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Journal of Bangladesh College of Physicians and Surgeons (JBCPS)

INFORMATION FOR AUTHORS

MANUSCRIPT PREPARATION AND SUBMISSION

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FIve types of manuscripts may be submitted:

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Original Articles: These should describe new and carefully confirmed findings, and experimental procedures should be given in sufficient detail for others to verify the work. The length of a full paper should be the minimum required to describe and interpret the work clearly.

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I. A. Preparing a Manuscript for Submission to JBCPS

Editors and reviewers spend many hours reading manuscripts, and therefore appreciate receiving manuscripts that are easy to read and edit. Much of the information in this journal's Instructions to Authors is designed to accomplish that goal in ways that meet each journal's particular editorial needs. The following information provides guidance in preparing manuscripts for this journal.

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- Permission of the patients and/or their families to reproduce photographs of the patients where identity is not disguised should be sent with the manuscript. Otherwise the identity will be blackened out.

Preparation of manuscript:

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3. Style should be that of modified Vancouver.
4. Each of the following section should begin on separate page :
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   - o Text
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   - o References
   - o Tables and legends.

Pages should be numbered consecutively at the upper right hand corner of each page beginning with the title page

I. A. 1. a. General Principles

- The text of observational and experimental articles is usually (but not necessarily) divided into the following sections: Introduction, Methods, Results, and Discussion. This so-called "IMRAD" structure is a direct reflection of the process of scientific discovery.
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Research reports frequently omit important information. Reporting guidelines have been developed for a number of study designs that JBCPS journals ask authors to follow. Authors should consult the Information for Authors of this journal. The general requirements listed in the next section relate to reporting essential elements for all study designs. Authors are encouraged also to consult reporting guidelines relevant to their specific research design. A good source of reporting guidelines is the EQUATOR Network (http: //www.equator-network.org/home/) or CONSORT network (http:// www.consort-statement.org).

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- Structured abstracts are essential for original research and systematic reviews. structured abstract means introduction, methods, results and conclusion in abstract
- Should be limited to 250 words
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- Provide only directly pertinent primary references, and do not include data or conclusions from the work being reported.

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The Methods section should be written in such way that another researcher can replicate the study.

I. A. 6. a. Selection and Description of Participants

- Describe your selection of the observational or experimental participants (patients or laboratory animals, including controls) clearly, including eligibility and exclusion criteria and a description of the source population. Because the relevance of such variables as age and sex to the object of research is not always clear, authors should explain their use when they are included in a study report—for example, authors should explain why only participants of certain ages were included or why women were excluded. The guiding principle should be clarity about how and why a study was done in a particular way. When authors use such variables as race or ethnicity, they should define how they measured these variables and justify their relevance.

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- Emphasize the new and important aspects of the study and the conclusions that follow from them in the context of the totality of the best available evidence.
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Final checklists before you submit your revised article for the possible publication in the Journal of Bangladesh College of Physicians and Surgeons:

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- **Discussion**
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  - Δ Provide and discuss with the literatures to support the study
  - Δ Mention about limitation of your study
- **Conclusion**
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  - Δ Any recommendation
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  - Δ Abide by the Vancouver style
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CONTENTS

EDITORIAL

Geriatric Medicine : Bangladesh Perspective

Ageing is a natural phenomenon which cannot be stopped or altered. From the dawn of civilization man is feared of being old. There is a German proverb, 'Old age is a disease that you die from'. In fact elderly suffer from variety of diseases which make them feeble, dependant and even bed bound. This is why, every human being wants to get rid of old age but this is inevitable.

From the era of ancient medicine, physicians tried their best to alleviate the sufferings of elderly but only a few people reached their maximum due to early death by diseases and war. When modern medicine evolved the average life span of human increased with time. This demographic transition started slowly but got it's pace very soon. Now 11% of the world population is of 65 years of age or over.[1] Sometime around the year 2020, for the first time ever, the number of people aged 65 and older in the world is expected to exceed that of children under the age of 5.[1]

Ageing in Bangladesh is a recent phenomenon. Though internationally accepted cut of age is 65 years, above which peoples are considered elderly, in Bangladesh, persons with age of 60 or above are cited as geriatric age group in different literatures. However, in reality people in this country become older before the age of 60 because of poverty, physical hard working and, inability and illness due to malnutrition and geographical condition as well. Population trends in Bangladesh show that Bangladesh is well into third phase of demographic transition, having shifted from a high mortality-high fertility regime to a low mortality-low fertility one. In 2007, the number of the elderly people aged 60 and over in this country was 9.41 million and it has increased from 1.94 million in 1951 which is quite phenomenal.[2] Every year approximately 80,000 new elderly are entered into the group of the older persons. The estimates and projections show that the amount is certain to increase markedly with time.[3] This trend is expected to accelerate and by 2050 the number of persons aged 60+ are projected to be approximately 40.5 million, which is roughly 6 times higher than what it was in the year 2000.[4] So it is worth saying that the burden of old age diseases in our low income country will be enormous.

The problem of old age is not merely medical; it is physical, mental, economical and socio-cultural.[5] A cumulative approach is needed to combat the problem which should involve the medical professionals, social workers, political leaders, NGOs and media personals. The western society has become successful to blend the above sectors together. As a result, life expectancy and quality of living of aged people have been improved. The most notable contribution is from medical sector. Though the victory over communicable diseases has been achieved, other non communicable diseases like CVD, IHD, DM, malignancy and neuro-degenerative disorders have become more prevalent in developed countries. To overcome this new situation they have established geriatric medicine department in the hospitals targeting the aged people.

The scenario of Bangladesh concerning the old people is quite frustrating. A full blown geriatric policy in our country yet to be formulated. But this is a matter of hope that a geriatric medicine unit as a wing of medicine department of Dhaka Medical College Hospital has been launched in the late 2014. Now it is the time to flourish this small initiative to a complete geriatric medicine department which will be a great achievement and milestone in the history of medicine in Bangladesh.

After the establishment of geriatric medicine unit in DMCH in 2014, total 158 patients have been admitted till 11th March, 2015. 83 of them are male and the rest 75 are female. Multi morbidity is common. Mortality rate was 5.70%. Patients were treated by supportive care only due to lack of emergency medical facilities. Patients with diseases from every subspecialties of medicine e.g. cardiology, nephrology, neurology, endocrinology, respiratory medicine, gastroenterology, were admitted and treated accordingly. In male patients, the mostly encountered diseases were CVD, HTN, COPD, DM and

bronchial carcinoma. Female patients were predominantly suffering from DM with its complications and UTI. The mean age of admission was 67.4 years & the patient with highest age was a 108 years male.

After few months of observation in our geriatric medicine unit, certain difference has been noted between the global geriatric trends and that of Bangladesh. The disease pattern is more or less same but infectious disease is more prevalent here. The management strategy adopted in our unit is also quite similar comparing the developed countries. But the most notable difference is economical rather than medical. In the western society, there is a social security service for old and health insurance policy run by government. They bear all the treatment and rehabilitation cost. In our country, as old people are mostly dependant,[6] they cannot cope with expensive investigations or treatment. So they often fail to maintain the follow up schedule. As a result they come with more serious complications later on.

Regardless of several limitations, the geriatric medicine unit is running successfully since its initiation. We hope that positive steps from the government and donor organisations towards a geriatric friendly health care system will soon be on the cards. Increase in allocation and change in policy will see a rapidly flourishing discipline. The elderly are the people who have fought for this nation it is time we repay them and should not forget that pretty soon we will also be joining them.

(J Banagladesh Coll Phys Surg 2015; 33: 63-64)

Md. Azizul Kahhar
Prof. of Medicine, Dhaka Medical College

Reference:

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Prediction of Foetal Well-being with Non-stress Test

J RAHMAN[a], HA BEGUM[b]

Summary:

Introduction: *Non-stress test (NST) is the most common antenatal test performed to assess the foetus at risk of intrauterine hypoxia. On the other hand non-reactivity detected by NST increases the interferences of pregnancy by Caesarean section.*

Methodology: *A cross sectional descriptive study was carried out in the department of Obstetrics and Gynaecology at Dhaka National Medical College between July2007 and June 2008.*

Objectives: *The objectives of the study were (1) To observe the mode of delivery in cases of non-reactive non stress test (NST) and (2) To evaluate perinatal outcome of non-reactive NST.*

Results: *A total 137 high risk pregnant women were included in the study. Age of the women ranges from 16 to 32 years. The mean age of the women was 23.74 ± 3.71 year. Among them 44.53% were primaegravida and 55.47% were multigravida.*

Gestational age was between 35 and 42 weeks and mean gestational age was 38.34±1.42 weeks. Regarding foetal reactivity 61.3% (n=84) were reactive and 38.7 % (n=53) were non-reactive. Among the babies of non reactive NST 98.11% and 1.89% were delivered by caesarean section and vaginal delivery respectively. Whereas, 48.81% and 51.19% babies of reactive NST were delivered by caesarean section and vaginal delivery respectively. The percentage of caesarean section was much higher in non-reactive NST cases in comparison to that of reactive NST which was statistically highly significant (p value 0.0000). One minute after birth APGAR scoring revealed that 56.6% and 43.4% newborn of non-reactive NST had no depression(APGAR score 7-10) and mild depression (APGAR score 4-6) respectively. On the other hand 65.47% and 34.5% newborn of reactive NST had no depression and mild depression respectively at one minute after birth. Therefore, small difference was noticed in the neonatal status between the reactive and non-reactive NST which had no statistical significance (p value 0.507). Evaluation of the neonates with APGAR scoring done 5 minutes after birth revealed mild depression (APGAR score 4-6) in 24.53% and 20.24% of non-reactive and reactive NST cases respectively and no depression (APGAR score 7-10) was found in 75.47% and 70.76% in reactive and non-reactive NST respectively. So, 5 minutes after birth the neonatal status among reactive and non-reactive NST made no significant difference (p value 0.9266).

Conclusion: *Neonatal evaluation revealed that all foetuses were not compromised as detected by NST. Relying on NST the rate of Caesarean section has been increased. Reassessment of the foetal conditions was needed with the help of other techniques. Therefore NST alone is insufficiently predictive of neonatal outcome.*

Key word: *Non reactive NST, Caesarean section, Neonatal outcome*

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Introduction:

The most commonly and widely accepted used antenatal test of foetal well-being is cardiotocography (CTG) – either contraction stress test (CST) or non-stress test (NST).

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NST is primarily a test of foetal condition, where as CST is a test of utero-placental function. Nonstress test (NST) is a screening method used in pregnancy to identify foetus at risk of developing hypoxia.[1] Grossly its interpretation can be inferred as reactive and non-reactive NST. Reactive NST (normal) means presence of two or more fetal heart rate accelerations within a 20-minute period, with or without fetal movement discernible by the woman. Whereas nonreactive NST is presence of less than two fetal heart rate accelerations within a 20-minute period.[2] Time is extendable to 40 minutes before saying nonreactive.[3] A non-reactive nonstress test does not necessarily mean it is abnormal. Many times, the fetus is asleep during

the procedure. To encourage movement, the mother may be given something to eat or drink to awaken the fetus or the person performing the test may use a buzzer to stimulate movement. However, a non-reactive test could indicate the fetus is not getting enough oxygen. Low levels of oxygen are often caused by problems with the umbilical cord or placenta. It is generally done when the foetus is at least 28 weeks. Indications for having NST test are decreased fetal movement, post date, multiple pregnancy, insulin dependent diabetes, high blood pressure, kidney disease, heart diseases, thyroid dysfunction, intrauterine growth retardation (IUGR), oligohydramnios, polyhydromnios, and previous history of foetal loss during the second half of the pregnancy. Depending on the reason, some women may need to be tested once or more every week until the baby is born.[4] It was originally thought that CTG would be an asset in detecting early poor fetal outcomes, indicating the need for interventions to help improve chances of survival for newborn infants. However, available data suggest that when CTG is used as non-stress test, it can lead clinicians to use unnecessary or inappropriate interventions.[5]

Methodology:

A Cross sectional descriptive study was carried out at in-patient department of Obstetrics and Gynaecology of Dhaka National Medical College Hospital between July 2007 and August 2008 with the objectives to observe the mode of delivery in cases of non-reactive non stress test (NST) and to evaluate perinatal outcome of non-reactive NST.

High risk pregnant women of gestational age between 36 and 42 weeks admitted in the hospital with hypertensive disorders, diabetes mellitus, hypothyroidism, heart diseases and patients had complaint for decreased foetal movements were included in this study purposively. History was taken from the respondents regarding their age, parity, duration of pregnancy, presence of the above mentioned medical disorder and foetal movement by face to face interview. A pre-structured questionnaire was used for collecting data on demographic and obstetric characteristics from the respondents.

During examination the pregnant women were kept at left lateral or semi-recumbent position. An ultrasound transducer for recording of FHR located to obtain best foetal heart signal and tocodynamometer for recording of uterine activity was placed on maternal abdomen at the fundus of uterus. Recording was carried out over a period of 20 minutes at first. If non-stress test remained non-reactive external stimulus was given by palpation or gentle movement of foetus. Then another 20 minutes test was performed. Records were interpreted by senior obstetricians.

Respondents were followed up till delivery and the newborns were observed to see any depression by APGAR scoring.

Results:

During this study period a total 137 high risk pregnant women were included in the study. Age of the women ranges from 16 to 32 years. The mean age of the women was 23.74 ± 3.71 years. Among them 44.53% were primaegravida and 55.47% were multigravida. Gestational ages of the fetuses were between 35 and 42 weeks and mean gestational age was 38.34±1.42 weeks. More than 67 %(n=93) and 32.12% (n=44) babies were born by caesarean section and normal delivery respectively. Indications of caesarean section were Obstetric and non reactive NST in 44% and 56% cases respectively. NST records showed foetal reactivity in 61.3 % (n=84) and non-reactivity in 38.7% (n=53) cases. Among the cases of non-reactive NST 98.11% and 1.89% babies were delivered by caesarean section and vaginal delivery respectively. On the other hand, among the reactive NST cases, 48.81% and 51.19% babies were delivered by caesarean section and vaginal delivery respectively. The percentage of Caesarean section was higher in non-reactive NST than that of reactive NST which was statistically highly significant (P value 0.0000). APGAR scoring 1 minute after delivery, revealed no newborn with severe depression. More than 56% newborns of non reactive NST had no depression (APGAR score 7 to 10) and 43.4% of them had mild depression (APGAR score 4 to 6). On the other hand 65.47% and 34.5% newborns of reactive NST had mild and no depression respectively at 1 minute after birth. No significant difference was noticed in the neonatal status between the reactive and non-reactive NST cases at 1minute after birth (p value 0.507). At 5 minutes after birth, evaluation of the neonates with APGAR scoring was done again which revealed mild depression (APGAR score 4-6) in 24.53% and 20.24% of non-reactive and reactive NST cases respectively and no depression (APGAR score 7-10) in 75.47% and 70.76% respectively. So, after 5 minutes the differences of the neonatal status among reactive and non-reactive NST were not significant (p value 0.9266).

Table-I

Characteristics of Mothers and newborns:

Maternal age (in years)	Minimum		Maximum	Mean
	16		32	23.74 ±3.71
Gestational age (in weeks)	35		42	38.34 ± 1.42
Birth weight(in Kg)	1.8		3.8	2.77 ± 1.32
Sex of newborn	Male		Female	
	n	%	n	%
	78	57	59	43
Parity	Primae		Multi	
	n	%	n	%
	61	44.53	76	55.47

Table-II

Foetal reactivity and mode of delivery

	NVD		LSCS	
	n	%	n	%
Non-reactive NST n=53	1	1.89	52	98.11
Reactive NST n=84	43	51.19	41	48.81

The percentage of Caesarean section was more in non-reactive NST than that of reactive NST which is statistically highly significant. (Chi^2: 30.85, Degree of freedom 1, P value 0.0000)

Table-III

Foetal reactivity vs APGAR score 1 minute after birth

Foetal reactivity	APGAR score (0-3)		(4-6)		(7-10)	
	n	%	n	%	n	%
Non-reactive NST	0	0	23	43.4	30	56.6
Reactive NST	0	0	29	34.5	55	65.47

APGAR score of newborns of non-reactive and reactive NST shows no significant difference. Chi2 test was done which was not also statistically significant.

