7000.00 taka in the postmenopausal group; where as in the premenopausal group, 15 (37.5%) patients had monthly income of less than 5000.00 taka, 13 (32.5%) had monthly income of 5000.00 to 7000.00 taka and 12 (30.0%) had monthly income of more than 7000.00 taka (p=0.875); indicating the study was monthly income matched (table-I).

In the postmenopausal group, the mean height of the patients was 148.15 ± 5.69 cm; whereas the mean height of the premenopausal group was 153.35 ± 6.43 cm (p<0.01) (table-I).

The mean body weight of the postmenopausal group was 54.05 ± 8.31 Kg; where as the mean body weight of the premenopausal group was 61.03 ± 9.74 Kg (p=0.01) (table-I).

The mean BMI was 23.88 ± 3.35 Kg/m^2 in the postmenopausal group and 25.28 ± 3.27 Kg/m^2 in the premenopausal group (p>0.05); suggesting BMI matched study (table-I).

Table-I
Distribution of the patients by baseline characteristics

Baseline characteristics	Postmenopausal group (n=40)	Premenopausal group (n=40)	p value
Parity (mean ± SD)	6.875 ± 2.580 (range 2 to 12)	3.100 ± 2.580 (range 0 to 7)	<0.01*
Education			
Illiterate	22 (55.0)	20 (50.0)	0.325 †
Primary	12 (30.0)	10 (25.0)	
Secondary	4 (10.0)	4 (10.0)	
Higher secondary	1 (2.5)	4 (10.0)	
e" Graduate	1 (2.5)	2 (5.0)	
Monthly income (in taka)			
<5000	18 (45.0%)	15 (37.5%)	0.875†
5000-7000	12 (30%)	13 (32.5%)	
>7000	10 (25.0%)	12 (30.0%)	
Height (mean ± SD)	148.15 ± 5.69 (range 140 to 160)	153.35±6.43 (range 139 to 165)	< 0.01*
Weight (mean ± SD)	54.05 ± 8.31 (range 40 to 72)	61.03 ± 9.74 (range 40 to 80)	<0.01*
BMI (mean ± SD)	23.88 ± 3.35 (range 19 to 29)	25.28 ± 3.27 (range 20 to 29)	>0.05*

*Unpaired t-test and †c^2 Chi-Squre test were applied to analyse the data.
n = total number.
SD = Standard deviation.

BMD in lumber vertebrae was nearly stable in premenopausal women (31-50 years), but decreased sharply in postmenopausal women. Distribution of BMD in lumber vertebrae in different age group was shown in figure-1.

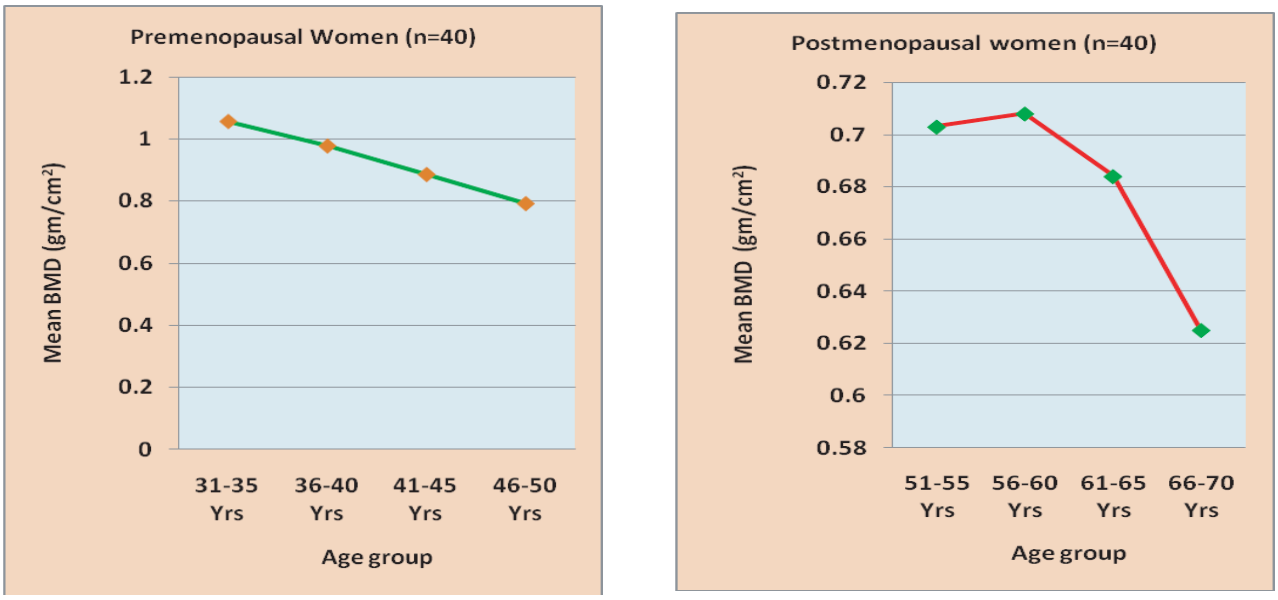

Fig.-1: *Distribution of BMD in lumber vertebrae in different age group*

BMD in femoral neck was nearly stable in premenopausal women (31-50 years), but decreased sharply in postmenopausal women. Distribution of BMD in femoral neck in different age group was shown in figure-2.

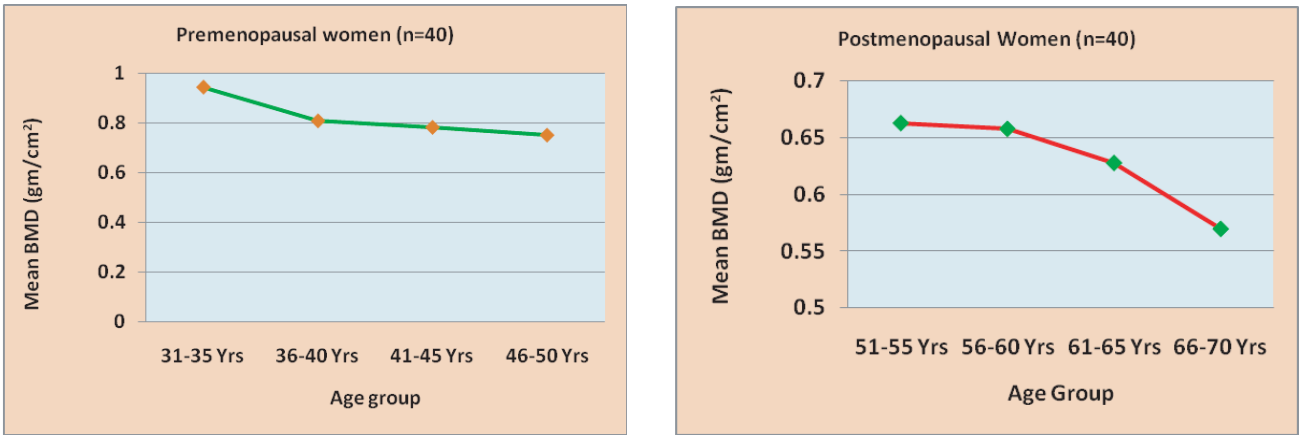

Fig.-2: *Distribution of BMD in femoral neck in different age group*

In the postmenopausal group, the mean BMD in lumber vertebrae was 0.68 ± 0.13gm/cm^2; whereas the mean BMD in lumber vertebra was 0.94 ± 0.03 gm/cm^2 in the premenopausal group ($p < 0.01$) (table-II).

The mean BMD in femoral neck was 0.63±0.12 gm/cm^2 in the postmenopausal group; whereas the mean BMD in femoral neck was 0.84±0.14 gm/cm^2 in the premenopausal group ($p < 0.01$) (table-II).

Data were presented as mean ± SD. Comparison was done between groups by * unpaired t test.

In the postmenopausal group, BMD was osteoporotic level in 21(52.5%), osteopenic level in 8 (20.0%) patients, combined in 9 (22.5%) and normal level in 2 (5.0%) patients; where as in the premenopausal group, osteoporotic level was in 5 (12.5%), osteopenic level in 17 (42.5%), combined in 2(5.0%) and normal level in 16 (40.0%) patients ($p<0.001$) (table-III).

Table-II

Distribution of the patients by BMD

BMD (gm/cm^2)	Postmenopausal group (n=40)	Premenopausal group (n=40)	*p value
Lumber vertebra	0.68 ± 0.13 (range 0.42 to 1.02)	0.94 ± 0.03 (range 0.51 to 1.27)	< 0.01
Femoral neck	0.63±0.12 (range 0.45 to 0.91)	0.84 ±0.14 (range 0.47 to 1.02)	< 0.01

Table-III

Distribution of patients by BMD (Based on WHO Criteria)

BMD	Study group		*p value
	Postmenopausal women (n=40)	Premenopausal women (n=40)	
Normal	2 (5.0)	16 (40.0)	<0.001
Osteopenia	8 (20.0)	17 (42.5)	
Osteoporosis	21 (52.5)	5 (12.5)	
Combined	9 (22.5)	2 (5.0)	

* WHO criteria (by T-score)
Normal: T-score of -1.0 or above
Osteopenia: T-score of -1.1 to 2.4
Osteoporosis: T-score of -2.5 or below

* Combined (either osteoporosis of LV and osteopenia of FN or osteoporosis of FN and osteopenia of LV).

* χ^2 Chi-square test was applied to analyse the data.

Table-IV

Correlation of bone mineral density and selected variables -

Variables	BMD	Age	Parity	Menopause	Height	Weight
BMD	1.000	-0.408 *	-0.456*	0.350*	0.512**	0.489*
Age	0.408*	1.000	-0.998**	0.988**	0.992**	0.990**
Parity	0.456*	-0.998**	1.000	0.985	0.996	0.994**
Menopause	0.350*	0.988**	0.985	1.000	-0.972**	-0.969**
Height	0.512*	0.992**	0.996	-0.972**	1.000	-0.989**
Weight	0.489*	0.990**	0.994**	-0.969**	-0.989**	1.000

Regarding correlation between BMD and selected variables showed that a significantly negative correlation was present between BMD and age (r=-0.408; p<0.05), parity (r=-0.456; p<0.05), years since menopause (r=-0.350; p<0.05). But a significantly positive correlation was present between BMD and height (r=0.512; p<0.01), weight (r=0.489; p<0.05) (table-IV).

Discussion

Osteoporosis and low bone marrow density are significant risk factors for morbidity and mortality in older adults. Not only it gives rise to morbidity but also markedly diminishes the quality of life of women after menopause.[19]

The parity of the postmenopausal women was significantly greater than the premenopausal women (6.88 ± 2.58 vs 3.10 ± 1.48; p< 0.01). Similar finding was observed in the study of Chowdhury.[6] Premenopausal women have got chance of having children in future which may cause this disparity. In the present study a significantly negative correlation was present between BMD and parity (r=-0.456; p<0.05). This result was correlated with the study conducted by Chowdhury .[16]

In postmenopausal group 55.0% patients were illiterate, 30.0% had primary level, 10.0% had secondary level; and higher secondary level and graduation or above constituted 2.5% each. In this regards Gur et al, [20] found that prevalence of osteoporosis had an inverse relationship with the level of education, 18.6% for the most educated to 34.4% for the no educated women.

The height of the patients was significantly lower in postmenopausal group than that of premenopausal group (148.15 ± 5.69 vs 153.35 ± 6.43; p<0.01) in this study, that also reported by Lau et al,[21] (149.3 ± 6.4 cm vs 153.5 ±5.7cm; p<0.001) in their study. a significant positive correlation was found between height and the Bone mineral density (r=0.512; p<0.05) that was similar to the study of Douchi[22] .

The weight was significantly lower in postmenopausal group than that of premenopausal group (54.05 ± 8.31kg vs 61.03 ± 9.74 Kg; p=0.001). This result was supported by Lau et al,[21] 53.8 ± 10.9 kg vs 56.0 ± 9.0 kg; p<0.01). Chowdhury and his group,[16] found the mean body weight was 48.2 ± 10.8 Kg in their postmenopausal women and 49.9 ± 9.4 years in premenopausal women; but the difference between the groups was not significant (p=0.23).

Bone mineral density had a positive correlation with body-weight (r=0.489; p<0.05) in the current study. This result was similar to the study of Chowdhury,[16] that bone mineral density had a positive correlation with body-weight (p<0.05).

The mean BMI was 23.88 ± 3.35 Kg/m^2 in the postmenopausal women and 25.28 ± 3.27 Kg/m^2 in premenopausal women. The BMI of the patients in both groups were almost similar statistically (p>0.05). No significant difference was found between postmenopausal and premenopausal women regarding BMI reported by Chowdhury (p=0.79).[16]

The bone mineral density in lumber vertebrae was 0.68 ± 0.13 in postmenopausal women and 0.94 ± 0.03 in the premenopausal women (p < 0.01). This finding was correlated with Lau et al,[21] that the bone mineral density in lumber vertebrae was 0.70 ± 0.16 in postmenopausal women and 0.96 ± 0.12 in the premenopausal women.

The bone mineral density was 0.63± 0.12 in postmenopausal women, and 0.84 ± 0.14 in the premenopausal women in the femoral neck (p< 0.01). This result was also supported by Lau et al that the bone mineral density was 0.55 ± 0.12 in postmenopausal women, and 0.80± 0.10 in the premenopausal women in the femoral neck.

A significant negative correlation was found between BMD and age (r=-0.408; p<0.05 and this finding was correlated with the study of Chowdhury [16] that a significant negative correlation was present between BMD and age (p<0.001).

A significant negative correlation was found between BMD and years since menopause (r=-0.350; p<0.05). This result was supported by the study of Douchi,[22] and Enchev.[23] A strong negative correlation was found between bone mineral density and years since menopause (p<0.001) in the study of Douchi,[22] and a weak negative correlation found between bone mineral density and years since menopause in the study of Enchev et al.[23]

In the present study bone mineral density was osteoporotic level in 21(52.5%) patients, osteopenic level was in 8 (20.0%) patients, combined in 9 (22.5%) and normal level in 2 (5.0%) patients in the postmenopausal women; whereas in premenopausal group, osteoporotic level was in 5 (12.5%) patients, osteopenic level was in 17 (42.5%) patients, combined in 2 (5.0%) and normal level was in 16 (40.0%) patients. The level of bone mineral density between the groups was statistically significant (p<0.001). In this regards Keramat et al,[24] reported that the prevalence of lumber spine osteoporosis and osteopenia in the postmenopausal women was 26.5% and 50% respectively.

In conclusion, the bone mineral density was significantly lower in postmenopausal women than premenopausal women both in lumber and femoral neck; and negative correlation was present between bone mineral density and age, parity and years since menopause.

Recommendation:

Considering the findings of this study the following recommendations are made:

- A study should be conducted using large sample size in the community level to find out the magnitude of the condition in general population.
- A study involving multicentre with random sampling should be conducted to determine any disparity of bone mineral density through out the country.

Acknowledgement:

This study was done in as a part of thesis in partial fulfillment of the requirement for Internal Medicine MD Part III examination in Sylhet MAG Osmani Medical College Hospital, Sylhet, Bangladesh.

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PRESENTATION OF BILIARY ASCARIASIS : A STUDY OF 30 CASES

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Introduction

Ascaris Lumbricoides, the round worm is one of the commonest and most widespread human parasites. It has been estimated that more than one billion people are infected and that it causes around 6000 death per annum [1]. In Asia it was estimated to affect more than 488 million people, in Europe 45 million, in Africa 49 million and in North America 3 million. Thus, possibly one out of every four people in the world's population is infected[2]. The incidence of Ascariasis varies widely from region to region including region within Asia. It is low in central Asian Republics where high temperature and widespread desert areas are unfavorable to the transmission of helminthes but in humid areas infection may be very common. In Africa infection rates up to 95% of the population have been reported[2].

Intestinal Ascariasis is very common condition in our country although there is no population based representative data. It thrives under condition of poor sanitation, where warm, humid soil facilitates embryonation of the eggs in the environment[3]. Bangladesh is one of the under developed country of the world lying in subtropical zone. The literacy rate here is very low, so also is the knowledge of sanitation and awareness of the disease and in addition living standard of majority of the people are also low. The vast majority of the people living in rural areas depend on agriculture as a mean for earning, where faeces are used as agricultural fertilizer, occupation presents a very important problem in Ascariasis[2]. Because of overcrowding in town of non industrialized country where planning has been unable to keep up with the population increase, the prevalence of infection may be higher in urban than in rural areas[3].

Infestation with Ascaris Lumbricoides is endemic and prevalent in Asia, Africa and South America. Intestinal Ascariasis is also very common condition in our country.

Bangladesh is one of the underdeveloped countries of the world lying in subtropics zone. The standard of living of the majority of the people of Bangladesh is low in addition.

The knowledge of sanitation and awareness of disease and consequences of the disease are minimum. A lion share of the people living in the villages depends on agriculture as mean of earning. In addition, environmental factors and lack of basic sanitation favour the growth of parasites mainly Ascaris Lumbricoides all over the country.

The mature worm inhabitants in the gastrointestinal tract commonly causes abdominal discomfort or colic and may be vomited or passed per rectum. An entangled mass of worm, from a bolus may produce intestinal obstruction, appendicitis, perforation of pre-existing ulcer of stomach and duodenum[4]. In case of heavy infestation it contributes to malnutrition.

Migration of the worm into the biliary tree and pancreatic duct via Ampulla of Vater giving rise to upper abdominal colic, nausea, vomiting, pyogenic cholangitis, stone formation around the ova of dead adult worm, obstructive jaundice, ductal stricture of even liver abscess secondary to infection and obstruction of the bilary tree. Uncommonly acute pancreatitis may result from pancreatic duct obstruction by the round worm. Even cholangiocarcinoma may develop.

Biliary complication of round worm is common in Bangladesh as well as in China & India[5]. It is also found in certain parts of USA where immigration from southeast asia are more. But biliary complications of round worm are not well documented in Bangladesh some sporadic expect case reports.

Aim of the study

The aim of the study is to see the clinical profile of biliary ascariasis in our country.

Materials and Methods

A total of thirty (30) patients were included in this study depending on their ultrasonographic findings

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irrespective of their age, sex and occupation. The cases were collected from Dhaka Medical College Hospital, BSMMU and different private hospitals of Dhaka city from June 2004 to June 2005.

Ultrasonographic Appearance of Biliary Ascariasis

Following were the criteria set for diagnosis of Biliary Ascariasis ultrasonographically.[6]

- On longitudinal scan : Linear echogenic structures without acquastic shadowing.
- On transverse scan : A tube within a tube – " Target sign" or " Bull's eye Sign".
- Dilatation of biliary tree.
- When infestation is heavy, multiple worms may lie adjacent to each other within a distended bile duct, giving a spaghetti like appearance.
- During scan the live moving worm could be seen also.

Observations and Results

Sex distribution of the patients

Out of the first thirty (30) patients, eleven (11) were male and the rest nineteen (19) were female. So, about 37% of this patients were male and 63% were female.(Table - 1)

Table – I

Sex distribution of patients (n = 30)

Sex	No	Percentage(%)
Male	11	37
Female	19	63
Total	30	100

Age distribution of the patients

The age range of the patients included in this study was from 19 years to 70 years. Eighteen (18) patients were within the range of 20 – 40 years (60%), Eleven (11) patients were more than forty (40) years old (36.6%) and one (01) patient was less than twenty (20) years old (3.33%). (Table - II)

Table – II

Age distribution of the patients

Age group(years)	No	Percentage(%)
>40	11	36.66
20 - 40	18	60.00
<20	01	03.33
Total	30	100.00

Occupation of the patients

Among thirty (30) patients, eighteen (18) were housewife (60%) and seven (07) were small businessman (23.3%), three (03) were service holder (10%) and two (02) were day labourer (6.66%). Among this female out of 19 (nineteen) all but one (01) was housewife (95%). That one (01) was a school teacher (0.05%), (Table-III)

Table – III

Occupation of the patients (N=30)

Occupation	No	Percentage(%)
Housewife	18	60
Businessman	07	23.3
Service holder	03	10
Day laborers	02	6.66
Total	30	100.00

Table – IV

Occupation among the female patients (N=19)

Occupation	No	Percentage (%)
Housewife	18	95
Service	01	05
Total	19	100

Economic status of the patients

Out of thirty (30) patients, twenty (20) belong to lower middle class (66.6%) and rest ten (10) belong to lower economic class (33.3%).(Table - IV)

Table – V

Economic status of the patients (n=30)