Chi2 0.44, Degree of freedom 1 and p value: 0.507

Table-IV

Foetal reactivity vs APGAR score 5 minutes after birth

Foetal reactivity	APGAR score (0-3)		(4-6)		(7-10)	
	n	%	n	%	n	%
Non-reactive NST (n=53)	0	0	13	24.53	40	75.47
Reactive NST (n=84)	0	0	17	20.24	67	79.76

APGAR score of newborns of non-reactive and reactive NST five minutes after birth shows no significant difference. Chi2 test was done. One expected cell <5, Yates corrected chi2 001 and p value: 0.9266 which was not also statistically significant.

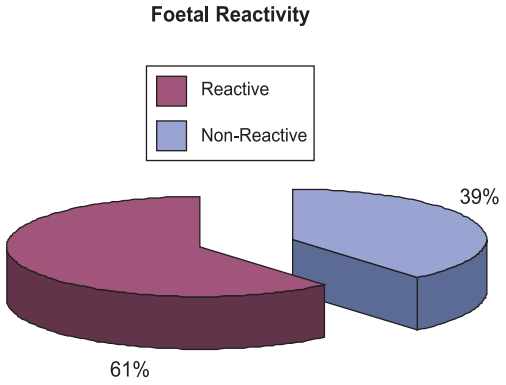

Fig.-1: *Distribution of the population according to foetal reactivity*

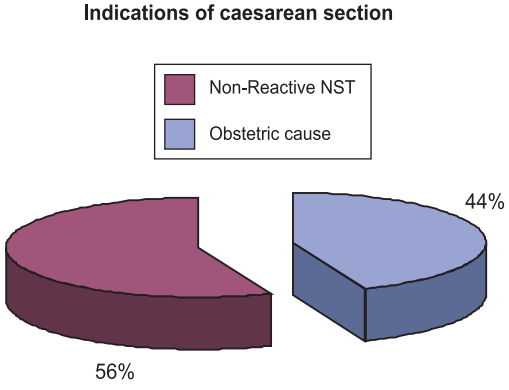

Fig.-2: Distribution of the cases of caesarean section according to indication

Discussion:

The present study was carried out among 137 high risk pregnancies. Non-reactive NST was found among 39% cases (fig1). Bhide A and coworker found 33.57% non-reactive NST among the high risk cases in a study which was similar to our finding.[4] In pregnancy medical disorder like hypertension, diabetes mellitus, heart diseases, kidney disease etc cause placental insufficiency and ultimately are responsible for foetal distress. In this study foetal distresses are detected as non reactive non-stress test (NST).

The study found that higher percentage of caesarean sections were performed among the cases of non-reactive NST than in reactive NST. Among the caesarean sections 56% were performed due to Non reactive NST and 44% due to other obstetric causes (Fig 2). We found that 98.11% babies of non-reactive NST were born by caesarean section and only 1.89% of them were born by normal vaginal delivery. In comparison, 48.81 % and 51.19% babies of reactive NST were born by caesarean section and normal vaginal delivery respectively (Table-II). Therefore the study found increased incidence of caesarean section due to non reactive NST which was statistically highly significant (Chi^2: 30.85, Degree of freedom 1, P value 0.0000). Similarly, a Cochrane Database of systemic review found high incidence of caesarean section due to non reactive NST.[6] An evaluation of NST in Nigeria also found that women who received a non-reactive NST were significantly more likely to deliver by Caesarean section than normal vaginal delivery.[7] Another study showed that over-reliance on the test has led to increased misdiagnoses of fetal distress and hence increased caesarean deliveries.[8]

In present study neonates were evaluated by APGAR scoring at 1 and 5 minutes after birth. Severe depression (APGAR score 0-3) was absent among the newborns of both reactive and non-reactive NST. We found mild depression (APGAR score 4-6) in 43.4% and 34.5% among the neonates of non-reactive and reactive NST respectively at 1 minute after birth. No depression (APGAR score 7-10) was noticed in 56.6% and 65.47% neonate of non-reactive and reactive NST respectively at 1 minute after birth. (Table-III) Therefore small differences were detected among the status of the newborns of non-reactive and reactive NST and the differences were not statistically significant (p value 0.507) also. Again, at 5 minutes after birth evaluation of the neonates with APGAR scoring was done which revealed mild depression (APGAR score 4-6) in 24.53% and 20.24% of non-reactive and reactive NST cases respectively and no depression (APGAR score 7-10) in 75.47% and 79.76% babies of non-reactive and reactive NST respectively (Table-IV). So, after 5 minutes the differences of the neonatal status among reactive and non-reactive NST were not significant (p value 0.9266).Therefore NST could not predict foetal reactivity properly in this study. Grivell RM also found NST insufficiently predictive of neonatal outcome in Cochrane Database of Systematic Reviews.[9]

Relying on non-reactive NST 56% of the population underwent caesarean sections. But, neonatal evaluation found 56.6% and 75.47% of the non-reactive NST babies had no depression at 1 and 5 minutes after birth respectively. Caesarean sections might not be necessary for many of these cases. Immer Bansil and coworkers also found in a study that unnecessary caesarean section is done relying on NST.[10]

In our study, the cases were high risk pregnancy and moreover the detection of non-reactive NST revealed that the foetus were at risk. Further evaluation of the at risk fetuses by sonographically related techniques like biophysical profile or Doppler velocimetry could not be availed in this study place. Therefore the obstetricians had to take decision relying on NST for termination of pregnancy by caesarean section to save the lives of the at risk fetuses which ultimately increased the rate of caesarean section.

Limitation of the study:

Whenever non reactive NST is found it should be reassessed with the help of Doppler velocimetry or biophysical profile. But Doppler velocimetry was not done here due to non availability of the investigation technique in this study place. Moreover many of the women belonged to low socio-economic condition and could not afford the cost of biophysical profile. NST is done with free of cost in this study place. As all these were high-risk pregnancies and reassessment of the non-reactive NST was not possible due to the above mentioned causes, in an intention to save the lives of at risk foetuses caesarean section was done in most of the cases which must not be practiced routinely.

Conclusion:

The study revealed that relying on NST the rate of Caesarean section was increased.

Neonatal outcome showed that in some cases caesarean sections were not needed.

Therefore, NST alone is insufficiently predictive of neonatal outcome. Caesarean section should not be done rationally with the only indication of Non reactive NST. Reassessment of non-reactive NST should be done by Doppler velocimetry or biophysical profile. This attempt was made with small number of study population. Large scale studies of longer duration and reliable interpretation of NST is necessary to get a real picture.

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Clinical and Biochemical Characteristics and Aetiology of Asymptomatic Raised Alanine Aminotransferase in Newly Detected Diabetes and Impaired Glucose Tolerance Patients

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Summary:

Objectives: Clinical evaluation and identification of aetiology of asymptomatic raised serum alanine aminotransferase (ALT) level in newly detected adult diabetic and impaired glucose tolerance (IGT) patients.

Methods: In this cross-sectional study, newly detected adult diabetic and IGT patients having asymptomatic raised serum ALT level of > 1.5 times of upper limit of normal were evaluated clinically and by laboratory tests.

Results: Total number of patients was 120, which was 3.1% of all newly registered diabetic and IGT patients over the study period. Male were 74 and female 46. Diabetes mellitus (DM) was found in 93.3% cases and IGT in 6.7%. Mean age was 43.1 years, mean body weight was 64.5 kg and mean body mass index (BMI) was 25.5 kg/m^2. Central (abdominal) obesity was found in 61.5% cases. Increased waist hip ratio was found in 86.3% cases. Hypertension and hepatomegaly were present in 35% and 5.8% cases respectively. Dyslipidaemia was found in 98.3% cases and 45% patients fulfilled criteria for metabolic syndrome. Regarding etiology, 76.7% cases had non-alcoholic fatty liver disease (NAFLD), 8.3% had HBsAg sero-positivity, 4.2% had anti-HCV sero-positivity and 3.3% had both NAFLD and HBsAg sero-positivity. In 7.5% cases no cause was found. Raised serum ALT level had a significant correlation with metabolic syndrome (p= 0.016) and increasing age (p= 0.008).

Conclusion: Elevation of serum ALT is common in DM and IGT. NAFLD is the commonest cause followed by hepatitis B and C virus infection.

Keywords: alanine aminotransferase, asymptomatic, diabetes mellitus, impaired glucose tolerance, non-alcoholic fatty liver disease.

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Introduction:

The prevalence of diabetes mellitus (DM) is increasing day by day. Patients with DM often have elevated levels of alanine aminotransferase (ALT).[1] Majority of them remain asymptomatic or have non-specific symptoms. The prevalence of asymptomatic elevated ALT is much higher in diabetic population than non-diabetic counterpart.[1] Elevated ALT is often a marker for non-alcoholic fatty liver disease (NAFLD), chronic hepatitis B and C virus infection and metabolic syndrome.[2] Up to 70% of type 2 diabetic patients may have NAFLD.[3] A strong association remains between hepatitis C virus (HCV) infection and type 2 DM.[4-

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[6] HCV related morbidity and mortality is higher in patients with DM than patients without DM.[7] Chronic hepatitis B virus (HBV) infection is also significantly higher in diabetic population.[8,9] If inadequately addressed, the spectrum of NAFLD can pass through simple hepatic steatosis to non-alcoholic steatohepatitis (NASH) to cirrhosis to hepatocellular carcinoma and death.[10-16] In this study, we have tried to clinically evaluate adult newly detected impaired glucose tolerance (IGT) and diabetic patients with asymptomatic elevated ALT and to find out the aetiology for raised ALT.

Materials and Methods:

In this prospective cross-sectional study adult newly detected IGT and DM patients with elevated (>1.5 times of upper normal limit) ALT were evaluated at Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM), Dhaka, Bangladesh from August to October 2009. Patients with diagnosis of chronic liver disease, acute hepatitis, congestive cardiac failure and history of regular alcohol or hepatotoxic drug intake were excluded from the study. The study was approved by the ethical review committee of Diabetic Association of Bangladesh. Informed written consent was taken from every patient after explaining the purpose and procedure. A standard questionnaire was filled up. Every patient was clinically evaluated and supplemented by appropriate laboratory investigations.

Anthropometric measurement

This included weight and height. Body mass index (BMI) was calculated as body weight in kilogram divided by height in meters square. Waist circumference was measured at the horizontal plane mid-way between anterior superior iliac spine and lower costal margin at the narrowest part of the waist line while the patient was standing and at the end of normal expiration. Hip circumference was measured at a horizontal plane passed through greater trochanter of femur on both side in standing position.

Blood Pressure

Blood pressure (BP) was measured by using a standard sphygmomanometer in the supine position after rest for at least 15 minutes. Hypertension was considered with blood pressure >130/85 mmHg and / or patient on regular use of anti-hypertensive medication.

Biochemical tests

All patients underwent the following tests: oral glucose tolerance test (OGTT), fasting lipid profile, serum ALT level, HBsAg, anti-HCV, ultrasonography of hepatobiliary system and pancreas. Additional investigations were done as required. Plasma glucose was measured by glucose oxidase method. Fasting plasma triglycerides (TG), high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c) were measured by enzymetic colorimetric method and total cholesterol was measured by enzymatic endpoint method using reagents of Randox Laboratories Ltd., UK. HBsAg was measured by micro-particle enzyme immune-assay (MEIA) method and anti-HCV was measured by ELISA.

Dyslipidemia

Dyslipidaemia was defined according to National Cholesterol Education Programme (NCEP) Adult Treatment Panel (ATP) III criteria- plasma triglyceride (TG) > 150 mg/dl, plasma total cholesterol > 200mg/dl, plasma HDL-c < 40 mg/dl in male / < 50 mg/dl in female or plasma LDL-c > 100 mg/dl.

Metabolic syndrome

Metabolic syndrome was defined according to International Diabetic Federation (IDF) criteria- central obesity (waist circumference >90 cm for male and >80 cm for female as for Asian) plus any two of the followings four criteria: (1) Raised plasma TG level >150 mg/dl or >1.7 mmol/L or specific treatment for this lipid abnormality. (2) Low plasma HDL-c <40mg/dl or <1.03 mmol/L for males or <50mg/dl or <1.29 mmol/L for females or specific treatment for this lipid abnormality, (3) Raised BP: systolic blood pressure >130 mm Hg or diastolic blood pressure >85mmHg, (5) Fasting plasma glucose $\geq$6.1 mmol/L.

Abdominal ultrasound examination

This was performed in all enrolled patients fasting for at least 10 hours by a single sonographer using SIEMENS Sonoline Antares. Liver echo pattern was graded as follow:

Grade I (mild): A single diffuse increase in fine echoes in the hepatic parenchyma with normal visualization of the diaphragm and intrahepatic vessel borders.

Grade II (moderate): A moderate diffuse increase in fine echoes with slightly impaired visualization of the intra-hepatic vessels and diaphragm.

Grade III (marked): A marked increase in fine echoes with poor or no visualization of the intra-hepatic vessel borders, diaphragm and posterior portion of the right lobe of the liver.

Statistical Analysis

Analysis was performed using statistical package for social science (SPSS) version 12. Data were expressed as mean, standard deviation (SD), percentage etc. Chi-

square test (x^2) was used for the comparison of qualitative data. Results were considered statistically significant at $p < 0.05$. Statistical analysis for association between raised serum ALT level and clinical parameter, biochemical parameter as well as ultrasonographic findings of hepatobilliary system were performed using univariate analysis with SPSS windows programme.

Results:

Over the study period a total of 3871 adult newly detected IGT and DM patients were enrolled in BIRDEM. Among them 120 patients were found to have asymptomatic elevated ALT of >1.5 times of upper normal limit (UNL) (considering exclusion criteria) which was 3.1%. Sixty (50%) of study subjects had ALT between 2-5 times of UNL, 52 (43%) had <2 times of UNL and 8 (7%) had >5 times of UNL. Central obesity, increased waist hip ratio, hypertension, hepatomegaly, dyslipidaemia, fatty liver and metabolic syndrome were present in 61.5%, 86.3%, 35%, 5.8%, 98.3%, 76.7% and 45% cases respectively. Base-line demographic, clinical and biochemical characteristics of the study subjects are shown in table I and II.

Raised serum ALT level had a significant correlation with the age of the study subjects (P 0.008) and metabolic syndrome (P 0.016), but not with BMI (P 0.891) (Table III).

NAFLD was the commonest aetiology for raised ALT in this study (Table IV). Other causes included hepatitis B and C virus infection. In 7.5% cases no cause could be identified.

Table-I

Demographic and clinical characteristics of the study subjects (N=120)

Male: Female	74 (61.7%): 46 (38.3%)
Mean age (years)	43.1±11.1
Mean body weight (kg)	64.5±11.1
Mean BMI (kg/m^2)	25.5±4.0
Mean systolic blood pressure (mm of Hg)	122.0±15.3
Mean diastolic blood pressure (mm of Hg)	79.4±7.7
Mean waist circumference (cm)	89.2±9.8
Mean hip circumference (cm)	90.7±6.8
Mean waist hip ratio	0.97±0.07

Table II

Biochemical characteristics of the study subjects (N=120)

Mean fasting plasma glucose (m.mol/L)	11.9±4.9
Mean 2 hour after 75 gm glucose (m.mol/L)	19.2±7.2
Mean ALT (U/L)	103.1±54.1
Mean total cholesterol (mg/dl)	196.3±44.9
Mean triglycerides (mg/dl)	187.9±99.9
Mean HDL-c (mg/dl)	36.8±8.2
Mean LDL-c (mg/dl)	125.8±36.6

Table-III

Frequency of raised ALT for different risk factors among the study subjects (N=120)

Risk factors	ALT<2XUNL (52, 43%)	ALT>2XUNL(68, 57%)	Total(120)	P value
Age (years)				
<35	7 (5.8%)	26 (21.7%)	33 (27.5%)	
35-50	29 (24.7%)	30 (25%)	59 (49.2%)	0.008
>50	16 (13.3%)	12 (10%)	28 (23.3%)	
Sex				
Male	34 (28.3%)	40 (33.3%)	74 (61.7%)	0.464
Female	18 (15%)	28 (23.3%)	46 (38.3%)	
BMI (kg/m^2)				
18.5-24.9	23 (19.2%)	31 (25.8%)	54 (45%)	
25-29.9	23 (19.2%)	28 (23.3%)	51 (42.5%)	0.891
e”30	6 (5%)	9 (7.5%)	15 (12.5%)	
Metabolic syndrome				
Present	30 (25%)	24 (20%)	54 (45%)	0.016
Absent	22 (18.3%)	44(26.7%)	66 (55%)	
Fatty liver (on USG)				
Present	42 (35%)	50 (41.7%)	92 (76.7%)	0.681
Absent	10 (8.3%)	18 (15%)	28 (23.3%)	

Table-IV

Aetiology of raised ALT in newly detected DM and IGT patients

Aetiology	Total (N=120)	DM (n=112)	IGT (n=8)
NAFLD	92 (76.7%)	89 (74.2%)	3 (2.5%)
HBV infection	10 (8.3%)	8 (6.7%)	2 (1.7%)
HCV infection	5 (4.2%)	5 (4.2%)	0 (0%)
NAFLD and HBV infection	4 (3.3%)	4 (3.3%)	0 (0%)
Unidentified	9 (7.5%)	6 (5%)	3 (2.5%)

Discussion:

Persistent elevation of ALT in asymptomatic patient accounts much of the challenge in clinical practice. In the United States, NAFLD is replacing alcoholic hepatitis and viral hepatitis as the most common etiology of chronically elevated serum ALT in both diabetic and non-diabetic individuals and 60-95% of them are obese.[17] In two different studies the prevalence of elevated ALT was considerably higher than in general population.[18,19] In the current study the prevalence of elevated ALT (>1.5 times of UNL) in newly detected IGT and DM was 3.1%.