Economic status	No	Percentage (%)
Lower middle class	20	66.66
Lower class	10	33.33
Total	30	100.00

Symptom of the patients

Main presenting symptoms were recorded in all patients. Recurrent upper abdominal pain was the main presenting symptom in all thirty (30) patients (100%). followed by vomiting in fourteen (14) patients (46.6%), fever in nine (09) patients (30%), jaundice in three (03) patients (10%).(Table - VI)

Table – VI
Symptoms of the patients (n=30)

Symptoms	No	Percentage (%)
Recurrent upper abdominal pain	30	100
Vomiting	14	46.6
Fever	09	30
Jaundice	03	10

Physical signs of the patients

There was no physical findings in nineteen (19) patients (63.3%), seven (07) patients showed fever (30.1%), four (04) showed jaundice (13.3%) and another four (04) showed upper abdominal tenderness (13.3). (Table - VII)

Table – VII
Physical signs of the patients (n=30)

Physical findings	No	Percentage (%)
No findings	19	63.3
Fever	09	30
Jaundice	04	13.3
Tender abdomen	04	13.3

Findings in stool routine examination

Out of thirty (30) patients, only two (02) patients showed ova of ascariasis Lumbricoides in their stool (6.66%) and in rest twenty eight (28) there was no such are (93.3%). (Table - VIII)

Table – VIII
Findings in stool routine examination (n=30)

Findings	No	Percentage (%)
Ova	02	6.66
Nil	28	93.3
Total	30	100.00

Total count of WBC

In total count of WBC, it was found elevated in six (06) patients (20%) and in rest twenty four (24) patients it was found within normal range (80%).(Table - IX)

Table – IX
Total count of WBC (n=30)

Findings	No	Percentage (%)
Elevated	06	20
Normal range	24	80
Total	30	100.00

Findings in GUIT endoscopy (Upper GIT endoscopy)

In endoscopic examination of upper Gastrointestinal tract (UGIT) five (05) patients showed presence of round worm in duodenum (16.6%) and in rest twenty five (25) patients there was no such findings (83.3%).(Table-XI)

Table – X
Findings in GUIT endoscopy (n=30)

Findings	No	Percentage (%)
Round worm	05	16.66
Normal	25	83.33
Total	30	100.00

Discussion:

In this study a total of thirty (30) Patients were included depending on their ultrasonographic findings. Out of these thirty (30) patients, 19 were female and 11 were male. So, female patients outnumbered the male. Similar observation was also found by others[4,7]. WHO expert committee also reported that in some countries where there is no sex difference in prevalence among children of school age the figures in adult female are significantly higher than in male which may be attributable to greater contamination in immediate vicinity of the house. In addition, poor literacy with poor hygienic senses, caring of the babies, cleaning of the excreta etc. may be the other factors responsible for higher prevalence among the female.

The age of the patients shown in this study was between 19 to 70 years, although it was more common between 20 – 40 years. Similar observation is seen by others,[7] but Hossain et al. revealed it to be more common among under age of 20 [4]. WHO expert committee also reported it to be more common in young age groups[2]. This is probably due to lack of natural and acquired resistance and differences in behavior and occupation. In this study, only the adult patients were selected as pediatric patients are not usually referred to gastroenterologists. So, this apparent discrepancy is not a real representation.

In this study the disease is found most commonly among housewife and low income group. Similar finding were observed by other also [4,7,8].

Pain in the upper abdomen was the invariable feature in this series which was also found in 80% cases by others [2,8-15]. In this series jaundice was found in 10% of the patients and fever was found in 30% cases. Similar prevalence of these findings was also found by others [1,7,12,13,14,15]. Others symptom were found

in variable percentages in different series. On laboratory examination ova of the worm was found in 6.66% patients and total WBC was elevated in 20% patients. Round worm was detected in UGIT endoscopy in 16.6% patients. These findings could not be compared with other studies. Other studies available did not report these findings.

Summary :

Ascariasis is common problem in our country.. Biliary ascariasis is also not uncommon in our country although there is no representative study in our population. A total of thirty (30) cases of ultrasonographically diagnosed biliary ascariasis were studied to see their clinical and some laboratory profiles. Out of these thirty (30) patients 19 (63%) were female and 11 (37%) were male with age ranging from 19 to 70 years, highest being between 20 – 40 years. Housewife (60%) and small businessman (23.3%) of lower middle class group (66.6%) was the most frequent group. None was from high income group. Upper abdominal severe pain was the invariable (100%) feature of presentation followed by vomiting (46.6%), fever (30%) and jaundice (10%). Majority of the patients (63.3%) revealed no physical findings. Fever, jaundice, upper abdominal tenderness were found in 30%, 13.3%, 13.3% respectively. Endoscopy of UGIT revealed round worm in 16.6% cases.

Conclusion:

With availability of better quality machines, development of excellent expertise among the specialists of concerned fields with increasing awareness among clinicians and radiologists about biliary ascariasis the more and more undiagnosed abdominal pain is now a day's attributable to biliary ascariasis. Now a days ultrasonography is a non-invasive, cheap, radiation hazard free, widely available means of diagnosing Biliary Ascariasis with accuracy comparable to other diagnostic modalities.

This study has got its limitation as only the cases which are diagnosed Ultrasonography to have biliary ascaria were selected. So, the cases which were missed in ultrasonography were not included in this study. So, further study including all cases of abdominal pain suspected to have biliary ascariasis but negative in ultrasonography should be conducted to have the accurate clinical picture of this illness.

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HOW ACCURATELY PHYSICIANS MEASURE BLOOD PRESSURE- AN OBSERVATIONAL STUDY IN ENAM MEDICAL COLLEGE AND HOSPITAL, SAVAR

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Abstract

Objective: *The key to blood Pressure (BP) control is good BP measurement. If BP measurements are not done accurately and reliably, there is a potential for great harm and great cost. Measuring blood pressure is a routine procedure but errors are frequently committed during recording. The aim of the study was to look at the prevalent practices in the institute regarding BP recording.*

Methods and Materials: *This study was conducted in the department of Medicine, Surgery and Gynaecology and Obstetrics in Enam Medical College, Savar. This is an prospective observational study performed amongst 50 doctors in EMCH. Doctors in each three departments were observed by one observer in each department during the act of BP recording. The observer was well versed with the guidelines issued by British Hypertension Society (BHS) and the deviations from the standard set of guidelines were noted. The errors were defined as deviations from these guidelines. The results were recorded as percentage of doctors committing these errors and analysis of results was done manually with percentage and number.*

Results: *In our study, 100% doctors used aneroid type sphygmomanometer. Ninety percent of apparatus were without error. Ninety six percent of the BP cuff was of standard size. Twenty two percent of the doctors did not let the patient rest before recording BP. None of them recorded BP in both arms. In outpatient setting, 70% recorded blood pressure in sitting position and 30% in supine position. In 44% patients where BP was recorded in sitting position BP apparatus was below the level of heart and 60% did not have their arm supported. Eighty four percent did not use palpatory method before checking the BP by auscultation. Sixty percent lowered the BP at a rate of more than 2 mm/s. Seventy six percent recorded BP only once and 75 % of the rest reinflated the cuff without completely deflating and allowing rest before a second reading was obtained.*

Conclusion: *Although the assessment of BP is the most cost-effective procedure in medicine, it is rarely performed according to guidelines. Efforts should be taken to improve the practice of BP measurement which would have a major impact on the health of the population*

Keywords*: Aneroid, palpatory method, auscultatory method.*

Introduction

The blood pressure (BP) measurement is one of the commonly performed procedures by the doctors. Raised blood pressure (hypertension) is a common condition that does not have specific clinical manifestations until target organ damage develops[1] Routine screening of all the patients, especially high risk patients, is the only way of detecting hypertension early and initiate treatment before target organ damage becomes evident.[2] Accurate measurement of BP is importance for labeling a patient as hypertensive. Consistently underestimating the BP by 5 mm Hg could result in two-thirds of hypertensive patients being missed and over estimating it by 5 mm Hg could more than double the number of patients being diagnosed as hypertensive.[3] Missing the diagnosis in a hypertensive patient could result in significant morbidity and mortality due to lack of treatment. Over diagnosis results in inappropriate labeling and treatment of healthy individuals. Most of us are aware of the exact methodology of recording of BP, yet most of us commit errors frequently resulting in erroneous high or low recording.

The measurement of BP in clinical practice is done by a century old Riva-Rocci/Korotkoff technique. The accurate measurement is dependent on the accurate transmission and interpretation of a signal (Korotkoff

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sound or pulse wave) from a subject via a device (the sphygmomanometer) to an observer[4] Errors in measurement can occur at each point but the commonest fallible component is the observer.[4]

Despite the clear guidelines on BP measurement technique, there seems to be large inter-observer variations, both among nursing staff and physicians as well as between the two groups. In an article by Graves and Ships in the American Journal of Hypertension , the authors are of opinion that 'physicians do not measure BP well, and even if they do, the usefulness of their BP measurements is significantly compromised by the white coat effect.'[5] The general belief amongst the researchers is that physicians dealing with diagnosis and treatment of hypertension do not follow the international society guidelines.[6] In a study by Perloff et al.[7], it was found that nursing staff abided by 40% of the recommended procedures while medicine teachers, physicians and residents abided by approximately 70%.[8]The wide gaps in the basic theoretic and practical knowledge seem to be common among interns and first-year family practice residents resulting in erroneous measurements.[9] In an interesting observational study, carried out at the Westminster Medical School in London, showed that 33% out of 80 doctors in training grades/junior hospital doctors, acknowledged no formal education on how to measure BP, a finding confirmed further by the poor accuracy in BP measurement displayed by one-third of the study group.[10] There has not been many study done in Bangladesh regarding objective performance of blood pressure recording according to guideline. This study was done to observe the practice of blood pressure recording of physicians in tertiary care hospital and to identify the pitfall.

Objectives: Our objective was to notice the common errors committed during routine blood pressure recording by the residents and consultants

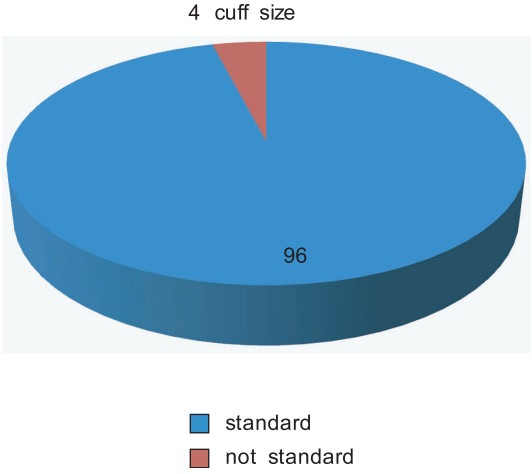
Methods and Materials

A prospective, observational study was performed amongst 50 doctors (10 consultants and 40 residents). The consultants belonged to the department of medicine, surgery and gynaecology and the residents included interns, house physicians and indoor medical officer. The study was conducted over a period of 2 months. A single observer from each department were trained uniformly regarding blood pressure measurement according to British Hypertension Society (BHS) and tested clinically by principal investigator for accuracy and consistency. A single observer in each department observed the enrolled subjects during the act of BP recording without any one of them being aware of the fact that they were being observed. The common errors committed were noted in a performance after having observed them but the recording physicians were not informed regarding the study procedure of observer. Some participants were observed again to note the practices that had been missed during the first observation. The observer recorded the finding retrospectively in a structured case report form.

The errors were defined as variations from the standard set of instructions issued by British Hypertension Society(BHS)[11] This variation from the standard guidelines were further analyzed and recorded as percentage of doctors committing these individual errors. At the end of the study the erring doctors were apprised of the results of the study and were told about the standard guidelines.

Results

Fifty study subjects were observed in different departments of Enam Medical College Hospital (EMCH) for their blood pressure recording techniques. In this study, 100% doctors used aneroid type sphygmomanometer. 90% of apparatus were without error. 96% of the BP cuff was of standard size. 22% of the doctors did not let the patient rest before recording BP. None of them recorded BP in both arms.



In outpatient setting, 70% recorded blood pressure in sitting position and 30% in supine position. In 44% patients where BP was recorded in sitting position BP apparatus was below the level of heart and 60% did not have their arm supported. 84% did not use palpatory method for noticing systolic BP and 58% did not raise pressure 20-30 mm Hg above the systolic level before checking the BP by auscultation.60% lowered the BP at a rate of more than 2 mm/sec . 76% recorded BP only once and 75%

of the rest reinflated the cuff without completely deflating and allowing rest before a second reading was obtained.

Discussion

The blood pressure in all the individuals varies considerably throughout the day. A variety of activities affects the BP and causes it to increase. Simple activities of daily routine like eating, dressing, commuting to work, talking on telephone and attending a meeting raises systolic BP by an average 10-20 mm Hg and diastolic BP by 8-15 mm Hg.[3] Numerous studies have proven time and again that the various exogenous factors also interfere with the accurate measurement of BP.[12,13,14,15,16,17,18.] The important factors being talking, exposure to cold, ingestion of alcohol and medications especially antihypertensive drugs.[12,13,14] Errors during the process of BP measurement also contribute to the erroneous reading.

There are only three sources of errors while BP is being recorded. These are observer bias, faulty equipment and failure to standardize techniques of measurement[19.]While it may not be possible to do anything for observer bias but following a standardized technique and using a good equipment may help to reduce the error rate to a great extent.

It is well known that mercury instruments provide the most accurate records and are the preferred instrument in hospital settings .[20] Aneroid sphygmomanometers are increasingly used due to ease of handling[21]but are a source of error if not maintained properly[22] .Since the majority recording apparatus in our hospital are aneroid based, so 100% of our recordings were made on them.

The defective apparatus may give a false high or low BP reading. Similarly, the BP in the dominant hand is usually higher [1] .Failure to record these facts may lead to differences in the subsequent BP recordings. Unfortunately this fact is commonly ignored and not recorded, as was evident in our study.

It has been shown in a recent study that both the bell and the diaphragm give equal results when used for office measurement of BP.[23] still the diaphragm is preferred in clinical practice, as was in our study. The reason for this lies in it being easier to secure with the fingers of one hand and also that it covers a larger area.[4]

In a survey of 114 doctors conducted by McKay et al 97% doctors used inappropriate cuff size.[24] It indicates that it is a common mistake made by most of the doctors. It is known that if the cuff is too small as in the case of a fat patient the systolic BP will be recorded falsely low by up to 8 mm Hg and diastolic BP will be recorded high by up to 8 mm Hg.[17]Our findings are not different from these observations. The failure to remove the clothing further adds to the arm circumference, hence erroneous recording. A number of studies have shown that measurement of BP in obese and large muscular arms requires adjustments. Monograms for adjusting BP recording in the obese are inadequate. The most important factor is choosing the correct cuff width-arm circumference (CW/AC) ratio. Such action reduces the intersubject variability of BP measurement in clinical settings.[25]

It is known that a number of activities of daily living raise the BP[3] and a period of rest before measuring BP may return it to normal level. Failure to do so may result in falsely high BP recording. Still 70% of our study group doctors did not wait and let the patient rest for some time before recording BP in the OPD. In a study by McKay et al. this figure was 97%.[24] None of our study group doctors recorded BP in both arms, which is much more than 77% reported in the literature.[22]

It is well established that if the BP is only measured in the supine position the systolic BP may increase by 3 mm Hg and the diastolic BP will be recorded lower by 3-5 mm Hg.[15,26] It would be worthwhile to record BP in both supine and sitting position if possible or at least the position in which the BP is recorded should be mentioned in the records. This would be helpful in follow-up visits by the patient. Unfortunately this fact is taught in the clinics but not followed by majority of us while recording BP, especially in the outpatient department.

If the position of the arm is either above or below the heart level the BP may be recorded false high or low. For every 10 cm above or below the heart level the systolic BP decreases (if above) by 8 mm Hg and increases (if below).[15]Similar changes are seen in the diastolic BP with change in the position of arm in relation to the heart level.[15] If the arm is not supported the systolic and diastolic pressures will be recorded high by 2 mm Hg.[15] Our study showed that this fact is commonly forgotten during BP recording in the outpatient department.

About 60% of our study group doctors did not use palpatory method for noticing systolic BP initially and 70% did not raise pressure 30-40 mm Hg above the systolic level before checking the BP by auscultation. McKay et al. in their study noted similar figures, where the number of such doctors was 61%.[24]

About 60% in our study group deflated the cuff at a rate of more than 2 mm/s which is little variationto 82% in another study.[24] Also 76% in our study group recorded BP only once and 75% of the rest reinflated the cuff without completely deflating and allowing rest before a second reading was obtained. This may further increase the incidence of erroneous recording in clinical practice. In one study by Jamieson et al. it was observed that the first systolic BP was on an average 3-4 mm Hg higher while the diastolic BP was not different when recorded twice.[26] Complete deflation of the cuff and allowing a few minutes rest between two consecutive measurements may circumvent this problem, however, this is not routinely done. The authors of this study have suggested an alternative, that taking two measurements and recording the average would help in reducing the errors, when the BP exceeds 155/90 mm Hg.[26]

There is tendency by physicians to expect either a high or low BP. This results in rounding off the systolic and diastolic BP to the nearest 5-10 mm Hg, which may result in erroneous high or low BP recording.[18] A British study carried out in 18 practices and 67 GP offices showed digit bias in systolic and diastolic readings to the nearest 10 mm Hg.[27] Also 60% of the doctors in our study group had a digit preference to the nearest 5-10 mm Hg. Both under estimation or over estimation of BP, due to this bias, could have enormous reflection on the sheer numbers of the patients either missed or over diagnosed.[3]

The most recent recommendations of AHA suggest that the auscultatory technique with a trained observer and mercury sphygmomanometer to be the method of choice for measurement of BP. Proper training of observers, positioning of the patient, and selection of appropriate cuff size are all essential.[20] However, training can reduce, but not abolish, these inaccuracies. Taking multiple BP measurements before making clinical decisions can limit the effect of these inaccuracies.[28]

Reevs points out that the efficient practitioner can reserve the proper method for 10-20% of patients who have known or newly detected elevated BP, cardiovascular damage, other risk factors or are receiving antihypertensive therapy.[29] This would go a long way in preventing the errors in patients where it matters the most.

Conclusion

Accurate measurement of the BP is very important in the clinical setting. It is a vital parameter to access and modify cardiovascular risk factors. Very commonly errors are committed during these simple procedures and efforts should be made to minimize them by following the international guidelines. This study looks at the practices prevalent in a teaching hospital and proves that accurate measurement of BP is not difficult provided we know the exact methodology and follow it too. The tendency to create shortcuts is likely to result in erroneous high or low recording. We can correct our mistakes only if we are made aware of them.

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A STUDY OF HYPOALBUMINAEMIA IN CHRONIC LIVER DISEASE AND ITS CORRELATION WITH DEVELOPMENT OF ESOPHAGEAL VARICES

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Abstract

Background: *The aim and objective of this study was to evaluate relationship of serum albumin and esophageal varices in chronic liver disease (CLD) admitted in the medicine unit and gastroenterology department of Dhaka Medical College Hospital.*

Method: *In this cross-sectional study, a total number of 100 randomly selected, clinically diagnosed patients of chronic liver disease were studied from June 2010 to November 2010 (6 months). All patients were assessed as per Child-Pugh class and had full blood count, HBsAg, Anti-HCV antibodies by ELISA, abdominal ultrasound and Endoscopy of upper gastrointestinal tract. Patients were divided into Group A (serum albumin <3.5 gm%) and Group B (3.55).*

Result: *Seventy-three male (73%) and twenty seven female patients (27%)] with age range of 16 to 75 years were evaluated. Out of 100 patients 24% were in between 46-55 years age group. 63% patient fall in child Pugh class A group, 32% fall in child Pugh class B & 5% fall in child Pugh class C. Mean Serum albumin was 3.8 gm%, (range 2.4-4.9). Esophageal varices (EV) were present in 32 patients (32%) and absent in 68 patients (68%). Group A had 29 patients (29% of the total) with 18 patients (62.06%) having EV. Group B had 71 patients (71% of the total) with 14 patients (19.71%) having EV. Sensitivity of hypoalbuminaemia as a marker of EV was 56% and specificity 83.8%, positive predictive value 62.06% and negative predictive value 80.2% and Odds ratio was 6.6. P value is <0.001.*

Conclusion: *In Group A that is hypoalbuminaemia (<3.5 gm%), the incidence of Esophageal varices was more than Group B that is albumin level (>3.5gm%). Hypoalbuminemia is a good surrogate marker for the presence of esophageal varices in CLD.*