NAFLD as the cause of raised ALT in our study was much higher than other studies, which may indicate insulin resistance as an important contributory factor in Bangladeshi diabetic and IGT population.[20,21] It was seen that the prevalence of HBV infection was higher among persons with diabetes than without diabetes.[8] In different studies HBV sero-positivity was found 2-4% but in our study it was much higher.[22, 23] The frequency of anti-HCV sero-positivity was lower in our study than two other studies. [24,25]

In different studies, male gender, younger age, greater waist circumference, presence of type 2 DM, poor glycaemic control, BMI >25 kg/m^2, long duration of DM were significant factors associated with an elevated serum ALT level.[19,26-28] On the other hand, high activity of ALT independent of age and obesity is associated with the occurrence of type 2 DM and metabolic syndrome.[29,30] In our study, elevated ALT had a significant correlation with the age and metabolic syndrome.

Our study had some limitations. Study sample was small and study period was short. Fibro-scan and liver biopsy was not done and NAFLD was diagnosed by USG only. Age and sex matched large-scale prospective study may provide more detail information. Liver biopsy should be considered for histological confirmation of etiology of raised serum ALT level.

In conclusion it could be said that mild to moderate elevation of serum ALT is a common in IGT and DM. So, serum ALT level should be checked in every diabetic patient at diagnosis and at regular intervals thereafter. NAFLD is the most common etiological factor followed by HBV and HCV infection.

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Influence of Number of Parity on Bone Mineral Density among Postmenopausal Women

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Summary:

Introduction- Osteoporosis is a common disease of postmenopausal women and is responsible for considerable morbidity and mortality. The most important single determining factor is low bone mass. Generally accepted risk factors of osteoporosis in women are low body weight, age, low physical activity and cigarette smoking. The effect of parity is controversial.

Objective-The main objective of the study was to assess the influence of parity on bone mineral density among the postmenopausal women.

Methods-In this study total 75 postmenopausal women aged 51-70 years of with parity 1-13 were studied. Parity was described as the number of births reported by the women. In this study T score of BMD of different bony sites lumber vertebrae and femur were analyzed. BMD were measured in the Institute of Nuclear Medicine at BSMMU. Correlations between BMD values with parity were detected.

Results- The mean age of the patients was 60 years with a standard deviation of ±9.32 years. All patients were within 51 to 70 years age range. A significant negative correlation was found in present study between parity and the T score measurement results obtained from L2, L3, L4, L2-4, Femur neck, Trochantor and Ward's triangle. This shows mean T-score of BMD were more negative as number of parity increases.

Key words: BMD. Postmenopausal women, Parity.

(J Banagladesh Coll Phys Surg 2015; 33: 75-78)

Introduction:

Osteoporosis is one of the long term health problems of postmenopausal women and is responsible for considerable morbidity and mortality. The most important single determining factor is low bone mass. Osteoporosis becomes clinically important only after fracture but treatment of the disorder after the onset of fracture is unsatisfactory[1.] Prevention is, therefore a more effective approach and thus measurement of bone density among postmenopausal women can protect from osteoporosis [2]. Bone densitometry by dual x-ray absorptiometry (DEXA) has been shown to be very reliable and sensitive in diagnosis of osteoporosis and decisions about treatment to prevent fracture. BMD is predominantly regulated by genetic. The rest of factors/ variance is influenced by environmental factors, such as a pregnancy or a period of lactation. [3.] Findings about the relation between parity and BMD are controversial [4]. So far in our country no study has been conducted to see influence of parity on BMD among the postmenopausal women. A better understanding of this relation may provide new opportunity for early intervention.

Objective-The main objective of the study was to assess the influence of parity on bone mineral density among the postmenopausal women.

Methods- This cross sectional observational study was carried out from the Jan 2006-Dec 2007 for a period of two years. This study was carried out in the Department of Obstetrics and Gynaecology Bangabandhu Sheikh Mujib Medical University (BSMMU) in collaboration with the Institute of Nuclear Medicine and Ultrasound, BSMMU campus , Dhaka. Ethical clearance was taken from the ethical committee of Department of Obstetrics and Gynaecology of BSMMU. Sampling technique was purposive consecutive sampling. The study population consisted of patients who had natural menopause from the age 51-70 years, attending the outpatient department of Department of Obstetrics and Gynaecology BSMMU Hospital and interested to perform BMD test and giving consent to participate in the study. Patients having menopause before the age of 50 years or patients having surgical menopause, history of fracture, chronic medical disorders affecting the

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bone mineral density such as chronic renal disorder, hypo or hyperthyroidism and medications known to affect the bone such as anticoagulant and anticonvulsant were excluded from the study. Cases were collected from outpatient department of Obstetrics and Gynaecology department of BSMMU. After describing the purpose and procedure of the study informed written consent was taken from every patient. All the patients answered the same specially designed questionnaire. Height and weight were measured with the participant wearing light clothing and no shoes. Body mass index (BMI) was used as an estimate of obesity. Bone mass density was measured at the Institute of Nuclear Medicine, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka by dual energy x-ray absorptiometry (DEXA), at the lumber spine and femoral neck by technician of BMD laboratory. The analysis is a computer-automated analysis and the measured BMD automatically displayed. Based on the definition of World Health Organization (WHO), T score of Bone Mineral Density values of different bony site such as lumber vertebra 2-4 and femoral neck were considered for analysis. Age of patients was divided into 4 groups, at 5 years interval such as 51-55years, 56-60years, 61-65 years and 66-70 years.

Data analysis was done by using SPSS by applying appropriate statistical formula such as unpaired t test, Chi squired test. Spearman's correlation and Pearson's correlation test were employed to measure the relation between the variable studied. The level of statistical significance was set at a p value of <0.05.

Results:

The present study was carried out in BSMMU from January 2006-December 2007. The study included 75 postmenopausal women ages of patients were within 51-70 years.

Table-I

Age distribution of the study subjects.

Age (year)	Frequency	Percent	Mean ± SD (range)
51-55	18	24.0	
56-60	27	36.0	
61-65	18	24.0	60.0±9.32 (51-70)
66-70	12	16.0	
Total	75	100.0	

Out of all patients maximum 27 (36.0%) were within 56 to 60 years age range followed by 18 (24.0%) within 51 to 55 years 18 (24.0%) within 61-65 years and 12 (16.0%) within 66 to 70 years age range. Mean age of the patients was 60 years with a standard deviation of ±9.32 years. All patients were within 51to 70 years age range. (Table I)

Table-II

Demographic related characteristics of the patients

Demography related factors	Mean±SD (range)
Age of menarche (year)	13.09±0.89 (10-15)
Age of menopause (year)	53.68±3.93 (50-57)
Duration of menopause (year)	11.41±7.95 (1-20)
Age of marriage (year)	16.65±2.50 (11-26)
Age of first pregnancy (year)	19.28±2.67 (15-28)
Para	6.07±3.23 (1-13)
Total duration of breast feeding (month)	133.35±69.89 (2-170)

Among the 75 patients, age of menarche were from 10-15 years, age of menopause ranges from 51-57 years, duration of menopause ranges from 1-20 years .Age of marriage were from 11-26 years , most of the patients age of first pregnancy was at the age of 15-28years. Among the patients parity varied from 1 to 13. As parity varies duration of breastfeeding varied according to number of children and it varied from 2-170 months.

Table-III

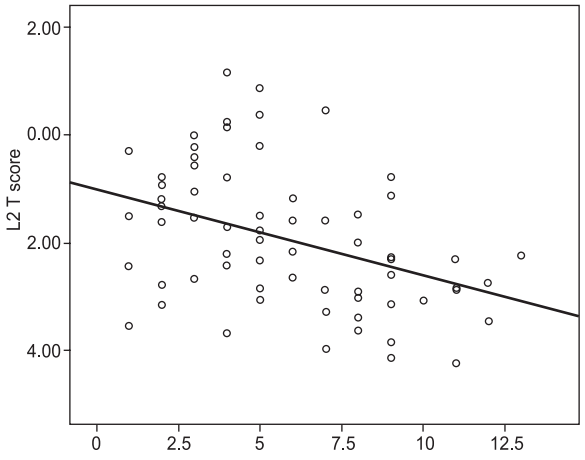
Correlations of parity with T score of different bony site

T score vs. parity	r value§	p value
L_2	-0.413 (**)	0.001
L_3	-0.402 (**)	0.001
L_4	-0.437 (**)	0.001
L_{2-4}	-0.436 (**)	0.001
Femur neck	-0.321 (**)	0.005
Trochantor	-0.308 (**)	0.007
Ward's triangle	-0.291 (*)	0.011

§r value was measured by Spearman's correlation
** Correlation was significant at the 0.01 level
* Correlation was significant at the 0.05 level

A significant negative correlation was found between parity and the T score measurement results obtained from L2, L3, L4, L2-4, Femur neck, Trochanter and Ward's triangle ($p<0.001$ in all areas, $r = -0.413$, $r = -0.402$, $r = -0.437$, $r = -0.436$, $r = -0.321$, $r = -0.308$ and $r = -0.291$ respectively). Table-III

r= -0.413, p= <0.001

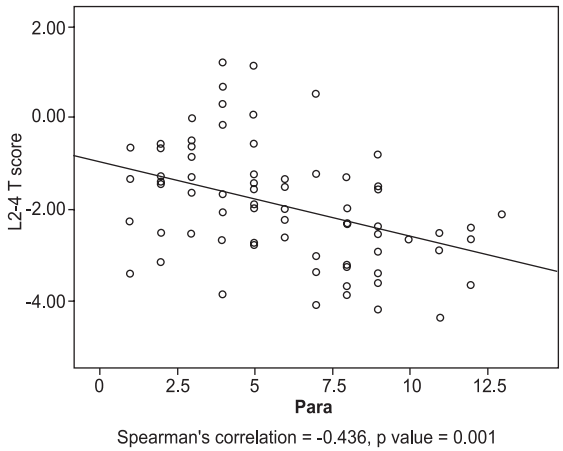


Spearmen correlation done

Fig.-1: *Scatter plot of correlation of T score (L2) and number of parity*

Figure-1 shows a significant negative correlation was found between parity and T score of BMD of lumber 2 vertebra. The value of Spearman correlation coefficient was -0.413 which indicates negative correlation between this two variables and it is highly significant, p value< 0.001.

r= -0.436, p= <0.001

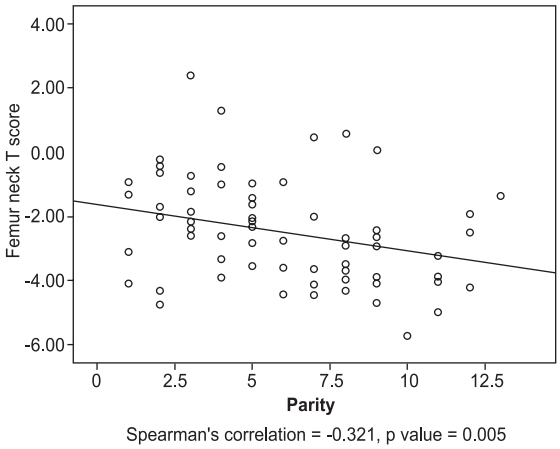


Spearmen correlation done

Fig.-2: *Scatter plot of correlation of T score (L_{2-4}) and number of parity*

Fig 2 shows a significant negative correlation between parity and T score of BMD of lumber 2-4 vertebra. The value of Spearman correlation coefficient was -0.436 which indicates negative correlation between parity and lumber 2-4 vertebra and it is highly significant, p value< 0.001 which indicates that with increasing parity T score of BMD of lumber 2-4 vertebrae decreases.

r= -0.321, p value= 0.005



Spearmen correlation done

Fig.-3: *Scatter plot of correlation of T score (Femur neck) and number of parity*

Fig 3 shows a significant negative correlation between parity and T score of BMD of femur neck. The value of Spearman correlation coefficient was -0.321 which indicates negative correlation between parity and femur neck and it is significant, p value < 0.01. This indicates that with increasing parity T score of BMD of femur neck decreases.

Discussion:

This cross sectional study was carried out in Bangladeshi postmenopausal women. The main aim of the study was to assess the influence of parity on bone mineral density among the postmenopausal women. For these purpose total 75 postmenopausal women aged 51-70 years of with parity 1-13 were studied. Parity was described as the number of births reported by the women.

World Health Organization (WHO) has published a guideline for the diagnosis of osteopenia and osteoporosis, which is related to an individual's BMD to the peak bone density of young adult (T-score). Osteoporosis is defined as a BMD of more than 2.5 SD below that of young adults (T-score -2.5 or more) where as a BMD between one and 2.5SD below that of young adults (T-score -1 to -2.5) is considered osteopenia.[5].

In this study T score of BMD of different bony sites lumber vertebrae and femur were analyzed. In this study the mean age of the patients was 60 years with a standard deviation of ±9.32 years.

A significant negative correlation was found in present study between parity and the T score measurement

results obtained from L2, L3, L4, L2-4, Femur neck, Trochanter and Ward's triangle. In a study conducted by Ozdemir et al. significant negative correlations were found between the number of pregnancies and BMD values for the spine and femur (neck, trochanter) which is resembled with present study.[6,7] In the present study mean T score of BMD of lumber 2,3,4,2-4 and femur neck, trochantor wards triangle were observed more negative as the number of parity increase. A study showed that BMD in healthy Saudi females was lower than that of their USA counterparts. That study also found that BMD value decrease as the number of parity increases. They contributed that to increase number of pregnancies, longer duration of lactation with prevalent vitamin D deficiency[8]. Hreshchyshyn et al. reported that the BMD of the femoral neck declined with an increasing number of live births, this finding is similar with present study but they found no change in lumber spine.[9] Fox et al. had observed a 1.4% increase in distal radius bone density with every pregnancy. In a study carried out by Hoffman et al. (1993), where the relation between parity and risk of fracture was investigated, among women who had three or more children, the risk of fracture were found 35-40% lower than that of nulliparous women. The study stated that BMD increases in subsequent pregnancies. These findings do not correlate with present study.[10]

Gur et alfound a significant negative correlation between the number of pregnancies and BMD of the spine, trochantor, and Wards triangle. But in the present study a significant negative correlation was found between number of pregnancies and spine, trochantor, Wards triangle and also femur neck.[4] These findings correlate with present study.

Carranza et al. found no significant correlation between the numbers of pergnancy T scores at lumber spine and femur. But in this study a significant negative correlation was found between the numbers of pregnancy and T scores at lumber spine and femur.[11]

The present study revealed a negative and correlation between increasing parity and decreasing BMD at lumber vertebrae and hip. The BMD values found decreased as the number of parity increased.