Key Words: *Chronic liver disease, hypoalbuminaemia, esophageal varices*

Introduction:

Chronic liver disease (CLD) is a common hepatobiliary problem encountered in day to day clinical practice in Bangladesh. CLD can occur at any age & often causes prolonged morbidity & is an important cause of premature death. The patient who we come across in the hospital ward is mostly in advanced stage with overt clinical manifestation and/or complication. Among them most important life threatening condition is vomiting out of blood (Haematemesis) & malaena due to ruptured esophageal varices. Although 90% of patients with cirrhosis develop varices, only 30% of them bleed and 30 – 50% die of the first episode.[1] Two thirds of the survivors will rebleed within six months if not treated with prophylactic â-blockers or endoscopic therapy.[2]

Albumin (50%-60% of total plasma protein), globulin, and fibrinogen make up the major share of plasma proteins with 24%-56% increased risk of death per 2.5 g% fall in serum albumin.[3,4] Child-Pugh score (serum albumin being integral part of the score) predicts advanced liver disease.[5] Liver produces albumin at a rate of 130–200 mg/kg/day.[3] Hypoalbuminemia in cirrhosis is multifactorial and may be due to reduced production (liver parenchyma replaced by fibrous tissue), increased removal by reticuloendothelial system (spleen) or increased loss through gut [6] (portal gastropathy/enteropathy): all

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related to portal hypertension. Hypoalbuminemia with associated ultrasonographic features e.g. gall-bladder wall thickness [5] and right liver lobe diameter[7] have been cited as non-endoscopic predictors of esophageal varices. Similarly, Serum Ascitic Albumin Gradient (the difference between the serum and ascetic albumin concentration) i.e. SAAG, is thought to be an indirect marker of portal hypertension,[8] with a high gradient (>1.1 g/dL) indicating portal hypertension and presence of EV.

Portal hypertension (portal pressure >12 mm Hg or >5 mm Hg gradient between the wedged hepatic venous pressure and the free hepatic venous pressure has been shown in animal studies to induce hypoalbuminemia.[9] Conversely, serum albumin is increased by 20% when portal pressure is reduced after Trans jugular Intra-hepatic Porto-systemic Shunt Procedure suggesting a link between portal hypertension and hypoalbuminaemia.

Materials and Methods:

In this cross-sectional study, a total number of 100 randomly selected, clinically diagnosed patients of chronic liver disease were studied for a period of June 2010 to November 2010 (6 months) at medicine units and gastroenterology department of Dhaka Medical College Hospital. Clinically CLD diagnosed interlaying one or more stisgment or child. All patients were assessed as per Child-Pugh class and had full blood count, HBsAg, Anti-HCV antibodies by ELISA, abdominal ultrasound and Endoscopy of upper gastrointestinal tract. Exclusion criteria were patients receiving sclerotherapy, band ligation of EV and prophylactic treatment for portal hypertension, hypoalbuminemia which in clinically correlated with congenital cardiac failure nephritic syndrome or malnutrition patient refusing to give consent to take part in our study. This study was carried out to correlate between serum albumin and EV in CLD. Patients were divided into Group A (serum albumin <3.5 gm and Group B (albumin >3.5 g/day). Statistical analysis was carried out by using SPSS v16.0 Windows statistical software. Descriptive statistics were used for the interpretation of the findings. Informed and written consent obtained from all patients or their guardian. Formal Ethical Clearance was obtained from the Research Review Committee of Dhaka Medical College and Hospital.

Result:

Seventy-three male (73%) and twenty seven female patients (27%) with age range of 16 to 75 years were evaluated. Out of 100 patients 24% were in between 46-55 years age group. 75% of CLD were due to Hepatitis B, 7% due to Hepatitis C, 18% due to others. 32% patients have EV and 68% patients do not have EV.

Figure-1 shows that majority of CLD patients 63% fall in child Pugh class A group, followed by 32% fall in child Pugh class B & 5% fall in child Pugh class C.

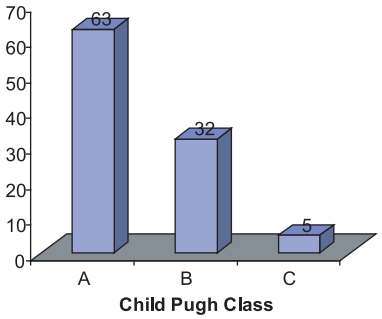

Fig.-1: *Distribution of CLD patients in Child Pugh class*

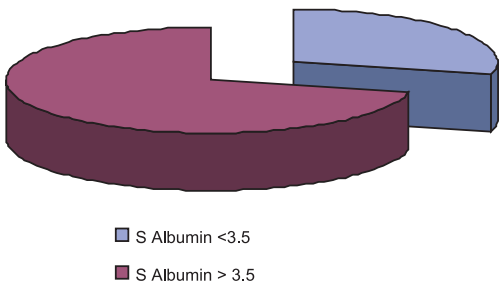

Fig.-2: *Prevalence of hypoalbuminaemia in CLD patients*

Table-I

Gradings of EV in different Child Pugh class

Child Pugh class	NO EV (%)	Grade I (%)	Grade II (%)	Grade III (%)	Grade IV (%)	Total (%)
A	47(74.60)	8 (12.69)	5 (7.93)	1 (1.58)	2 (3.17)	63 (100)
B	20 (62.5)	2 (6.25)	1 (3.13)	2 (6.25)	7 (21.87)	32 (100)
C	1 (20)	0 (0)	2 (40)	1 (20)	1(20)	5 (100)
Total (%)	68 (68)	10 (10)	8 (8)	4 (4)	10 (10)	100

Table-I shows among 100pts of CLD pts 63 pts (63%) were in Child Pugh class A from which 74.60% have no varices. 32 pts (32%) were in Child Pugh class B from which 62.5% have no varices. 5pts (5%) were in Child Pugh class C from which 40% have Grade II varices.

Fig.-2 shows maximum 71% CLD patients have serum albumin >3.5 gm/dl and 29% patients have serum albumin <3.5 gm/dl.

Table-II

Frequency of Esophageal varices in group A & group B in Chronic Liver Disease pts

Albumin Level (gm/dl)	EV Present (%)	EV Absent (%)	Total (%)	*P* value
Group A (<3.5gm/dl)	18 (62.06)	11(37.93)	29 (29)	<0.001
Group B (>3.5gm/dl)	14 (19.71)	57 (80.28)	71 (71)	
Total	32	68	100	

EV : Esophageal varices, CLD: Chronic liver disease

Table-II shows that in group A 62.06% have EV while in group B only 19.71% have EV, So EV is more in group A in CLD patients. This Table shows P value is <0.001 that is association between hypoalbuminaemia and Esophageal varices is significant. Sensivity: 56%, Specificity: 83.8%, positive predictive value (PPV): 62.06%, negative predictive value (NPV): 80.2%, odds ratio: 6.6.

Discussion:

In this study all the patients were grouped in six age groups. Majority of the study subjects were in between 46-55 years age group 24% followed by 23% between 36-45 years age group. Ahsan T et al [10] found 28% in between 46-55 years age group which coincide with our study. 73% were male and 27% female. Male female ratio was 2.7:1. Mahtab et al [11] found male-female ratio of 2.97:1, which almost coincide with our study. In this study 63% patient fall in child Pugh class A group, 32% fall in child Pugh class B & 5% fall in child Pugh class C .This study almost coincide with the study of khan H et al [4] where 70.1% patient fall in child Pugh class A group, 24.9% patient fall in child Pugh class B group, 5.1% patient fall in child Pugh class C group. Present study shows that 32% of CLD patients have EV & 68% patients did not have EV. Fook-Hong et al [12] found 53pt (57.60%) have EV, 39 pt (42.39%) did not have, Schepis et al found [13] 80 pt (55.9%) have EV, 63 pt (44.05%) did not have. This difference may be due to sample size and etiological difference.

In child pugh class A pts, 16pts out of 63 pts(25.40%) have varices among them 8 pts(12.69%) have grade I, 47 pts (74.60%) have no varices. In child pugh class B pts, 12pts out of 32 pts (37.50%) have varices among them 7 pts (21.88%) have grade IV, 20 pts (62.5%) have no varices. In child pugh class C pts, 4 pts out of 5 (80%) have varices among them 2 pts (4%) have grade II, 1 pt (20%) have no varices.

Present study shows that 29 pt (29%) out of 100 have hypoalbuminaemia (<3.5gm/dl that is group A) while 71 pts (71%) had albumin level > 3.5gm/dl that is group B. In group A (Albumin level <3.5 gm/dl), 18 pts (62.06%) have EV, and 11 patient (37.93%) did not have EV. In group B (Albumin level >3.5 gm/dl), 14 pts (19.71%) have EV, and 57 patient (80.28%) did not have EV. Mean serum albumin level was 3.8 gm/dl. Khan H et al.[4] found 57 pts (28.9%) out of 197 have hypoalbuminaemia (<3.5gm/dl). Among them 35 pts (61.4%) have EV, while 140 pts (71.1%) had albumin level >3.5gm/dl) with 28 having EV (20%). This consistent with the present study. From this study, hypoalbuminaemia (<3.5gm/dl) is 56% sensitive & 83.8% specific for presence of EV with positive predictive value of 62.06% & negative predictive value of 80.2% and odds ratio is 6.6 and *P* value is <0.001(Table 2) that is association between hypoalbuminaemia and Esophageal varices is significant. This study almost coincide with the study of khan H et al [4] which shows that hypo-albuminaemia (<3.5gm/dl) is 53.2% sensitive &91% specific predictor of EV with positive predictive value of 73.3% and negative predictive value of 80.8% and odds ratio of 11.57. Zein et al [14] in a study of 183 patients with primary sclerosing cholangitis, found 66% sensitivity, 80% specificity, 53.4% PPV, 87.2% NPV and odd ratio of 7.8 for albuminaemia of <3.5 gm%; almost similar to our results. The minor differences in figures could be explained on the basis of difference in etiology of the study population and sample size. khan H et al [4] used study population with uniform etiology of infective hepatitis & Zein et al[14] in a study of 183 patients with primary patients with CLD of diverse etiologies. The minor differences in sclerosing cholangitis, Bressler et al[15] found albuminemia of <4gm% as an independent risk factor for EV with odd ratio of 6.02 We used albuminemia of <3.5 gm% while Schepis et al [13] and Sarwar et al [16] used level of <2.95 gm% to predict the presence of EV. Odds ratio was 6.6 and the difference could be explained by <3.5 gm% albumin level we used and our study population with CLD with diverse etiology. Specificity of 83.8% and PPV of 62.06% suggests that hypoalbuminemia is a good indicator of EV. However,

low sensitivity of hypoalbuminemia (56%) and NPV of 80.2% indicates that absence of hypoalbuminema does not rule out EV.

Conclusion:

Patients with chronic liver disease frequently undergo endoscopy of upper GIT to detect EV. Doing endoscopy in all patients of CLD will increase socio-economic and medical load because of the rising numbers of such patients. Therefore, there is a particular need for a noninvasive predictor for the presence of EV to ease the medical, social and economic burden of the disease. Many previous studies have documented good predictive value of various non-endoscopic variables for the presence or absence of varices, but available data in our country is limited. We consider simple, commonly available parameter serum albumin. From this study it is assumed that hypoalbuminaemia is a good non-endoscopic marker for the presence of esophageal varices.

Conflict of interest: We have no conflict of interest.

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REVIEW ARTICLE

A RARE AND CLINICALLY IMPORTANT BLOOD GROUP- BOMBAY BLOOD GROUP

CHOWDHURY F.S[1], SIDDIQUI M.A.E[2], RAHMAN KGM[3] ,NASREEN Z[4] , BEGUM H.A[5], BEGUM H A [6]

Introduction:

Bombay blood group is the rarest blood group in the world. It is a blood group which shows absence of A,B,H antigens on red cells and presence of anti- A, anti-B and potent wide thermal range anti-H antibodies in serum reacting with all O blood group[1].

Dr. Y.M. Bhende first discovered Bombay blood group in 1952 at Bombay in India now known as Mumbai. This is the reason why this blood group got the name Bombay blood group[2].

Prevalence

At present about 0.0004% of the general human population have Bombay blood group, though in some places such as Mumbai local populations can have occurrences as much as .01% of inhabitants[3]. People with this blood group are found in Maharashtra and some places of Karnataka which lies at the border of Maharashtra. In a recent study an incidence of 1 in 33 among Kutia Kondh tribe, 1 in 127 in Kondh tribe and 1 in 1244 among the tribal populations of Orissa is found. This is the highest incidence of Bombay blood group so far reported from India.[4.] The incidence of this phenotype as 1 in 13,000 individuals in Mumbai[5]. An incidence of 1 in 7600 after screening a large number of samples in Mumbai.[6] In Maharashtra, reported the incidence of the Bombay phenotype as 1 in 4500[7]. Incidence is 1 in 18,404 amongst Indians settled in South Africa[8]. Of the 179 cases 112 (62.6%) cases belonged to the state of Maharashtra. A slightly higher frequency of the Bombay phenotype was also found in the neighboring state of Karnataka (14 cases), Andhra Pradesh (8 cases), Goa (6 cases), Gujarat (5 cases), Uttar Pradesh (5 cases), and so on in the decreasing order[8]. The incidence of the Bombay phenotype is high in those states of India where consanguineous marriages are more prevalent, i.e., Andhra Pradesh, Tamil Nadu, Karnataka, Maharashtra, Gujarat, etc. than in the other states. Three cases of a rare blood group, Bombay (Oh) phenotype, in the Bhuyan tribe of Sundargarh district in North-Western Orissa were detected, Individuals with the Bombay blood group were also detected in Japan (Okubo 1980;Kaneko et al. 1997),Malaysia (Lopez, 1972),Thailand (Sringarm et al.1977) and Sri Lanka (De Zoysa 1985)[9]. H-deficient Bombay phenotype is rare, since it occurs in about 1 in 10,000 individuals in India and 1 per 1,000,000 individuals in Europe [10]. More recently, a large series (42 H-deficients) of H-deficient individuals (~1:1000) were found in a small French island 800 km east of Madagascar in the Indian Ocean, called Reunion Island[11]. This indicates that the Bombay phenotype is mostly confined to South-East Asian countries.

People with bombay blood group in Bangladesh:

No specific statistics of people with Bombay blood group in Bangladesh is available. The first person with Bombay blood group was identified in Bangladesh in Narayangonj. Till now four people of Bombay blood group found in Bangladesh. Among them three are sisters of same family[12].So the transfusion centers should have the means and the thought to test for Bombay group.

Genetics

99.9% of all individuals have an HH or Hh genotype. Individuals with the Bombay group have inherited two recessive alleles of the H gene (their genotype is "hh") and so do not express H antigen[13]. A antigen and B antigen are made from H antigen. As a result, people with Bombay blood group cannot make A antigen or B antigen on their red blood cells. Because both parents must carry this recessive allele to transmit this blood type to their children, the condition mainly

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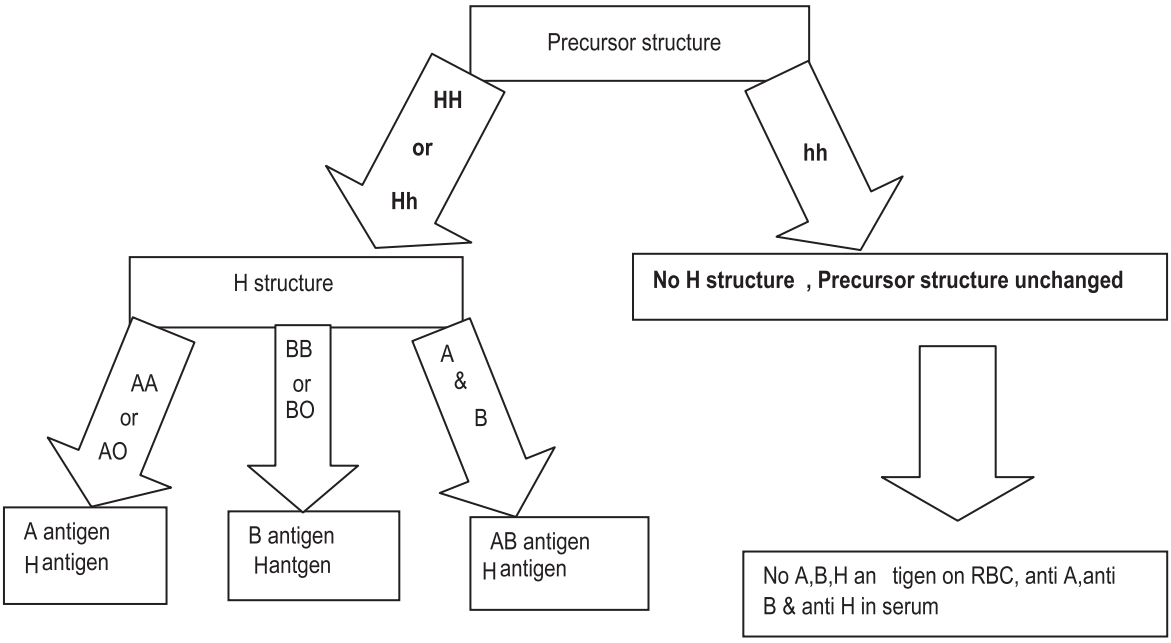

Fig.-1: *Interaction of the Hh and ABO gene.*

occurs in small closed-off communities where there is a good chance of both parents of a child either being of Bombay type, or being heterozygous for the "h" gene allele and so carrying the Bombay characteristic as recessive. Although H structure is responsible for formation of A and B antigen, there is separate gene for formation of A and B enzymes. So It is then no longer matters whether the A or B enzymes are present or not, no A or B antigen can be produced since the precursor antigen H is not present. When Bombay blood group was first encountered, it was found not to contain antigens A or B and so was thought to be of group O. But experience showed that Bombay group patients could not even safely receive normal O-group blood, and this proved to be because they lacked the H antigen and have potent wide thermal range anti H.

Detection

It is very difficult to detect Bombay group people when usual blood group test is conducted. The usual tests for ABO blood group system would show them as group O. There is a serum grouping called reverse grouping for accurate test of a person's ABO group. If this test is conducted then we can detect the presence of H antibody, which indicate Bombay blood group. This test is conducted with the help of a reagent called H – Lectin which has anti-H like activity. Thus it is used to determine the presence or absence of the H antigen on the surface of RBCs. The Bombay phenotype detected was further confirmed by certain specialized tests like absorption-elution studies, titration of naturally occurring antibodies at different temperatures, hemagglutination-inhibition study on anti-H by O saliva secretor, and secretor-status of the person[14,15].

Cell grouping		Serum grouping				Interpretation
Anti A	Anti B	Anti AB	A cells	B cells	O cells	
+	-	+	-	+	-	A
-	+	+	+	-	-	B
+	+	+	-	-	-	AB
-	-	-	+	+	-	O
-	-	-	+	+	+	Bombay blood group

Diagram as shown in above diagram, cell grouping is carried out using anti A, Anti B andante AB commercially available sera. Serum grouping is carried out using A cells, B cells and O cells

General Characteristics of Bombay Blood Group: [13]

1. Absence of H, A, and B antigens; NO agglutination with anti-A, anti-B, or anti-H lectin.
2. Presence of anti-A, anti-B, anti-AB and potent wide thermal range anti-H in the serum.
3. A, B, H non-secretor (no A, B, or H substances present in saliva)
4. Absence of H enzyme in serum and H antigen on red cells.

5. Presence of A or B enzymes in serum and red cells.
6. A recessive mode of inheritance.
7. Red cells of the Bombay group are compatible only with the serum from another Bombay individual.

Transfusion Compatibility

Individuals with Bombay blood group can donate to all ABO blood group people and can only accept from Bombay blood group people. The Bombay anti-H is an IgM antibody that can bind complement and cause red cell lysis.Because the H antigen is common to all ABO blood group, Bombay blood is incompatible with all ABO donors[16].

Given that this condition is very rare, any person with this blood group who needs an urgent blood transfusion will probably be unable to get it, as no blood bank would have any stock.Those anticipating the need for blood transfusion (e.g. in scheduled surgery) may bank blood for their own use (i.e. an autologous blood donation), but this option is not available in case of accidental injury[17].