This study was conducted in one tertiary hospital with a small sample size so it reflects a small population. Hormonal factors that influence osteoporosis were not measured in this study. History of oral contraceptive intake should be evaluated for further study. Further prospective studies with a large sample should be carried out for evaluation of an association between parity and bone mineral density.

Conclusion- The results of the study conclude that among the postmenopausal women ,T score of bone mineral density decreases as the number of parity increases.

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Pregnancy Profile and Perinatal outcome in Gestational Diabetes Mellitus: A Hospital Based Study'

FN MUSTAFA

Summary:

Objective: *The objective of the study was to measure the proportion of GDM in antenatal outdoor of a hospital, to find out the mean gestational period at which most delivery occurred in GDM and to assess the perinatal outcome.*

Study design : It was a prospective analytical study conducted in BSMMU, from March,2010 to February,2011.

Method: 1489 pregnant women, not known to be diabetic previously, were selected by consecutive sampling in first trimester from Obstetrics outdoor, BSMMU. Their FBS and blood glucose 2hrs after 75gm oral glucose were recorded. We investigated blood glucose in first, second and third trimester in the same pregnant women for screening GDM. Cut off GDM values in fasting stage was e" 6.1mmol/l and 2 hrs after 75gm oral glucose was e" 7.8mmol/l .

Result: The proportion of GDM in Obstetrics Outdoor of BSMMU was 6.85%. The mean gestational period at which delivery occurred was lower in GDM (36.9 ± 2.2 wks) than that in non-GDM (39 ± 1.6 wks) .The most common (31.4%) gestational week during delivery in GDM was 37 completed weeks. Birth weight of 40.2% babies were in the range of 2.5kg to 3.0kg, 31.4% in the range of 3.1 to 3.5 kg and 1% neonate died after birth in GDM.

Conclusion: Presently GDM is diagnosed early. The mean gestational period at delivery in GDM is 36.9±2.2 wks. Majority neonatal birth weight is of normal range. Neonatal mortality rate is not increased and is not significantly different from non-GDM women.

Key words: GDM, Gestational week at delivery.

(J Banagladesh Coll Phys Surg 2015; 33: 79-85)

Introduction:

Gestational diabetes mellitus (GDM) is defined as carbohydrate intolerance of varying degrees of severity with onset or first recognition during pregnancy[1] . Diabetes mellitus is the commonest medical complication of pregnancy [1].

GDM carries significant and often potentially grave fetal and maternal complications. Its early detection and treatment reduces much of the maternal and fetal complications. So, it is very important to diagnose and manage GDM for maternal and fetal wellbeing. The maternal consequences of diabetes in pregnancy are increased incidence of preeclampsia, infection, postpartum bleeding and operative delivery. The consequences to the fetus are more serious than those to the mother. Among the fetal effects, the frequency of congenital abnormalities is increased in women with poorly controlled type I diabetes and the incidence of fetal macrosomia is increased in women with gestational and type II diabetes. There is also increased frequency of birth trauma, neonatal metabolic complications, perinatal death etc.

The prevalence of diabetes varies worldwide and among racial and ethnic groups[2] . Prevalence may range from 1-14% of pregnancy depending upon the population studied and the diagnostic tests employed [3] . These wide ranges of variation in prevalence are due to different diagnostic criteria applied for GDM diagnosis (i.e. ADA, WHO)[3,4].

There are various recommendations and testing methods to diagnose GDM. In Bangladesh, BIRDEM, BSMMU adopt the diagnostic criteria of WHO (1999) for diagnosing Gestational Diabetes Mellitus. The cut off value of Fasting Venous Glucose Concentration is e"6.1 mmol/lit, and cut off value of 2 hr post 75 gm oral glucose load is ≥7.8 mmol/lit[7] for GDM. Important risk factors of Gestational Diabetes Mellitus include higher maternal age, marked obesity (BMI> or equal to 30kg/m^2), personal history of GDM, ethnicity and family history of Type II diabetes in first degree relatives[5] . The prevalence of GDM increases with age, becoming more frequent over the age of 30 years [6,7] .

The above are the important risk factors for GDM. They actually affect blood glucose level by increasing insulin

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resistance. And the most important reason why pregnancy exacerbates the diabetic tendency of asymptomatic women is the progressive increase in insulin resistance that occurs during gestation. During the first and early part of the midtrimester, there is increased sensitivity to insulin and diabetic patients have a tendency towards hypoglycemia. This enhanced insulin sensitivity is probably due to the high levels of estrogen. The opposite occurs in the third trimester when a given dose of insulin has a decreased hypoglycemic effect. The increased insulin resistance stems mainly from the antagonistic effect of human placental lactogen. Accelerated insulin catabolism by renal and placental insulinases and the anti-insulin effects of other hormones (cortisol, estriol, progesterone) produced in large amounts during pregnancy also contribute to insulin resistance. The increased insulin resistance in the third trimester explains why gestational diabetes is more common after 26 weeks.[8]

Diabetes increases the risk of maternal morbidity, perinatal morbidity and mortality which are many times higher than that of non-diabetic pregnant women. The most significant maternal risk with gestational diabetes is the 35% to 50% probability to developing type II diabetes later in life. Older studies indicated a significant increase in the incidence of preeclamsia but recent evidence questions this finding[9]. The incidence of cesarean section is higher in GDM women than in a non-diabetic population. Polyhydramnios occurs frequently in GDM, particularly when the fetus is macrosomic.[10]

Fetal and Neonatal Risks:

Fetal macrosomia, defined as a birth weight greater than or equal to 4000 g, occurs in 17 – 29 % of pregnancies with gestational diabetes as compared to 10% in nondiabetic population [11]. The incidence of neonatal hypoglycemia is greater in GDM than in normal pregnancies[12]. In a recent study, neonatal morbidity was assessed by a composite outcome that included stillbirth, neonatal macrosomia / LGA (Large for Gestational Age), neonatal hypoglycemia, erythrocytosis and hyperbilirubinemia. Composite morbidity is present in 59% of untreated GDM, 18 % of treated GDM and in 11 % of nondiabetic subjects and the incidence of fetal death was 5.4, 3.6, and 1.8 per 1000 in untreated, treated and nondiabetic mothers, respectively[13].

In the above context, the present study was undertaken to measure the proportion of gestational diabetes in antenatal outdoor of a hospital and to find out the clinical profiles, pregnancy events and perinatal outcome of the gestational diabetes mellitus.

Materials and methods:

The objective of the study was to see the proportion of gestational diabetes in antenatal outdoor of a hospital and also to find out the mean gestational period at which most delivery occurred in GDM and to assess the perinatal outcome. It was a prospective analytical study conducted in BSMMU, from March,2010 to February,2011. Sampling procedure was consecutive sampling.

A total number of 1852 pregnant women were enrolled into our study, who attended for antenatal check up in BSMMU in their first trimester from 1 March 2010 to 28 Feb 2011. But finally we could collect full data of 1489 women which we took as our sample size. An informed consent was taken from all women who fulfill inclusion criteria and were interested to participate in this study. There was no age limit. The women in first trimester were enrolled. All relevant data including medical and obstetric history, family history and other risk factors for GDM were recorded in a data form. We requested the women to come next day for investigation of blood sugar level after overnight fasting. Their fasting blood glucose level was recorded, thereafter 75 gm glucose was given to take orally, then 2 hours later blood glucose level was again recorded. We investigated blood glucose level in first, second and third trimester; three times in the same pregnant women for screening GDM. Cut off values of Venous Glucose Concentration for GDM , in Fasting is e" 6.1 mmol/l and 2 hr after 75 gm glucose is e" 7.8 mmol/l . The women who were found to have Gestational Diabetes Mellitus were considered as cases and those who were found to have normal blood glucose level were considered as control group. Then comparison between the findings of two groups were done. The women were followed up and we kept contact with them till delivery. Many women were delivered in BSMMU, but many were delivered in other hospitals too, but we collected the data from the women later by communicating with them through telephone. Women, whose blood glucose level was not controlled by diet alone, were referred to medicine department

where insulin was added. The pregnancy events and outcome were assessed. Statistical analysis of all the data was done to evaluate the significance of the findings of the two groups.

Result:

The proportion of GDM in Obstetrics Outdoor of BSMMU was 6.85 %.

Age distribution:

Table-I

Age distribution between GDM and non-GDM groups

Age*(years)	Group		p-value
	Case (n = 102)	Control (n = 1387)	
< 20	0(0.0)	187(13.5)	
21 – 30	71(69.6)	1045(75.3)	
>30	31(30.4)	155(11.2)	
Mean ± SD	29.0 ± 4.2	24.8 ± 4.1	< 0.001

* Data were analysed using Student's t-Test and were presented as mean ± SD.

The mean age was significantly higher in the case group than that in the control group (29.0 ± 4.2 years vs. 24.8 ± 4.1, p < 0.001) . (Table I). The pregnant women of case group were generally older with 30.4% being more than 30 years old and none was 20 or <20 years; whereas the control group was comparatively young with 13.5% below 20 years and only 11.2% > 30 years old.

Detail age distribution of case and control groups

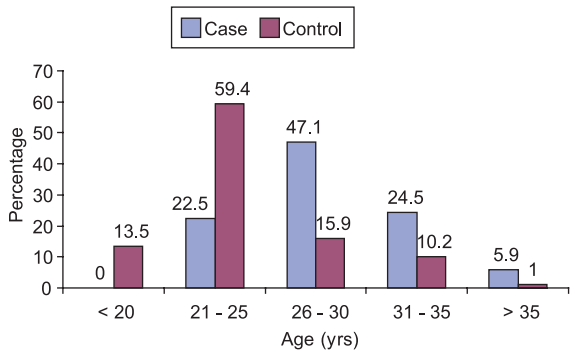

Fig.1: *Detail age distribution of case and control groups*

In non GDM group the largest age group is 21 – 25 yrs, whereas, in the GDM the largest age group is 26 – 30 yrs.

Table-II

Parity distribution between cases and control groups

Parity	Group		p-value
	Case (n = 102)	Control (n = 1387)	
Primipara	22(21.6)	462(33.3)	< 0.001
Multipara	80(78.4)	925(66.7)	

Figures in the parentheses indicate corresponding percentage.
* Data were analysed using Chi-square ($\div^2$) Test

Over three-quarter (78.4%) of the cases and 66.7% of the controls were multipara (p < 0.001).

Table-III

Socio-economic condition of case and control groups

Social class	Group		p-value
	Case (n = 102)	Control (n = 1387)	
Lower class	6(5.9)	435(31.4)	< 0.001
Middle class	43(42.2)	447(53.9)	
Upper class	53(52.0)	205(14.8)	

Figures in the parentheses denote corresponding percentage.
* Data were analysed using Chi-square ($\div^2$) Test.

52% of the case group belonged to upper social class and very few to lower class, while about one-third (31.4%) of the control group belonged to lower social class and only 14.8% to upper class. The groups were significantly different with respect to social class (p < 0.001) (Table III).

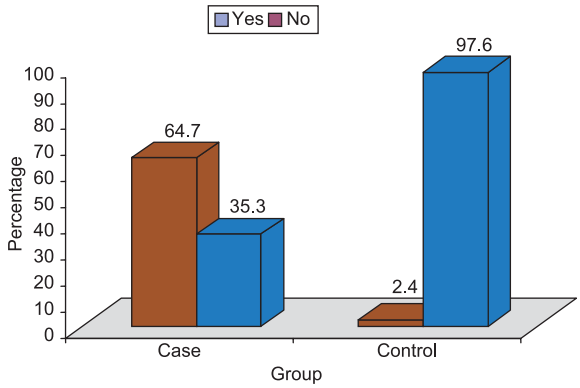

Fig.-2: *Distribution of cases and controls by family history of diabetes*

About 64.7% of the cases had family history of diabetes mellitus as compared to only 2.4% of the control group ($p < 0.001$) (Fig. 2). The difference is very very significant.

Treatment given:

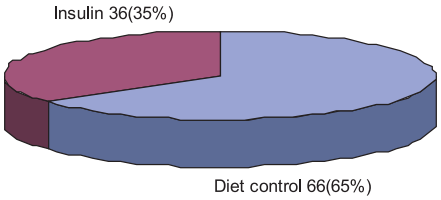

Fig. 3: *Distribution of GDM patients by measures taken for diabetes control*

About two-third (65%) of the cases of GDM were controlled by dietary manipulation, while the rest 35% required insulin.

Table IV

Gestational period at which delivery occurred

Gestational period *(weeks)	Group		p-value
	Case (n = 102)	Control (n = 1387)	
< 37	32(31.4)	187(13.5)	
37 – 40	68(66.7)	1145(82.5)	
> 40	2(1.9)	55(4.0)	
Mean ± SD	36.9 ± 2.2	39 ± 1.6	< 0.001

* Data were analysed using Student's t-Test and were presented as mean ± SD.

Table IV shows that the incidence of preterm delivery (delivery before 37 weeks of gestational period) was much higher in the case group (31.4%) than that in the control group (13.5%). The mean gestational period at which delivery of the babies occurred was significantly lower [36.9 ± 2.2] in the GDM than that in non-GDM [39 ± 1.6] ($p<0.001$).

Detail distribution of GDM patients by gestational period at delivery

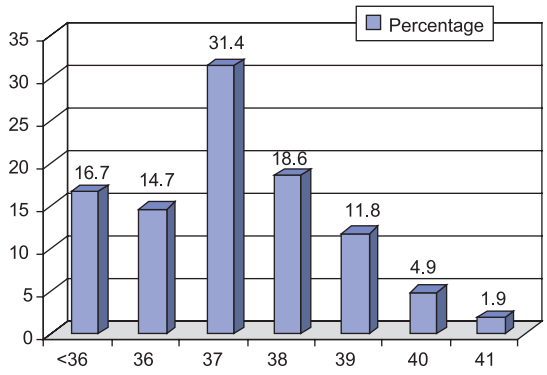

Fig.-4: *Detail distribution of GDM patients by gestational period at delivery*

The most frequent group of gestational period at delivery in GDM women (31.4%) is delivery at 37 completed weeks of pregnancy.

Table-V

Mode of delivery in two groups

Mode of delivery	Group		p-value
	Case (n = 102)	Control (n = 1387)	
Vaginal delivery	23(22.5)	747(53.9)	< 0.001
LSCS	79(77.5)	640(46.1)	

Figures in the parentheses denote corresponding percentage.

A significantly higher proportion of case group was delivered by caesarean section (77.5%) compared to their control counterpart (46.1%) ($p<0.001$).

Table-VI

Comparison of birth weight of the babies of two groups

Birth weight*(kg)	Group		p-value
	Case (n = 102)	Control (n = 1387)	
< 2.5	11(10.8)	126(9.1)	0.001
2.5 – 4.0	83(81.4)	1244(89.7)	
> 4	8(7.8)	17(1.2)	

Figures in the parentheses denote corresponding percentage.
* Data were analysed using Chi-square ($\div^2$) Test

Large majority of the GDM babies are not macrosomic, 92.2% of the GDM babies and 98.8% babies of the non-GDM women are in the non-macrosomic level, < 4.0 kg.

Though the incidence of macrosomia (birth weight of a baby > 4 kg) was observed to be significantly higher in

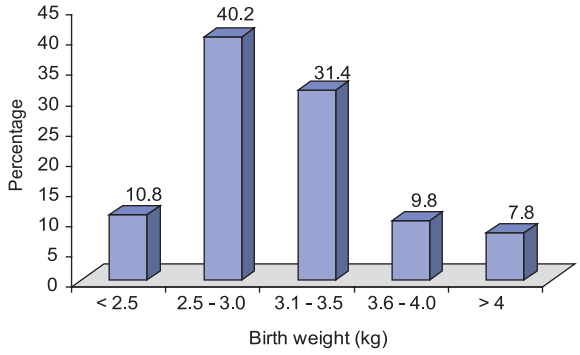

Fig.-5: *Detail picture of Birth weight of neonates born of GDM women*

the case group (7.8%) than that in the control group (1.2%). (p = 0.001), but the proportion of macrosomia in GDM group itself is not very high (Table VI).

The largest single group of birth weight of neonates (40.2%) is in the range of 2.5 - 3.0 kg. The second largest group (31.4%) is in the range of 3.1 to 3.5 kg. Macrosomic babies are only 7.8 % of the total GDM group babies.

Table-VII

Comparison of perinatal outcome between two groups

Perinatal outcome*	Group		p-value
	Case (n = 102)	Control (n = 1387)	
Intrauterine death	6(5.9)	1(0.1)	< 0.001
Congenital anomaly	3(2.9)	5(0.4)	0.001
Neonatal death	1(1.0)	3(0.2)	0.247

Figures in the parentheses denote corresponding percentage.

Table VII shows that 5.9% of the cases had intrauterine death of the foetus, 2.9% of the neonates had congenital anomaly and 1% died after birth. In the control group these figures were 0.1%, 0.4% and 0.2% respectively. The intrauterine death and congenital anomalies were significantly higher in the case group than those in the control group (p < 0.001 and p = 0.001 respectively).

Discussion:

Among 1489 antenatal women studied 102 women were found to have GDM. So the proportion of GDM in obstetrics outdoor of BSMMU was 6.85 % . In our country, we have not got any prevalence data of GDM. In a study first published in 2008, the prevalence of GDM in India varied from 3.8 to 21% in different parts of the country.[14] In a study, the prevalence of GDM in south India (Tamil Nadu) was detected as 17.8% women in urban, 13.8% in semi urban and 9.9% in rural areas.[15]

In a study , conducted in Maharaj Nakorn Chiang Mai Hospital in Thailand in 2001, the prevalence of GDM was found to be 7.05 % . In their study , 69% were multipara, mode of delivery was by vaginal route in 72.4 % and by LSCS in 27.6 %. [16]

The age range of our study was less than 20 years to more than 40 years. The pregnant women of case group were generally older with 30.4% being more than 30 years old and none was 20 or < 20 years; whereas the control group was comparatively younger with 13.5% below 20 years and only 11.2% > 30 years old. The mean age was significantly higher in the former group than that in the latter group (29.0 ± 4.2 years vs. 24.8 ± 4.1, p < 0.001). Since in 26 - 30 years age range pregnancy is frequent and more women of this group are either 2nd gravida or more. So an evaluation of age distribution reveals that the majority of GDM women were of 26 -30 yrs age range (47.1%).

A study was run in the Aga Khan Maternity Home, a regional obstetric centre providing maternity care in the northern areas of Karachi. All pregnant women registered for antenatal care and delivery who were not known to be diabetics were included in the study, which ran from January 1990 to December 1992. It was seen that almost one-half were in the range of 25.1-30 years of age [17] (same as in our study, 47.1% in 26 -30 yrs).

In our study, among GDM women 78.4% were multipara. In a study, conducted in Maharaj Nakorn Chiang Mai Hospital in Thailand in 2001, 69% were multipara .[16]

In our study, it is clearly evident that GDM was more frequent in higher socioeconomic status. This finding might be probably due to family history of GDM was more frequent in higher socio economic status for their life style. In the majority of GDM women (64.7%) family history of Diabetes Mellitus was present, while it was almost absent (only 2.4%) in non- GDM control group. In a study conducted among GDM women in Kuwait, in 2002, 61.8 % had family history of DM, the finding is almost same as ours. [18]

In our study, majority of women (65%) had controlled GDM by dietary intervention alone. 35% women needed insulin. The finding contradicts with the finding in the study conducted among GDM women in Kuwait , in 2002, there 71.1 % GDM women needed insulin therapy and 28.9 % were treated by diet control alone.[18]

A significantly higher proportion of GDM group was delivered with caesarean section (77.5%) compared to their control counterpart (46.1%) (p<0.001) . LSCS was done mainly due to patients' complaints of less fetal movement, abdominal pain in previous history of LSCS, higher level of blood glucose level and induction failure. In a study , conducted in Maharaj Nakorn Chiang Mai Hospital in Thailand in 2001, among GDM women, mode

of delivery was by vaginal route in 72.4 % and by LSCS in 27.6 %.[16] It is in contrast to the findings in our study. Among 394 women delivered at St. Paul's Hospital, University of British Columbia, Vancouver BC., between January 1, 1995, and December 31, 2001, the rate of LSCS was 36.3% vs. 23% in control non GDM women ($P < 0.05$). [19]

The caeserean delivery rate was higher in our study may be because our study was done in 2011 and their study was done in 2001. In this time period, caeserean delivery rate has increased worldwide as a whole. In a study conducted in Virginia, from 2000-2004, caesarean sections increased in mothers with GDM by 84 percent.[20]

The incidence of preterm delivery (delivery before 37 weeks of gestational period) was much higher in the GDM group (31.4%) than that in non GDM group (13.5%). The mean gestational period (36.9 ± 2.2 wks) at which delivery of the babies occurred was significantly lower in GDM than that in non-GDM (39 ± 1.6 wks) .

In our study we have ascertained that the single largest group (31.4 %) of GDM women were delivered at the gestational period of 37 completed weeks. Since women were suffering from GDM and some were treated with Inj Insulin , so they were electively delivered at 37 completed weeks. Many also complained of less fetal movement , they were also delivered at this stage. The second largest group of delivery was at the gestational period of 38 completed weeks. Lesser percentage of women were delivered at 40 weeks (4.9%). Since they were attending antenatal chek up, so only a few percentage (1.9%) were delivered at 41 weeks. Since chance of intrauterine death increase in the last few weeks, women had regular antenatal check up and were not allowed beyond EDD. But some women themselves decided to carry the pregnancy further and were delivered at 41 weeks.

In France, in a study in 2001 to 2004, in Reunion Island, there was increased rate of induction of delivery at the gestational period of 38 week.[21]

In our study, birth weight of single majority group of babies (40.2%) of GDM women were in the range of 2.5kg to 3.0kg. Since a very large group of GDM women delivered at 37 completed weeks and diabetes were under control, so baby weight was in this range. In this study, second largest group 31.4% babies were in the range of 3.1 to 3.5 kg. Only 17.6% babies of GDM women weighed > 3.5 kg . Since the women were under regular antenatal check up, blood glucose level was controlled, larger portion of babies were not macrosomic. Though the incidence of macrosomia was observed to be significantly higher in the case group (7.8%) than that in the control group (1.2%).

In a study done in the United States in 1996 on Maternal gestational diabetes and birth weight of babies, it was seen that the mean birth weight was 3.4 kg for girls and 3.6 kg for boys.[22] In another study in the USA macrosomia may affect 20% of patients with GDM.[23] In an study in Uganda , macrosomia was found in 36.7% of GDM women in comparison to 5 % in control group. [24] In a study in India macrosomia in GDM women were found to be 13% .[25]

In the study, conducted in The Aga Khan Maternity Home of Karachi, which ran from January 1990 to December 1992, there was an aggregate birth weight of babies of GDM women was 3.24kg , and perinatal loss was 2.08%.[17] In our study, perinatal death occurred to 7 babies out of 102 (6.86 %) . Six babies became IUD and one baby died after 2 days of delivery. About 6% of the GDM cases had intrauterine death of the foetus, 2.9% of the neonates had congenital anomaly, 1% neonate died after birth. In the control group, these figures were 0.1%, 0.4% and 0.2% respectively. The intrauterine death and congenital anomalies were significantly higher in the case group than those in the control group.

Limitations:

We had some limitations of our study. Initially 1852 women were enrolled into our study, but after dropout we could finally collect the full data of 1489 women. Next limitation was that all the sampled women were not delivered in the same institution they were enrolled first, in BSMMU. We had to collect some data over telephonic conversation with the women in the study.

Another limitation of the study was that a large number of women delivered their babies around 37 completed weeks for various complications, if their pregnancy could continue till EDD, many more babies would have been macrosomic.

Conclusion:

In our study, 5.9 % of the cases had intrauterine death of the foetus, 2.9% of the neonates had congenital anomaly and 1% neonate died after birth. In the control group these figures were 0.1%, 0.4% and 0.2% respectively. Neonatal mortality rate is not significantly different among GDM & non-GDM . So, we can conclude that now a days GDM is diagnosed early . In our study, most common gestational period at delivery is 37 completed weeks in GDM, majority neonatal birth

weight is within normal range and neonatal mortality rate is not increased or significantly different from non-GDM.

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REVIEW ARTICLE

Antiphospholipid Syndrome in Pregnancy

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(J Banagladesh Coll Phys Surg 2015; 33: 86-90)

Introduction:

The antiphospholipid syndrome (APS) is a systemic autoimmune disease characterized by arterial and venous thrombosis, gestational morbidity and presence of elevated and persistently positive serum titers of antiphospholipid antibodies. Antiphospholipid syndrome may exist as an isolated immunologic derangement primary APS (PAPS) or as secondary antiphospholipid syndrome (SAPS) where it occurs in association with autoimmune disease, most commonly SLE. The diagnosis of APS should be suggested whenever patient has history of repeated pregnancy loss without any fetal malformation or foetal death in utero. Other pregnancy complications mainly include intrauterine growth restriction (IUGR), oligohydramnios, preeclampsia, fetal distress, and preterm labor [1]. A severe complication of pregnancy, which greatly increases its risk in case of APS, is VTE. Pregnant and postpartum women are approximately 4 to 5 times more likely to develop VTE compared with non pregnant women [2]. Many other clinical manifestations may occur. Pregnant women with APS are considered high-risk obstetric patients, and medical care should be instituted keeping this in mind.

Methods:

The review article is based on systematic search through Pubmed and other search engine like google and google scholer. The materials was mostly taken from Medline databases, Medscape references,the Cochrane databases systematic reviews .The literature search was done focusing more on the latest research. We obtained additional articles from bibliography of the selected manuscripts. We paid attention to systematic reviews, randomised clinical trials, consensus documents and review articles on the diagnosis and therapy of Antiphospholipid Syndrome.

Pathogenesis: APS does not have a known etiology, like other autoimmune disorders. Recently proposed mechanisms are antibody-mediated interference with coagulation homeostasis, platelet activation, endothelial cell activation, placental tissue injury, T-cell immune response and complement activation [3,4]. The aPL antibodies causes thrombosis most likely by interfering with normal hemostasis by interaction with phospholipids or phospholipid-binding protein components such as $\hat{a}_2$ GP1(having anticoagulant properties),prostacycline, prothrombin, protein C, annexin V, and tissue factor. Intraplacental 'thrombosis' is commonly presumed to be the main cause of pregnancy failure and pregnancy complications in women with PAPS. But some authorities have focused on defective endovascular trophoblast invasion for both the occurrence of early pregnancy loss and later pregnancy complications in women affected by PAPS.[5]

Diagnostic Criteria for APS

According to the consensus-derived diagnostic criteria formulated by the Sapporo international workshop, APS is present in patients with 1 clinical and 1 laboratory criterion. Clinical criteria include objectively confirmed arterial, venous, or small-vessel thrombosis, or pregnancy morbidity consisting of recurrent fetal loss before the 10th week of gestation, 1 or more unexplained fetal death at or beyond the 10th week of gestation, or premature birth due to placental insufficiency, eclampsia,

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or preeclampsia. Laboratory criteria include medium or high titer IgG or IgM of aCL or the presence of LA on 2 or more occasions at least 6 weeks apart.[6,7]

Diagnostic criteria were updated in 2006 [1],where the clinical criteria remained unchanged; but, two important modifications in laboratory criteria were made: the time elapsed between two positive

determinations was extended to 12 weeks to assure the detection of persistent antibodies only; and anti-beta 2-glycoprotein 1, both IgG and IgM, were added to the laboratory criteria.

Clinical criteria

- Vascular thrombosis: one or more clinical episodes of arterial or venous thrombosis or thrombosis of small vessels of any organ or tissue, confirmed on Doppler or histopathology, vasculitis being excluded;
- Gestational morbidity:
  - One or more unexplained deaths of a morphologically normal fetus after the 10th gestational week, confirmed on ultrasound or by examining the fetus;
  - One or more premature births of a morphologically normal fetus before the 34th gestational week due to eclampsia, preeclampsia or causes of placental insufficiency;
  - Three or more unexplained spontaneous abortions before the 10th gestational week, with neither maternal hormonal nor anatomical abnormalities, paternal and maternal chromosomal causes excluded.

Laboratory criteria

- Presence of lupus anticoagulant antibody (LA) in the plasma on two or more occasions at a minimum 12-week interval, detected according to the recommendations of the International Society on Thrombosis and Hemostasis (ISTH);
- Moderate (> 40) to high (> 80) titers of IgG or IgM anticardiolipin antibodies (ACL) on two or more occasions at a minimum 12-week interval, detected by using standard ELISA test;
- IgG or IgM anti-beta 2-GPI antibodies in the plasma on two or more occasions at a minimum 12-week interval, detected by using standard ELISA test.

Anticoagulant therapy may interfere with the detection of LA[8] . Enzyme-linked immunosorbent assay tests for ACL are poorly standardized and aCL testing has shown poor concordance between laboratories [9]. The presence of more than one class of antiphospholipid antibodies increased thrombotic risk[10] .

The association between antiphospholipid antibodies and thrombosis is stronger with LA than with aCL. Both ACL and LA predict fetal loss. The association between antiphospholipid antibodies and fetal loss is strongest for loss occurring after 10 weeks[11]. Obstetric manifestations of APS are not restricted to fetal loss. [12,13,14]Early delivery, oligohydramnios, pre- eclampsia/ eclampsia and HELLP syndrome , arterial or venous thrombosis and placental insufficiency , neonatal complications (such as prematurity-estimated at 30-60% and more common in SLE patients, intrauterine growth restriction - IUGR , fetal distress [15] and rarely fetal or neonatal thrombosis)[16] are described.

Management of APS in Pregnancy:

The goals of treatment in pregnant women with antiphospholipid syndrome are to improve maternal and fetal/ neonatal outcomes by preventing morbidity, including , preeclampsia, placental insufficiency, fetal growth restriction, fetal loss and iatrogenic preterm birth [17].Also to reduce or eliminate aPL antibodies that induce the disease. With proper management, more than 70% of pregnant women with antiphospholipid syndrome will deliver a viable live infant[18] ,compared to 10% without treatment [19].

Prepregnancy:

Preconception counseling gives the physician the opportunity to understand the specific context of each patient with the syndrome and to outline the risks of pregnancy and treatment Pregnancy should be discouraged in all women with pulmonary hypertension because of the high risk of maternal mortality [20], and should be postponed in uncontrolled hypertension or recent thrombotic events eg, stroke[20] . A complete profile of antiphospholipid antibodies, including repeated anticardiolipin and lupus anticoagulant, should be available before pregnancy is planned. Patients should be counseled in all cases regarding symptoms of thrombosis and thromboembolism and should be educated regarding, and examined frequently for, the signs or symptoms of thrombosis or thromboembolism, severe preeclampsia, or decreased fetal movement.

Antenatal:

Frequent prenatal visits, at least every 2-4 weeks before mid-gestation and every 1-2 weeks thereafter is recommended. In patients with poor obstetric histories, evidence of preeclampsia, or evidence of fetal growth restriction, ultrasonography is recommended every 3-4 weeks starting at 18-20 weeks' gestation. The objectives of prenatal care in the second and third trimesters are close observation for maternal hypertension, proteinuria and other features of preeclampsia, frequent patient assessment, ultrasound to assess fetal growth and amniotic fluid volume, and appropriate fetal surveillance testing. Surveillance testing should begin at 32 weeks' gestation, or earlier if the clinical situation for placental in- sufficiency is suspected, and should continue at least every week until delivery. Regular and coordinated medical consultation every 2-4 weeks, especially in women with systemic lupus erythematosus, is recommended. Decreased fetal growth may reflect uteroplacental insufficiency in patients with APS . Colour doppler examination of early uteroplacental blood flow, may identify abnormal intervillous flow patterns prior to pregnancy failure and hence provide useful prognostic and therapeutic information[5]. Uterine and umbilical artery Doppler velocimetry can assess the risk for preeclampsia, placental insufficiency, and fetal growth restriction after the 20th week of gestation, and normal result have high negative predictive values .[21]

Antithrombotic management of patients with APS is mainly based on, antiaggregation (aspirin) or anti-coagulation (unfractionated or low molecular weight heparin) agents and immunomodulatory (prednisolone, intravenous immunoglobulins, plasma exchange. Most studies have shown that the best therapy of APAS is offered by aspirin (60 to 100 mg) and heparin of LMW (an injection a day in preventive dose). A meta-analysis [22] of randomized controlled trials examined the outcomes of various treatments –including aspirin, steroids, intravenous globulin and heparin–given to improve pregnancy outcome of women with recurrent miscarriage associated with antiphospholipid antibodies. This meta-analysis reported that the only treatment or treatment combination that leads to a significant increase in the live birth rate among women with antiphospholipid syndrome is aspirin plus unfractionated heparin. This combination therapy significantly reduced the foetal loss by 54%.[22] Aspirin should be started when she plans conception and low-molecular-weight heparin in prophylactic doses should be started when a viable intrauterine pregnancy is documented .The aspirin should be stopped by the 35th weeks of pregnancy, while the heparin is continued during 6 weeks after the childbirth. This treatment seems to be deprived of major maternal or foetal complication.