End Note

Bombay blood group is a rare blood group mainly found in the South East Asia including Bangladesh. People having this blood group are very small in number. It doesn't mean that these people are having any disease or it's the symptom of any disease. People with Bombay blood group should report to the nearest blood bank. They are advised not to give blood in donation programs because it is not necessary that these Bombay blood will be used within 45 days. Relatives can have Bombay blood group, so they should be screened for Bombay blood group. People having Bombay blood group should be listed.Since Bombay Blood Group is the rarest of the rare group, it is desirable to develop cryopreservation facilities for rare donor units. Every blood bank can easily maintain a rare blood type donor file from their regular voluntary donors.It is only possible to solve problems related to rare blood groups like Bombay blood group if each blood bank has a large number of committed regular voluntary donors.

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CASE REPORTS

A YOUNG GIRL WITH REPEATED EPISODES OF PNEUMONIA AND INTERMITTENT DIARRHOEA- THE PRIMARY IMMUNODEFICIENCY SYNDROME

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Abstract:

A description is given of a case of a young girl who presented with repeated episodes of pneumonia since childhood. In addition, she had episodes of intermittent diarrhoea. Her investigative work-up revealed a deficiency of serum immunoglobulins. The profile was suggestive of common variable immunodeficiency. Because of a relative rarity of this disease, it is often misdiagnosed earlier. Late diagnosis and delayed institution of immunoglobulin replacement therapy results in increased morbidity with a wide variety of organ-specific complications and increased mortality.

Introduction:

The primary immunodeficiency syndromes are a rare group of disorders that can present at an age for which delay in diagnosis remains common. Persons with hypogamma globulinaemia are more likely to get infections and patient suffer from recurrent infections of various body systems, more so of the respiratory tract. Replacement therapy with immunoglobulins in primary immunodefiency increases life expectancy and reduces infection frequency and severity[1]. We present a case of a young female who developed repeated chest infections, recurrent diarrhoea and arthritis since early childhood.

Case report:

A 23 years old female presented with pain and swelling of multiple joints for last 3 months. Pain first appeared at right side of the pelvis. About one month later both wrists, all metacarpophalangeal, shoulders, elbows and knees were successively involved. Pain was constant, moderate to severe in nature and sometimes became so severe that patient could not move the joints and she had to take analgesics regularly. Pain was associated with swelling of the joints but not associated with morning stiffness and redness. Pain spared back, chest, neck and feet. For this pain she consulted a rheumatologist who diagnosed her as a case of ankylosing spondylitis and prescribed Salphasalazine. But the patient could not tolerate the drug. She had also history of getting intra-articular steroid injection. The patient also mentioned that for last 20 days she developed fever and burning sensation during micturition and loose motion. Her father also stated that since childhood she was growth retarded and suffered recurrent fever, respiratory tract infection, loose motion, burning sensation during micturition and oral sore and for this she got medication for multiple times. On query, patient had a history of taking anti-tubercular drugs for sputum negative pulmonary tuberculosis. She also underwent lobectomy of left lung for bronchiectasis 4 months back. (Fig 1)

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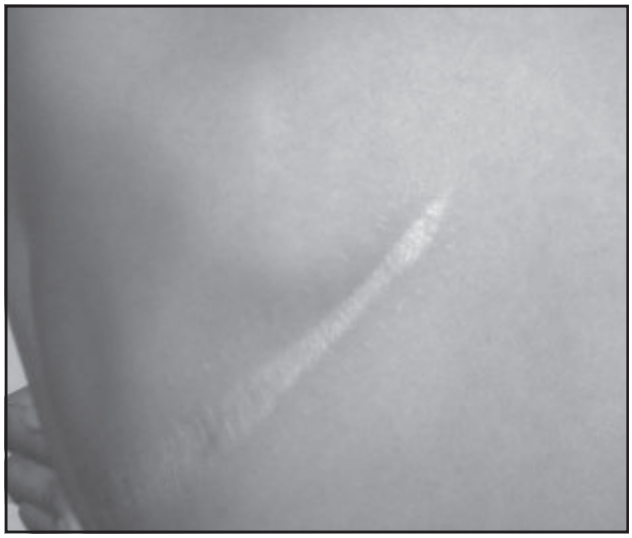

Fig.-1: *The scar of left sided lobectomy due to bronchiectasis*

Patient is asthmatic and it was diagnosed as cough variant asthma with positive bronchoprovocation test 4 months back and for this she was on medication for last 3 months. On examination, patient was emaciated; pulse-80/min ; blood pressure-70/50 mm of Hg; temp-99°F ; Anaemia –moderate; clubbing present; no palpable lymphadenopathy & thyroid gland was not enlarged. Nutritional status: BMI-9.4kg/m2; mid upper arm circumference-15 cm. Examination of lower extrimities & pelvis- bilateral sacroiliac joint tenderness; tenderness over both knee joints but no restrictive movement ; tenderness over lower lumbar spine but no gibbus; Schober's test was positive; SLR negative. Examination of upper extrimities-wasting of the small muscles of the hand & dorsal guttering, ulnar deviation of hands; tenderness over both wrists, metacarpophalangeal joints, elbow & shoulder joints and there was restricted movement of the wrist & shoulders, no tenderness over proximal & distal interphalangeal joints.(Fig.2)

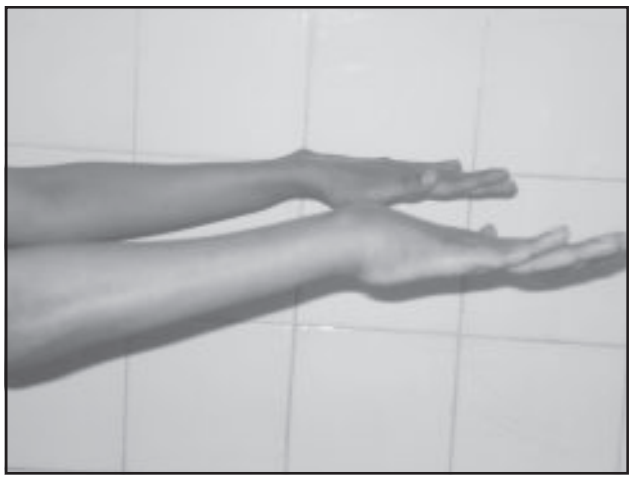

Fig 2: *Wasting of hand muscles and ulnr deviation*

Investigations revealed- CBC:Hb-7.7 gm%, ESR-45 mm in 1st hour, WBC count-10.68 thousand/mm3 , Neutrophil-76%, Lymphocyte-18% ,Hematocrit-26.60%, MCV-78.50 fl. CRP-1.72 mg/L(0-3 mg/L). RA test –negative,Anti CCP antibody < 1 unit/ml. ANA-negative. HLA-B27-positive.Serum ferritin-827 ng/ml(15-200ng/ml). Blood C/S- no growth of organisms. Urine R/M/E-pus cell: 10-12/hpf, epithelial cells:2-4/hpf, RBC- nil, Albumin-absent. Urine C/S-no growth of organisms. Urine AFB-not found. Stool R/M/E: mucous ++, vegetable cells ++.Stool culture – no growth of organism. X ray Pelvis A/P view: no significant skeletal abnormality. X ray of lumbosacral spine B/V: no significant skeletal abnormality. X ray chest P/A view: old fracture or expansile bony lesion at the posterior aspect of left 6th rib. X ray hand B/V: Left- suggestive of inflammatory joint disease; Right –mild subluxation of fifth metacarpophalangeal joint.(Fig.-3).

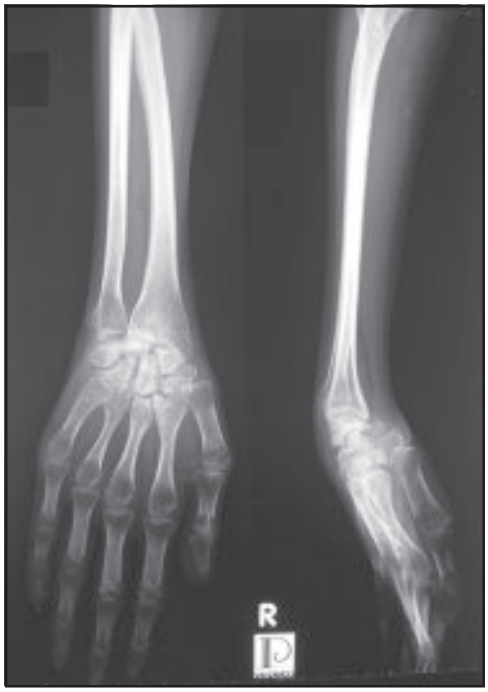

Fig.-3: *X ray hands and forearm revealed subluxation of fifth metacarpophalangeal*

USG of whole abdomen- Echogenic liver parenchyma. Liver Function Test-normal. FBG-4.6 mmol/L. S. creatinine- 1.08 mg/dl. S electrolytes: sodium-135mmol/L, potassium-4.7 mmol/L,chloride-98 mmol/L, CO2-24 mmol/L. Serum immunoglobulin study- Ig G-2.56 gm/L(7-16), IgM-0.17 gm/L(0.4-2.3), Ig A-0.23 gm/L(0.70-4.00**)** CT scan of chest- triangular density areas containing air densities are seen along medial aspects of left lower lobe, impression of small

chronic pneumonia with bronchiectatic changes suggestive of chronic TB. Pneumonia CT guided FNAC of lower left lung- smear shows scanty cellular material containing a few polymorphs, lymphocytes & pulmonary macrophages in the background of blood. No malignant or granuloma is seen; suggestive of inflammatory lesion. Histopathology : section of the lung shows multiple epitheloid granuloma with caseation necrosis with bronchiectatic changes; no focal alveolar haemorrage-suggestive of bronchiectatic changes with PTB. Sputum for AFB (three consecutive morning samples)-negative. Echocardiography-normal. After evaluation clinically and investigations she was diagnosed as a case of Primary Immuno-deficiency Syndrome

Discussion:

Immunoglobulin deficiency disorders are an important though uncommon cause of recurrent infections, which are not often suspected by treating physicians. The classical feature of such disorders is an increased susceptibility to infections and immunodeficiency should be suspected in any patient who presents with recurrent or persistent infections[2]. Defects in antibody synthesis may be primary or secondary and may be quantitative or qualitative. The major cause of primary antibody deficiency includes common variable immunodeficiency, selective IgA deficiency, infantile sex linked hypogammaglobulinaemia (Brutons disease), transient hypogammaglo nulinaemia of infancy, IgG subclass deficiency etc. Secondary causes of antibody deficiency could be either due to decreased production(malnutrition, lymphoproliferative disease, drugs) or increased losses(nephrotic syndrome, protein losing enteropathy, burns) [3]

Patient with defects in humoral immunity have recurrent or chronic sinopulmonary infection, meningitis, and bacteremia, most commonly caused by pyogenic bacteria such as Haemophilus influenzae, Streptococcus pneumoniae, and Staphylococcus aureus. Abnormalities of T cell-mediated immunity predispose to disseminated virus infections, particularly with latent viruses such as Herpes simplex. Varicella zoster and Cytomegalovirus. In addition, patient so affected almost invariably develop mucocutaneous candidiasis and frequently acquire systemic fungal infections[4].