LMWH does not cross the placenta and is safe for both mother and fetus. Various clinical studies have demonstrated its function in improving live birth rates in patients with APS [23,24]. Initiation of heparin in the face of a failing pregnancy should be undertaken with caution due to risks of bleeding. The treatment by the heparin requires a weekly supervision of platelet especially during first weeks of the treatment.[25] The patient should be counseled regarding potential adverse effects of heparin. Heparin-induced osteoporosis occurs in 1-2% of cases. Bone density studies should be considered .This may be most important in women who have been treated in a previous pregnancy or are planning pregnancy.Calcium supplementation and weight bearing exercise is to be encouraged.

Treatment for APS with recurrent pregnancy loss with combination of high dose prednisone and low-dose aspirin, has successful outcome in 75% of treated pregnancies, but high maternal and fetal morbidity resulted, including gestational diabetes, hypertension, and premature rupture of membranes. A randomized controlled study showed low-dose subcutaneous heparin and low-dose aspirin to be equally efficacious with less morbidity in comparison with prednisone and aspirin.

Intravenous immunoglobulin (IVIg) contains anti-idiotypic antibodies, has an immunomodulatory action and may be used to treat autoimmune diseases. There are numerous case reports of successful pregnancy outcomes after treatment with IVIg .Some studies indicate that LMWH and IVIg may be more efficient [23,24]. A Cochrane analysis concluded that intravenous immunoglobulins were associated with an increased risk of pregnancy loss or premature birth, compared with heparin and low-dose aspirin.[22] Jing Xiong at el.[26] evaluated the effect of traditional treatment (prednisone and aspirin) and comprehensive treatment [prednisone, aspirin, low molecular weight heparin (LMWH) and IVIg] on the pregnancy outcome, obstetric complications and

fetal outcome in women with antiphospholipid syndrome (APS) and found that in traditional treatment group and comprehensive treatment group, the live birth rate was 83.91% and 97.62% (P<0.05), respectively, and the obstetric morbidity was 22.99% and 7.14% (P<0.05), respectively. The neonatal weight in the comprehensive treatment group was increased compared with the traditional treatment group (P<0.05), however, no differences were found in gestational age at delivery or preterm labor. Comprehensive treatment improved the result of gestation and reduced obstetric complications, and is a more effective treatment for APS than the traditional method of using prednisone and aspirin. The combination of heparin, particularly low molecular weight heparin (LMWH) and aspirin is considered superior to prednisone and aspirin, not because it achieves higher live birth rates, but because it causes less maternal morbidity .

Several potential new therapeutic approaches for APS are emerging .The only new drugs for APS that pregnant women can use are dipyridamole and hydroxy-chloroquine. Hydroxychloroquine is safe for the fetus and neonate [25] and the absence of associated bleeding, hydroxychloroquine can be considered for an adjuvant antithrombotic agent in patients with systemic lupus erythematosus who are positive for antiphospholipid antibodies.

Labour and delivery:

Labour and delivery of women with APS should be managed like a patient at high risk for preeclampsia and uteroplacental insufficiency.Pregnancy to be terminated near term,postdates to be avoided. Continuous electronic fetal monitoring is recommended during labour. Patients on heparin should withhold at the onset of labour.Heparin should be discontinued 24hours before planned induction of labour or caesarean section.

Postpartum:

Heparin and LMWHs are not secreted into breast milk and can be safely administered to nursing mothers. Warfarin does not induce an anticoagulant effect in the breast-fed infant when the drug is given to a nursing mother. Warfarin may be substituted for heparin during the postpartum period to limit further risk of heparin-induced osteoporosis and bone fracture. Therefore, women using this drug should be encouraged to breast feed. Oral contraceptives containing estrogen is absolutely contraindicated in women with APS.

Conclusion:

APS pregnancies are regarded as being at high risk for complications and should be treated and follow up by a team of doctors from different specialties. Medical treatment should be individualised, taking into account the obstetric history, presence or absence of a personal or family history of thromboembolic events, comorbidity, current drugs, and thrombotic risk factors, so that the miseries of pregnant women with APS can be minimized.

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CASE REPORTS

Gas in the Kidney (Emphysematous Pyelonephritis) often Misdiagnosed as Colonic Gas

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Summary:

Emphysematous pyelonephritis (EPN) is a serious and often life threatening infection of the renal and perirenal tissues. The characteristic feature of this infection is the presence of gas within the kidney and perinephric tissues. EPN usually occurs in diabetic women. CT Scan is the investigation of choice not only to establish the diagnosis but also to plan the line of management. Renal preservation must be the aim of management. This can be achieved to a great extent by medical management combined with percutaneous drainage of obstructed kidney. However nephrectomy is indicated in life threatening infection of the kidney. Prompt diagnosis and the timing of drainage could be the prognostic factor. A case of EPN in a diabetic patient who was successfully managed by nephrectomy is presented.

(J Banagladesh Coll Phys Surg 2015; 33: 91-94)

Introduction:

Emphysematous pyelonephritis (EPN) is defined as a severe, necrotizing renal parenchymal infection that is characterized by the bacterial production of gas within the kidney parenchyma. Schultz and Klorfein[1] first used the term 'EPN' in 1962, but the condition might have already been described by Kelly and MacCullem[1] as far back as the end of the previous century. EPN involves a spectrum of disease processes that result in the production of gas in the renal parenchyma; the gas can be focal or diffuse, and can spread to the collecting system or track into the perinephric and paranephric spaces. EPN can be classified into many ways and most of them are based on their CT findings. Middle-aged females with diabetes comprise the majority of patients with EPN[2].

Case Report:

A 40 years old female diabetic (insulin dependent) was admitted into Square Hospital Ltd (SHL) through emergency room (ER) to the medical ward with complaints of left sided abdominal pain, tightness of chest and decreased urine output for 7 days. There was no history of fever, cough or dysuria. She was admitted in a primary level hospital as a case of acute abdomen and treated conservatively for 3 days. As there was no improvement she was referred to a higher centre. On our medical ward she was treating as a patient of urosepsis with acute kidney injury. On the third day of admission we were called to see the incidentally diagnosed left renal stone. She had a plain X-ray kidney and urinary bladder (KUB) outside our hospital which was reported as normal except distended colonic gas shadow. By looking to the X-ray KUB with the diagnosis emphysematous Pyelonephritis (EPN) in mind we transferred her to urology department. Clinical evaluation revealed a co-operative but very ill patient. The temperature was 98 F, pulse rate 88 per minute, blood pressure was 120/70 mm Hg and the respiratory rate 18 per minute. Cardio-vascular and respiratory systems were within normal limits. Abdominal examination revealed severe tenderness at the left upper abdomen and the left renal angle. There was no mass palpable, and the remainder of the physical examination was normal.

Laboratory investigations on admission showed a haemoglobin of 10.6 gm/dl (normal range: 11.5 to 16.5 gm/L), total leukocyte count of 11.5 K/µL (normal 4.0 to 11 K/µL) with 88 % neutrophils (normal 40 to 75 %). The platelet count was 99 K/µL (normal 150 to 400). The the serum creatinine 2 mg/dl (normal range: 0.4 to 1.4 mg/dl)

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and blood urea was 116 mg/dl (normal range : 35 to 40 mg/dl). The random blood sugar was 15 mmol/L (normal range 3.90 to 6.10 mmol/L). The serum electrolyte was Sodium 130 mmol/L , potassium 4.8 mmol/L (normal range : Sodium 135 to 145 mmol/L and potassium 3.5 to 4.5 mmol/L). Urine microscopic examination revealed plenty pus cells, sugar 3+ with trace amount of blood and ketones. Urine and blood cultures were sent and both showed no growth, possibly due to taking of antibiotic prior to admission. Blood group was B positive. CPR was 236.9 mg/dl (normal range <5 mg/dl).

A plain X-ray of the abdomen revealed kidney shaped gas in the left renal area (Fig. 1). Computerised tomography of the KUB confirmed the presence of gas in the renal and perirenal area with extensive renal parenchymal destruction (Fig. 2a and 2b) and pus tracking towards the left iliac fossa. The diagnosis was type II EPN.

The patient was initially treated with meropenem 500 mg 8 hourly. An emergency PCN was performed which immediately drained 100ml pus. As the patients condition was not improving expectedly left nephrectomy was done with left subcostal incision on the next day. Post operative period was otherwise uneventful except one day stay in ICU for delayed extubation. She was discharged on the 8th POD with IV antibiotics for another 7 days. 6 months follow up was excellent.

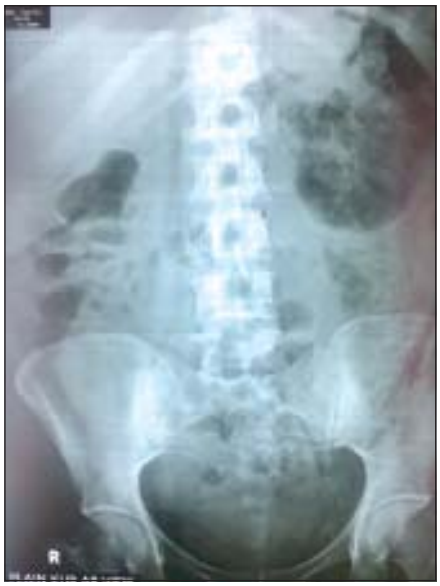

Fig.-1: *X-ray KUB showing kidney shaped gas in left renal area*

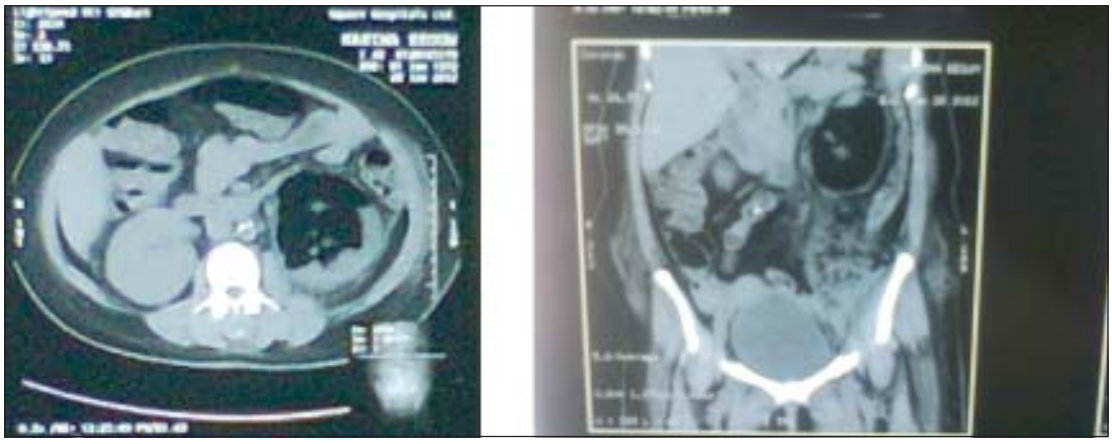

Fig.-2a and 2b: *showing gas in left kidney and perirenal tissue*

Discussion:

EPN is a rare renal infection. However due to modern imaging techniques and practice of routine ultrasound more cases are reported now a day. EPN predominantly affects females. The female to male ratio is 3:1. The left kidney is more frequently involved than the right (60% Vs 35 %). Both the kidneys are involved in about 5% of the reported series. 90% of the reported cases have occurred in diabetic patients. EPN has also been reported in debilitated (alcoholic) and immunocompromised patients.[4]

EPN can be classified into many ways and most of them are based on their CT findings. One of the classifications of EPN by Huang and Tseng[2] (based on CT) is Class I: Gas in collecting system only. Class II: Parenchymal gas only. Class IIIa: Extension of gas into perinephric space. Class IIIb: Extension of gas into pararenal space. Class IV: EPN in solitary kidney, or bilateral disease. Another simple classification of EPN by Wan et al[3](based on CT) Type I: Renal necrosis with presence of gas but no fluid. Type II: Parenchymal gas associated with fluid in renal parenchyma, perinephric space or collecting system.

The organisms commonly responsible for causing EPN are *Escherichia coli, Klebsiella pneumoniae, Proteus mirabilis, Pseudomonas aeruginosa, Citrobacter* and rarely yeast. If left untreated the condition is uniformly fatal.[4 5] The exact mechanism of gas formation in EPN is not known. Gas formation is believed to be due to pathogenic bacteria capable of mixed acid fermentation acting in a hyperglycaemic environment on tissues that are ischaemic. This results in tissue destruction, and encourages purulent infection and inhibition of the removal of locally produced gas[8]

The clinical presentation of emphysematous pyelonephritis was similar to that of upper urinary tract infection with fever, nausea and vomiting, lethargy, confusion, dyspnoea and shock. Laboratory data showed high glycosylate haemoglobin, leukocytosis, thrombocytopenia and pyuria.[3]

Diagnosis of EPN rests on the clinical awareness and confirming it by appropriate investigations. The triad of symptoms of fever, flank pain and pyuria especially in diabetic patients who do not respond promptly to antibiotic treatment must raise the possibility of EPN.[8] These patients require to be investigated and treated aggressively. The diagnosis of EPN is classically made by demonstrating gas in the renal or peri-renal tissue by plain abdominal X-ray. However gas can be demonstrated only in 33% of plain abdominal radiographs in patients with EPN.[6] Even by abdominal ultrasonography it may be technically difficult to distinguish the renal gas filled area from gas in the bowel. On the other hand, CT scan can not only confirm the diagnosis, but also show the extent of the disease. Abdominal CT scan is recommended for all patients in whom EPN is suspected.

Several studies have been done to correlate clinical features of EPN with the treatment outcome.[7]These showed that age, sex, site of infection, blood urea nitrogen level and blood glucose level were not prognostic factors. But patients initially seen with thrombocytopenia, acute renal function impairment, disturbance of consciousness and shock were associated with very high mortality.[8]

Management of patients with emphysematous pyelonephritis has been a subject of controversy. Huang and Tseng reviewed the management of 48 patients with emphysematous pyelonephritis. They concluded that for localized emphysematous pyelonephritis (class I and II) according to CT scan, percutaneous drainage with antibiotic treatment can provide a good outcome. For extensive emphysematous pyelonephritis (class III and IV) with more benign manifestations, when saving the kidney is possible, percutaneous drainage combined with antibiotic treatment may be attempted because of its high success rate. However, nephrectomy can provide the best management outcome and should promptly be attempted for extensive emphysematous pyelonephritis with a fulminant course.[3] The rapidly deteriorating general condition of the patient and the onset of septicaemic shock prompted us to go ahead with nephrectomy rather than adopt a more conservative line of management.

Conclusion:

EPN is a severe and often life threatening infection. CT Scan is the investigation of choice for not only making a proper diagnosis but also in planning the treatment option. Renal preservation must be the aim of treatment, but this must not be at the cost of patient's life. One should not hesitate to resort to nephrectomy as and when indicated.

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Acrorenal Mandibular Syndrome: A Case Report

BS NAHER

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Introduction:

Acral (limb) and renal anomalies occur together as a single developmental field defect (DFC)[1] or as components of several malformation syndromes. The embryogenesis of that defect was studied experimentally by Lash[2] and the developmental relationship between kidneys and limbs has been reviewed by Buchta[3] and Gilbert[4]. In 1980, Halal et al. first described the association of bilateral split foot malformation, severe mandibular hypoplasia and bilateral renal malformation in female sibs born to consanguineous French- Canadian parents. Bilateral renal agenesis was found in one sib and bilateral polycystic kidneys in the other[5].These authors proposed the name acro-renal-mandibular syndrome (ARMS) for the condition and considered it most likely that the mechanism of inheritance was autosomal recessive.