Recurrent infections usually begin at about one year of age because maternally transferred antibody affords some passive protection till that time. Growth retardation is a common symptoms. Delay in diagnosis is frequent and is overlooked for many years before a diagnosis is made and treatment started A survey in England showed a delay in diagnosis in half of all children and virtually all adults. The length of ranged from 2-27 years in adults and 1 to 5 years in children[5] . IgA deficiency of common variable immunodeficiency(CVID) represent polar ends of a clinical spectrum due to the same underlying gene defect(s).Over a period of years, IgA-deficient patient may progress to the panhypogammaglobulinaemia phenotype characteristic of CVID, and vice versa. Patient with isolated IgA deficiency may present with an increased number of respiratory infections that may lead to bronchiectasis. Chronic diarrhoeal diseases also occur. It is also associated with arthritis and systemic lupus erythematosus. Patient with CVID may also present with signs and symptoms suggestive of lymphoid malignancy, including fever, weight loss, anemia, thrombocytopenia, splenomegaly, generalized lymphadenopathy, and lymphocytosis. Once suspected, lab diagnosis is simple and measurement of serum immunoglobulins will provide the diagnosis. If the level of immunoglobulins is normal, testing for antibody subclasses or functional antibody may be indicated. Serum complement levels and a normal lymphocyte count will serve as screening test to rule out co-existing deficiency disorders[6].

Pulmonary abnormalities develop in most patients with primary hypogammaglobulinemia. A new finding is that silent and asymptomatic progression of pulmonary changes may occur in patients despite an adequate immunoglobulin replacement therapy. High-resolution computed tomography is the method of choice in monitoring pulmonary changes [7] .The administration of intravenous immunoglobulin in adequate doses is an essential part of the prevention and treatment of all these complications[4]. We treated our patient with intravenous immunoglobulin(dose 400 mg/ kg) once. Immediately after giving immunoglobulin she felt better including improvement of joint pain. She had improvement on general well being, appetite and was afebrile during hospital stay. There was no adverse event seen during or after administration of immunoglobulin. Patient was advised for follow up.

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A CASE OF DEEP VEIN THROMBOSIS DUE TO PROTEIN C, PROTEIN S DEFICIENCY AND HYPER-HOMOCYSTINAEMIA, A RARE GENETIC ABNORMALITIES

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Abstract

Approximately 80% of Deep Vein Thromboses (DVTs) are clinically asymptomatic, 20% of those that actually demonstrate signs and symptoms. DVT associated with protein C and protein S deficiencies are rare genetic abnormalities that cause thrombophilia and lead to thrombosis. Here we describe a case of 55-year old male who presented with recurrent DVT of left lower limb and eventually diagnosed as a case of DVT due to protein C and protein S deficiency with hyperhomocystinaemia.. The particular interest in this case report is that it is important to consider sceening for thrombophilia incase of DVT with uncertain aetiology.

Key words *:Deep Vein Thromboses, protein C ,protein S, thrombophilia,hyperhomocystinaemia*

Introduction

Deep vein thromboses are a common and occasionally, fatal condition that are often clinically silent. whenever symptomatic, they can easily mimic or be masked by musculoskeletal conditions. Deficiencies of natural anticoagulants such as protein C and protein S leads to inherited thrombophilia which is defined as an enhanced inherited tendency to form venous thrombo-embolism without any apparent causes and tend to recur.[1,2] In the early 1980s protein C and protein S deficiencies were set as causes of inherited thrombophilia. Their prevalence in general population vary from 0.2% to 0.4% for protein C deficiency and 0.2% for protein S deficiency.[3] Homocystein has been recognized as an independent risk factor for atherosclerosis, arterial & venous thrombosis.[4] Individuals with unexplai- ned venous thrombosis has demonstrated a greater than expected number of individuals with blood homocystein level above the 95th percentile.[5]

Therefore, when any patient presents with deep venous thrombosis without precipitating conditions the screening for thrombophilia should be considered to find out the aetiology.

Case Report

A 55 years old gentleman, previously healthy male presented with a seven day history of gradual swelling and pain in the left lower limb. Pain was located over whole limb associated with redness, increased temperature and dilatation of the superficial veins. The pain was aggravated by sitting ,immediately after walking for certain distance and rest being the only relieving factor. The patient's sleep was disturbed, particularly lying on the affected side. The patient's past medical history revealed similar event of swelling of his left leg 1 year ago which had been treated successfully. In the patient's history, there were no history of hypertension, recent major surgery, immobilization or occurrence of any serious medical conditions. On physical examination,he had no abnormalities except swollen left lower limb. Local examination of left lower limb revealed , skin redness, swelling engorged superficial veins,mild tenderness and increased temperature over whole limb. All lower extremity pulses were present and of equal volume in both sides and there were no digital ischemia.

On Investigation, Complete blood count and blood film reveals, WBC-6.25X10^9 /L Hematocrit-38.8%,

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platelets-196X10^9/L, ESR:20 mm/1st hr, Hb-12.7 g/dl .Urine analysis was negative for protein or red cells. S. Creatinine-1.33 mg/dl. Fasting Blood Sugar and Lipid profile were normal. Prothrombin time-14.2 sec on admission (control 12 sec).USG of whole abdomen showed thrombus in Inferior Vena Cava . Duplex Examination of left lower limb showed; deep venous thrombosis of lf common femoral and popliteal segment. Protein C -12.4% (70-100) and protein S: 25% (60-130). Serum Homocysteine 26.96 micro mole /L.(5-15 micro mole)

Discussion

Deep venous thrombosis defined as a partial or complete occlution of a deep vein by thrombus, is a relatively uncommon yet important diagnosis in primary care practice.[6,7] The common clinical presentation of the venous thrombo-embolic disease (VTE) is with deep vein thrombosis of the leg and /or pulmonary embolism.[8] Risk factors of DVT include past history of deep vein thrombosis, pulmonary oedema, operative intervention, immobilization, trauma, neurological deficit, malignancies, sepsis, central venus catheter and hyper–coagulable state etc.[9] The prothrombotic states are protein C & protein S deficiency, activated protein C resistance, antithrombin III deficiency, elevated homocystiene level & abnormal lipid profile. The antiphospholipid syndrome, thrombocythemia & severe bacterial infection cause acquired hyper coagulable states.[9-13] Homozygous homocystinaemia accounts for 1 in 335 000 live births and is characterized by pre-mature vascular disease, thrombosis, mental retardation, skeletal abnormalities and lens disloca-tion. Heterozygous homocystinaemia is far more common, affecting up to 0.3-1% of the general populatnd is associated with recurrent deep venous thromboembolism.[14] Congenital protein C & protein S deficiencis are inherited disorders & the prevalence of protein C deficiency is 1 in 300 & that of protein S deficiency is less frequent than 1 in 1000. So the possibility of someone inheriting both deficiencies together is less than 1 in 300000, extremely rare.[15] Protein C and protein S systems are the major regulatory system of haemostasis. These proteins are vitamin K dependent proenzymes synthesized in the liver and become activated after binding to thrombin-thrombomodulin complex on the surface of endothelial cells. Thus activated protein C inhibits factors VIIIa & Va. Here protein S acts as a cofactor & exhibiting their anticoagulant property. Acquired causes of these deficiencies are seen in liver disease, DIC, therapy with L-asparaginase, Coumarin & severe infections etc.[16] These deficiencies commonly present with DVT, account for nearly 90% of all venous thrombotic episodes & Pulmonary embolism. & thrombosis in other venous sides about 5% of cases. Chronic sequelae can be extremely debilitating because of the post-thrombotic syndrome that can affect up to 20 % of the patients.[17] Recurrent DVT can occur with. or without thrombophilic conditions , the highest risk being in the first 6 month.[18] Homocysteinaemia has been identified as an independent risk factor for coronary artery disease, peripheral vascular disease and thrombosis. Heterozygous homocysteina- emia has been recognized in up to 25% of patients with recurrent venous thrombosis. [14,19]

Doppler ultrasonography and venography although being regarded as the diagnostic ‘‘gold standard”,require a high level of training for the operator in order to be reliable.Venography is also an invasive procedure.Recently, the diagnosis of DVT has been improved by performing a probability test using the measurement of D-dimer levels (the products of fibrin degradation that increase with venous thromboembolism).[20]

It is often make a fundamental error by testing for protein C & S deficiency when a patient is taking Coumarin (warfarin) or when they have had recent thrombotic event , as both of these causes protein C & S to be temporarily decreased So to confirm this deficiency, the patient must be off Coumarin for at least 14 days & must not have an active clotting episode in progress. So to confirm , we should repeat the test after 12 weeks & also testing the patient’s first-degree relatives.[15]

Hyperhomocysteinaemia appears to increa-and measurement of total fasting homocysteine levels alone can be misleading as it appears to misclassify 40% of patients.Conclusive results can be obtained using a moresensitive test by measurement of serum homocysteine levels before and after oral methionine loading.[19]

The goals of treatment for DVT include halting clot formation & preventing recurrence of thrombi & PE. About 30% of DVT patient have a thrombophilia. The main stay of treatment was unfractionated heparin followed by warfarin. Out patient anticoagulant therapy is recommended for 3 to 6 month for VTE & for more than 12 month for recurrent VTE.[21]

Conclusion

So whenever a DVT of uncertain aetiology is suspected, it might have benefit from hypercoagulability testing because it is important to focus on inherited blood clotting problems. Except in

the most severe cases, inherited problems are usually enough to cause DVT or pulmonary embolism on their own but this problems contribute to clot only when they are combined with environmental factors which activates the body's blood clotting machinery.If an inherited thrombophilia is diagnosed further screening and possible identification of other family members would lead to avoidance of known secondary risk factors and subsequent thrombo-embolic manifestations.

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