The acral anomalies consist of split hand/ split foot, varying combination of oligodactyly, ectrodactyly, syndactyly, brachydactyly, polydactyly or fusion of carpal, tarsal, metatarsal bones of hands and feet. Urinary tract dysplasias are renal agenesis, duplication, ureteral hypoplasia, polycystic kidneys, hydronephrosis and bladder neck obstruction.

The purpose of this paper to report a newborn with acral (polydactyly) and renal malformation (polycystic kidney) rather than split hands/ feet (acral) malformation, the most typical digital malformation in acrorenal mandibular syndrome as much is not known about the occurrence of such case in Bangladesh.

Case Report:

A 10 day old male baby, 1st issue of consanguineous parents hailing from Keranigang, Dhaka was admitted into Sir Salimullah Medical College and Mitford Hospital on 2nd November, 2013 with the complaints of inability to suckle breast properly and limitation in upper limb movement since birth. Mother Farzana 20 year old nondiabetic, normotensive, nonasthmatic had good antenatal health. She received one dose of tetanus toxoid during her antenatal check up. There was no history of taking any drug other than vitamins and folic acid in pregnancy. She did not suffer from fever or rash at any time of gestational period. Her pregnancy was uneventful up to 39 weeks of gestation. Then she delivered a male baby per vaginally in the hospital. Baby was mildly asphyxiated at birth. There was no history of prelacteal feeding. Baby was put to breast within one hour of birth but he was not able to feed properly and mother noticed that the lower part of the face was depressed (Fig. 1). On examination, the baby was pink in colour, weight was 2.3 kg, length 43 cm and occipitofrontal circumference was 32 cm. All vital parameters were normal. High arched palate and polydactyly was present in both upper and lower extremities (Fig. 2 and 3). Extension of elbow joints was restricted on both sides. Air entry was good on both sides of the chest. Heart sounds were normal. Abdomen was soft, liver and spleen were normal in size but the left kidney was bimanually palpable and ballotable. Spine was normal.

Fig.-1: *Showing right sided mandibular hypoplasia*

On investigation, Haemoglobin was 19 gm/dl, TC of WBC was13000/cmm, platelet count was 380,000/cmm. Differential count of WBC and CRP was normal. Blood

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Fig.-2: *Showing polydactyly of hands*

Fig.-3: *Showing bilateral polydactyly of feet*

culture showed no growth of any organism. Doppler echocardiography was also normal. Ultrasonography of brain was normal but whole abdomen revealed bilateral echogenic kidneys with multiple tiny cysts in both kidneys (Fig. 4). Serum creatinine was 3 mg/dl but

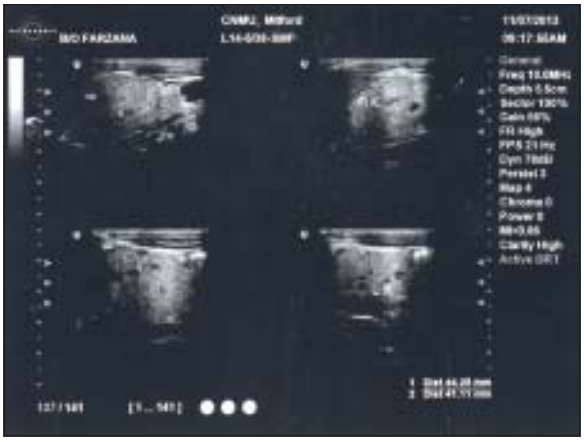

Fig.-4: *Bilateral echogenic kidneys with multiple tiny cysts in both kidneysReferences:*

electrolyte was within normal level. Skeletal survey was done and was unremarkable. So on the basis of hypoplastic mandible, high arched palate, polydactyly, cystic kidneys, flexion contractures of upper limb she was diagnosed as acrorenal mandibular syndrome. We managed the baby by helping her in breast feeding in proper technique. We consulted with paediatric nephrologists and followed their advice. We also consulted with the paediatric surgeon regarding polydactyly and according to their suggestion we advised her to come for follow up visit in paediatric surgery out patient department later on. We counseled the parents regarding the risk of recurrence which is 25% and offered prenatal diagnosis using ultrasonographic examination.

Discussion:

The association between skeletal and renal anomalies is well known. This association is seen in a very heterogeneous group of acrorenal syndrome[6].The ARM syndrome is also known as acrorenal-uterine-mandibular syndrome or split hand and split foot syndrome with mandibular hypoplasia.

The pathogenesis has been linked to an abnormal epithelial-mesenchymal interaction during embryonic development [7]. Such interaction is obviously crucial to the formation of the apical-ectodermal ridge and subsequent limb development and to the development of ureteric bud. This is also involved in the morphogenesis of the palate, jaw, skin derivatives, the mammary glands, the paramesonephric ducts and the eye. Thus the major anomalies found in this patient appear to share a pathogenetic relationship.

Although the most typical limb deficiencies (LD) for the acrorenal syndrome is split hand/ split foot[8]; Kroes et al.[6] did not support this suggestion as several digital malformation like polydactyly, syndactyly, ectrodactyly other than split hand/foot are also observed in cases of acrorenal syndrome indicating clinical/ phenotypic variability in their reports.

Our case report suggests that bilateral symmetrical polydactyly of both hands and feet is a part of ARUMS/ ARMS. This case is born to consanguineous parents. So the mechanism of inheritance is thought most likely to be autosomal recessive as is seen in the reports by Kroes [6].

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Citrullinemia Type I - A Case Report

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Summary:

Citrullinemia type I (CTLN1) is an inherited urea cycle disorder where the enzyme argininosuccinate synthetase is deficient. It can lead to recurrent hyperammonemic crisis that may result in permanent neurological sequelae, even death. Vomiting in patients with urea cycle disorders may either be the result or cause of acute hyperammonemia, particularly if due to an illness that leads to catabolism. Therefore, age-appropriate common etiologies of vomiting must be considered when evaluating these patients. We present a case of a 2 year 5 month old female child with CTLN1 who had a history of frequent vomiting after the age of one year and some recent neurological manifestations like excessive crying and lethargy and one episode of unconsciousness. Investigations revealed high level of ammonia. Amino acid profile using tandem mass spectrometry showed markedly increased plasma level of citrulline. After administration of sodium benzoate and protein restricted diet there was dramatic improvement of all the symptoms.

(J Banagladesh Coll Phys Surg 2015; 33: 98-100)

Introduction:

Citrullinemia (CTLN), a rare autosomal recessive disorder, is characterized by the accumulation of citrulline and hyperammonemia caused by a deficiency in argininosuccinate synthetase (AS), the third enzyme in the urea cycle that catalyzes the formation of argininosuccinate from citrulline and aspartate.[1] Two clinically and genetically distinct form of citrullinemia has been identified. The classic form (type I, CTLN 1) is due to deficiency of AS enzyme which has a severe or neonatal form and a subacute or mild form. Citrullinemia type II (CTLN2) is due to deficiency of mitochondrial transport protein named citrin. Citrullinemia due to citrin deficiency (CTLN2) is caused by mutations in chromosome 7q21.3.[2] Citrin deficiency leads to a failure to shuttle aspartate and glutamate to and from the mitochondrion, leading to a mild hyperammonemia .[3] CTLN2 is characterized by a less pronounced elevation of plasma citrulline. It has neonatal form with neonatal intrahepatic cholestasis and adult-onset form. Here we present a case of a 2 year 5 month old female child with CTLN1 who had a history of frequent vomiting after the age of one year and some recent neurological manifestations like excessive crying and lethargy and one episode of unconsciousness. She was referred to BIRDEM hospital as a case of suspected diabetic ketoacidosis (DKA)

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Case Report:

A 2 year 5 month old female child, 3rd issue of non consanguineous parents was referred to BIRDEM hospital as a suspected case of diabetic ketoacidosis (DKA). She had recurrent, nonprojectile vomiting since one year of age and was treated with anti emetic and antibiotic on several occasions. For last few weeks patient had developed increased vomiting, lethargy and excessive crying for which she was admitted in a local hospital. There she became unconscious after taking sedative on an attempt to do a CT scan of brain. She regained consciousness after one day but became more lethargic. On investigation she had thrombocytosis (platelet count 6,29,000/cu mm),hyperglycemia (blood glucose 12.1 mmol/L), raised SGPT(101mU/L) and metabolic acidosis. Urine routine examination showed pus cell and culture yielded growth of E Coli. She was treated there with intravenous fluid, anti emetic and

parenteral antibiotic initially as a case of urinary tract infection (UTI) but as her condition deteriorated and diabetic ketoacidosis (DKA) was suspected, she was referred to BIRDEM hospital for further management. There was no history of fever, convulsion and trauma. Her birth history was uneventful. She was on exclusive breast feeding upto 7 months of age and then complimentary feeding with khichury was started. Her bowel and bladder habit was normal. There was history of one sib death at 11 months of age due to fever, convulsion with excessive crying. On admission in BIRDEM hospital, she was found conscious, lethargic, afebrile, mildly pale, anicteric and non dehydrated. There was no facial dysmorphism. Vital parameters were normal. Eye and ear, nose, throat examinations were normal. Signs of meningeal irritation were absent. Bed side capillary blood glucose was 7.7 mmol/L (normal <7.8 mmol/L), urine for glucose and ketone were absent. Anthropometric measurements were normal for her age and sex. She had mild developmental delay. Her muscle tone was reduced, muscle bulk and power were normal. All reflexes and sensation were intact, gait was normal. She had non tender hepatomegaly, 3 cm from subcostal margin in midclavicular line. All other systems revealed normal findings. Investigations revealed: leucocytosis (Total WBC count 17,600 /cu mm), thrombocytosis (platelet count 5,79,000/cu mm), raised level of serum transaminases (ALT 316 U/L, AST 288 U/L), with normal bilirubin, alkaline phosphatase, prothombin time and serum albumin, normal lipid profile and normal level of serum creatinine. Her fasting, random blood glucose and HbA1c were normal thus excluding diabetes mellitus. There was respiratory alkalosis with normal anion gap. Her serum ammonia level was 307ìmol/L (normal range 9-30 ìmol/L), blood urea level was 16 mg/dl (normal range 10-50mg/dl). The citrulline level on amino acid profile using tandem mass spectrometry (TMS) was 908.76µM (normal range 4-45 µM), which was very high. Urine routine examination was normal and culture yielded no growth. Ultrasonogram of whole abdomen showed increased hepatic parenchymal echogenicity. Initially we thought of sepsis with some inborn error of metabolism and after getting all the reports the patient was finally diagnosed as a case of citrullinemia typeI. Patient was initially managed by intravenous fluid, Anti emetic, parenteral antibiotic was continued. After getting the biocemichal reports, protein restricted diet, sodium benzoate powder with food and carnitine supplementation was advised. Consultation from paediatric neurologist was also taken. Parents were counseled about the disease process, its prognosis and management. Genetic counseling was also done. Parents were advised to come for regular follow up. After one month, there was significant improvement in patient's general well being. The vomiting had stopped. Patient was alert, well and playful. Serum ammonia level came down to 68 ìmol/L (normal range 9-30 ìmol/L) and liver enzymes were normal (ALT 40 U/L and AST 37 U/L).

Discussion:

Citrullinemia, an autosomal recessive disorder, occurs in 1:57,000 births[4] and causes a dramatic elevation of plasma citrulline Following the recent report by Kobayashi et al, who identified the citrin gene responsible for adult-onset type II CTLN. CTLN is now classified as CTLN1 and CTLN2. Citrullinemia type I is caused by a mutation in the AS gene located on chromosome 9q34.[5] Most patients with classical CTLN1 present with symptoms during the early neonatal period with acute hyperammonemia and life threatening encephalopathy. Seizures progressing to coma and death are typical in untreated patients.[6] The outcome is poor with significant risk of neurological damage or demise. In the subacute or mild form, clinical findings such as failure to thrive, frequent vomiting, developmental delay and dry, brittle hair appear gradually after one year of age. Acute hyperammonemia, triggered by intercurrent catabolic state, may bring the diagnosis to light as occurred in our patient.[5] There are also reports of women with onset of severe symptoms during pregnancy or in the postpartum period.[7] Individuals remaining asymptomatic up to at least ten years of age have been reported; it seems possible that they may remain asymptomatic throughout life.[8,9] In CTLN1 the plasma level of citrulline is markedly elevated, usually 50-100 times normal. Urinary excretion of orotic acid is moderately increased. The diagnosis is further confirmed by enzyme assay in cultured fibroblasts or by DNA analysis.[5,10,11] Newborn screening by tandem mass spectrometry (TMS) using a dried blood spot can detect elevated level of citrulline. Prenatal diagnosis is possible with the assay of the enzyme activity in the cultured amniotic cells or by DNA analysis of chorionic villi biopsy.[4,5] Treatment of acute hyperammonemia in an infant includes provision of adequate calorie, fluid

and electrolyte intravenously, adding minimal amounts of protein preferably as a mixture of essential amino acids, giving priming dose and then continuing infusion of sodium benzoate, sodium phynylacetate, arginine hydrocholoride, peritoneal dialysis or hemodialysis may also be needed in severe case.[12,13]

Our patient had the mild form of CTLN1 and was successfully managed by protein restricted diet, oral sodium benzoate and carnitine supplementation. Sodium benzoate is given to conjugate glycine, a major amino acid that contributes ammonia to the urea cycle, forming hippurate, which is subsequently excreted in the urine. Carnitine supplementation is recommended because benzoate may cause carnitine deficiency.[5] She initially had UTI and sepsis which along with chronic cerebral edema might be responsible for exacerbation of her symptoms. Our patient had respiratory alkalosis which strongly suggests a urea cycle defect. It is the result of hyperventilation due to stimulation of the central respiratory drive by hyperammonemia.[14] The initial hyperglycaemia with metabolic acidosis could be explained by UTI and the stress of acute infection. Ammonia can cause swelling of hepatic mitochondria, leading to increase in serum transaminase, as found in our patient. Our patient had the mild form of CTN1, therefore the prognosis is better than that of symptomatic neonates. These patients usually do well if treated properly.[5] Catabolic states (infection, fasting) should be avoided or treated vigorously. They need close monitoring of growth, development and nutritional indices (blood albumin, pH electrolytes, amino acids, zinc). Long term care of these patients is best achieved by a team of experienced professionals (physician specialist, nutritionist, neurologist and geneticist).

In conclusion, we describe a case of CTLN1 which can lead to recurrent hyperammonemic crisis that may result in permanent neurological sequelae or even death. Therefore, this case report shows the importance of biochemical and metabolic investigations, to reach an early and definitive diagnosis and proper management of such cases.

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Young Lady with Acute Upper Limb Ischemia due to Cervical Band

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Summary:

Introduction: Acute upper limb ischemia is usually caused by compression of cervical rib on subclavian artery. But in very few cases compression may be caused by cervical band which is not evident in conventional radiography.

Results and Discussion: This is a case of a 26 year old lady, housewife, having rudimentary cervical rib, presented with acute left upper limb ischemia due to subclavian artery aneurysm. Peroperatively cervical fibrotic bandwas identified and diagnosed as the cause of subclavian artery aneurysm instead of rudimentary cervical rib. Scalenectomy, excision of the cervical band, excision of the aneurysmsac and arterial reconstruction of the subclavian artery was performed. Her operation was uneventful, postoperative recovery was excellent. She was discharged to home on 4th postoperative day ensuring good distal pulsation of affected limb.

Conclusion: Though rare, cervical band is a cause of acute upper limb ischemia. So, we must not forget while diagnosing this type of cases.

Key Words: Cervical fibrotic band,Cervical rib,Subclavian artery aneurysm

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Introduction:

Compression over the subclavian artery causes thoracic outlet syndrome. Arterial thoracic outlet syndrome may cause acute upper limb ischemia. It is rare, but in most of the cases compression is caused by cervical rib and is associated with subclavian artery aneurysm. In few cases, compression over the subclavian artery may occur by fibrous bands that cause acute upper limb ischemia[1]. Cervical band is usually not visible inconventional radiography and mostly diagnosed peroperatively. In this case report we present a young woman with rudimentary cervical rib who suddenly developed acute upper limb ischemia due to cervical band.

Case Report:

A 23 year old woman, normotensive, nondiabetic housewife, presented with pain and tingling in left upper limb for 3 months and blackening in distal part of left hand for 2 months. Pain at first started at pulp of the left hand, used to persist for 1-2 days and subsided by taking medicine. Gradually pain had spread up to palm, then proximally towards forearm and arm. Blackening began at tip of the fingers and was limited there. She also noticed sensory impairment at distal hand and fingers. She gave no history of smoking or taking oral contraceptive pills. She used to do normal household works.

On examination, her pulse was 80beats/min, regular with normal volume andblood pressure was 110/70 mm of Hg. On examination of left upper limb,there was no gross muscle wasting. Colour of skin of left upper limb was normal except the tip of fingers which were black. Local temperature of the left forearm and hand was cooler than that of right. But temperature of arm was normal. Bulk and power of muscles of left upper limb was normal. Though motor function was intact, there was diminished sensation of left fingers. Pulsation was felt over left supraclavicular fossa. Left axillary pulse was palpable with normal volume and quality. Left brachial, radial and

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ulnar pulses were not palpable. Bruit was heard over left supraclavicular fossa. Other systemic examination revealed normal findings. Her hemoglobin was 12.6 gm/dl, ESR was 26 mm in 1st hour, C-reactiveprotein was 1.6 mg/dl. ELISA test for ANA was negative. Duplex study of the left upper limb revealed arterial thromboses involving distal end of the axillary, whole length of brachial, ulnar and radial arteries up to the level of wrist.X-Ray Cervical Spine (B/V) reported left sided rudimentary cervical rib (Figure 1). CT Aortogram with left upper limb angiogram was done (Figure 2). Findings were, ascending aorta and arch of the aorta appeared normal with normal branching pattern. Focal aneurysmal dilatation of 2nd part of the left subclavian artery for a length of about 20 mm and diameter was about 10.2 mm. Thrombus within left axillary and brachial artery having faint flow in forearm arteries. MRI of neck reported no cervical rib or band. Since admission patient received medical management including Clopidogrel-Aspirin combination and Warferin. As there was diagnostic dilemma and clinical condition of the patient was not improving, surgery was planned to explore the cause and for arterial reconstruction of thesubclavian artery aneurysm.

The patient was in supine position. After she was under general anaesthesia, a sand bag was placed under her left shoulder to expose the operation field properly. Two fingers above the left clavicle a supraclavicular incision was made from lateral border of the starnoclidomastoid muscle anteriorly upto anterior border of the trapezius muscle posteriorly. The scalene fat pad was mobilized and the anterior scalene muscle was excised preserving the phrenic nerve. The left subclavian artery aneurysm was identified and exposed.Proximal and distal control of the subclavian artery was taken and the artery was retracted downwards. Systemic heparinization was done. A whitish, glistening fibrous band was identified (Figure 3).The band was dissected. After doing the subclavian arteriotomy,Fogarty embolectomywas done upto the mid arm. Good distal flow was found. Excision of the aneurysm sac was done. Arteriotomywas closed with 6/0 prolene. Haemostasiswas ensured and wound was closed in layers keeping a drain in situ. Early postoperative period was smooth and uneventful. Heparin was continued as 2500 units subcutaneously 8 hourly. On 1st post-operative day, the drain was removed. Distal limb was warm and blackening of the finger tips found diminishing. On 2nd post-operative day both radial and ulnar pulses were palpable with good quality. On 4th post-operative day, patient was discharged from hospital with advices of cutting the stiches at surgery department of local hospital after 4 days, to take Clopidogrel-Aspirin combination daily for at least 3 months and to come for follow up at vascular surgery outpatient department after 1 month.

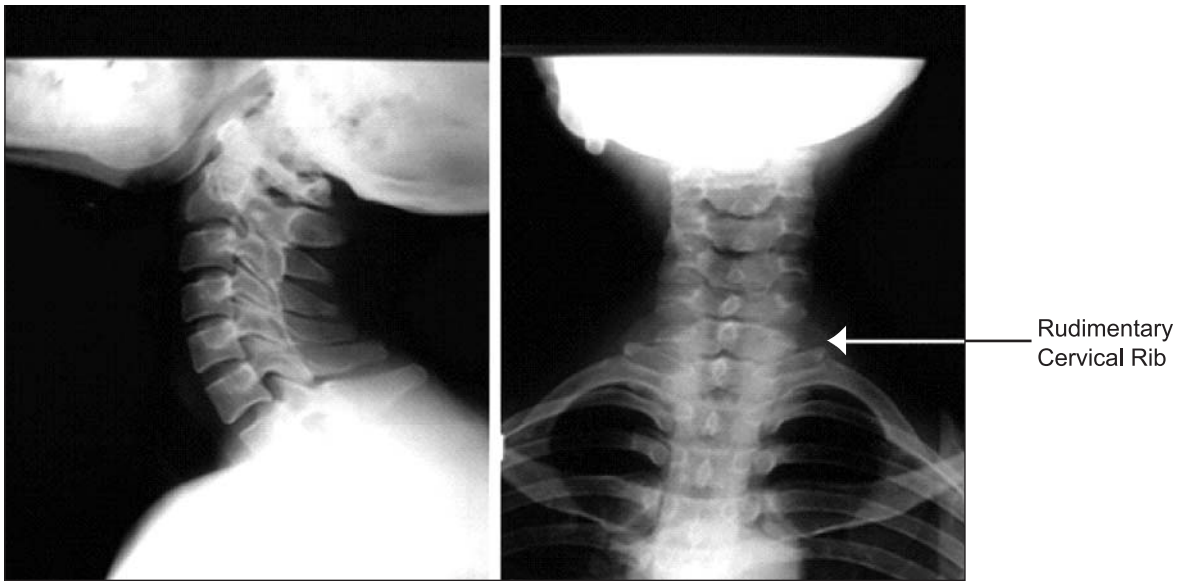

Fig.-1: *X-ray cervical spines of the patient showing left sided rudimentary cervical rib.*

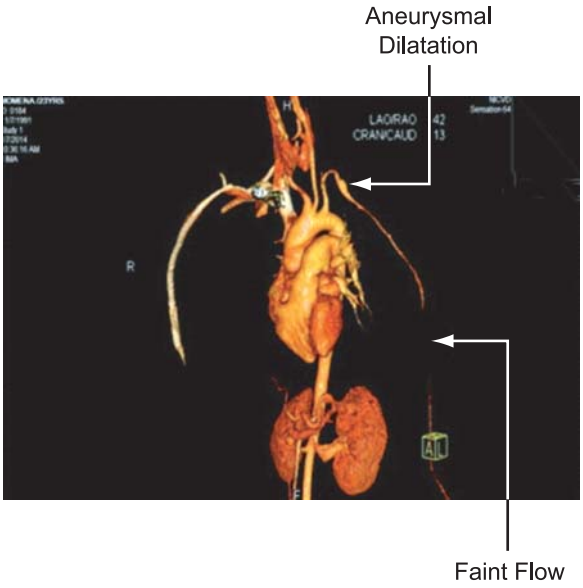

Fig.- 2: *CT angiogram of the patient reported focal aneurysmal dilatation of 2nd part of the left subclavian artery. Thrombus within the left axillary and brachial artery having faint flow in forearm arteries.*

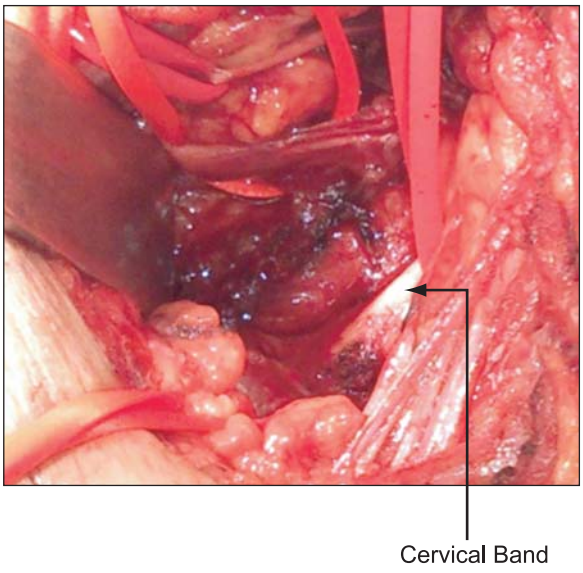

Fig.- 3: *Per-operative finding of the patient was presence of cervical band.*

Discussion:

Arterial complications following the subclavian artery compression represent a type of Thoracic Outlet Syndrome (TOS). The term Thoracic Outlet Syndrome (TOS) was first introduced by Peet et al. in 1956[2]. Manifestations of arterial TOS represent a progressive course characterized by extrinsic compression, post-stenotic dilatation, aneurysmal degeneration and secondary embolization[3]. Among the course of neurovascular structures from the base of the neck into the arm via the axilla, the most important narrow passageway is the interscalene triangle. Its boundaries are the anterior scalene muscle anteriorly, the middle scalene muscle posteriorly and the medial surface of the first rib inferiorlyt .

In most of the cases arterial complications of TOS is caused by cervical ribs in the interscalene triangle. Less common causes include anomalous first ribs, fibrocartilaginous bands associated with the anterior scalene muscle and hypertrophic callus from healed cervical fractures[1].This push the subclavian artery forward, where it is compressed between the first rib and the anterior scalene muscle. This compression causes injury to the inferior aspect of the third segment of the subclavian artery which may lead to localized intimal damage or post-stenotic dilatation. This dilatation may progress to aneurysmal change and localized intimal damage may lead to embolization or thrombosis. In our case, aneurysmal dilatation of subclavian artery was due to above mentioned mechanism.

Among the anatomic abnormalities causing arterial TOS, cervical rib comprises 63%, anomalous 1st rib 22%, fibrocartilagenous band 10%, clavicular fracture 4% and enlarged C7 transverse process 1%[3].Sanders and Haug have reported that up to 12% of patients with arterial TOS have fibrous bands without any definable arterial abnormalityu .In this case, it was also for the fibrous band like Sanders and Haug's finding. Most patients with symptomatic arterial TOS are young with mean age 37 years, with equal proportion of male and female[3].Our patient was 36 years aged young lady. Fibrocartilagenous bands may occur due to fibrosis of the muscles. Common bands are middle scalene muscle band, outlet band and anterior scalene muscle band. In the complete form cervical rib articulates with the manubrium or the first rib. In some cases a relatively short bony cervical rib can be extended by fibrous bands which are not visible by conventional radiography but can cause pathology identical to that of a complete bony cervical ribv . It may be diagnosed per-operatively by

direct observation and feeling of the band. Our case was also diagnosed during operation.

The first successful treatment of a subclavian artery aneurysm was achieved in 1864 by Smyth in New Orleans, who ligated the right common carotid artery and the innominate arteryw .The three main components of treatment include relieving the arterial compression, removing the source of embolus and restoring the distal circulationx .Our surgical treatment was like Smith and Valentine. Relieving the arterial compression involves routine division of anterior scalene muscle and excision of cervical rib or fibrous band.Resecting a subclavian aneurysm or repairing an arterial stenosis with intimal damage removes the source of embolus. Restoring the distal circulation involve thrombolysis, thromboembolectomy or bypass.

Conclusion:

While diagnosing a case of acute upper limb ischemia, one must not forget about fibrous band as a cause of compression over subclavian artery along with cervical rib.As fibrosis is usually not evident by conventional radiography, diagnosis may be difficult. This is a rare conditionbut more common in younger age. Person with cervical rib or cervical band fibrosis, doing physical exercise or carrying weight, are at increased risk of developing arterial thoracic outlet syndrome. Early diagnosis and management is essential to save the limb from necrosis following acute limb ischemia.

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(J Banagladesh Coll Phys Surg 2015; 33: 105-106)

Detection of Omental Recurrence of Colorectal Cancer by FDG-PET/CT

MM RAHMAN

A 48 year old lady underwent anterior resection of rectosigmoid colon for a tumour in the upper rectum in September 2011.High ligation of the inferior mesenteric artery and total mesorectal excision done during the operative procedure. Histopathology report revealed moderately differentiated adenocarcinoma. All ends of the colon were tumour free. There was no evidence of lymph node involvement. She received six cycle chemotherapy containing Oxaliplatin and capacetabine afterwards. She was running well, enjoying a healthy family life after the treatment. Dietary and bowel habit was normal. She was under strict follow up with S CEA level, USG examination of the abdomen and colonoscopy every six months after initial treatment. All were within normal limit. In January 2015, she developed lower abdominal pain, but bowel habit was normal, serum CEA level was 50 ng/ml, colonoscopy, ultrasonography was normal, only a lump found adhered with parietal wall which was detected in CT scan. But PET/CT examination revealed multiple high FDG uptake suggesting recurrences in the abdominal cavity in the area of lower abdomen, not related with gut in addition to the parietal lump which was earlier detected by CT scan. After laparotomy, along with a lump adhered to parietal wall on the right side, there was multiple seedlings on the omentum matching with the PET/CT findings . Excision of the lump along with omentectomy done. Histopathological analysis yielded a diagnosis of metastatic adenocarcinoma from the omental lesion.

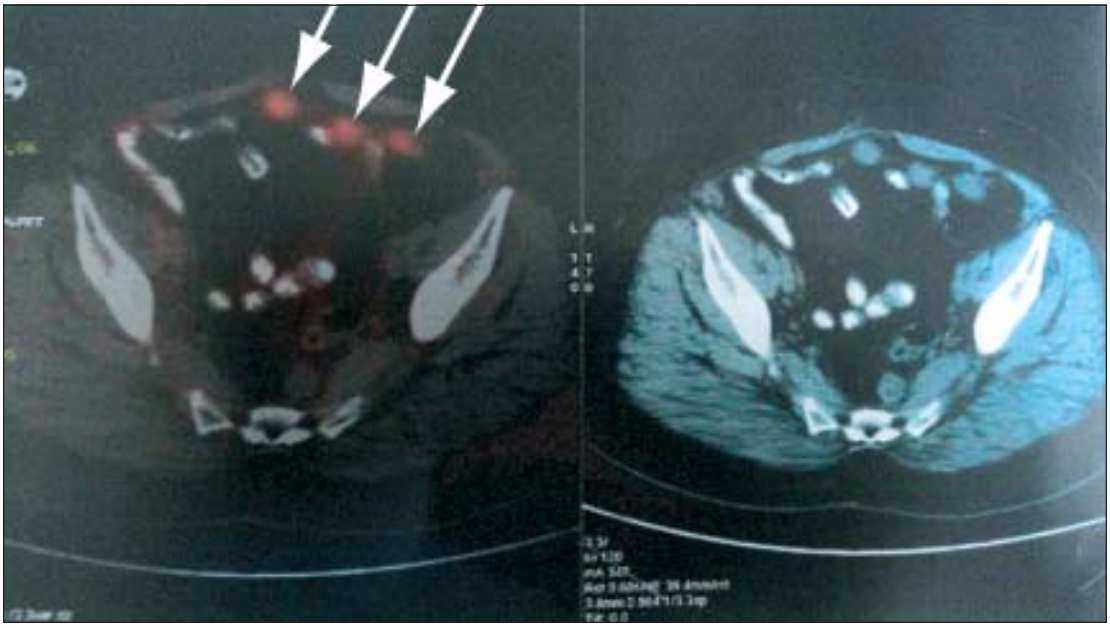

Fig.-1: *Marked areas show increased FDG uptake in a PET/CT Scan suggesting metastatic deposits*

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Recurrence develops within 3 years after radical surgery for co-lon cancer in 83.6% of recurrent colon cancer cases and is very rarely detected after 5 years (3.6% of cases)[1]. Therefore, regular follow-up with various diagnostic modalities until 5 years after surgery is a reasonable strategy to detect recurrent disease. The commonly used imaging modalities in such cases include ultrasonography, computed tomography (CT), and mag-netic resonance imaging (MRI). However, ^{18}F-fluorodeoxyglucose glucose-positron emission tomography (FDG-PET) is more ef-fective in determining the presence of malignancy, especially in cases of colorectal cancer (CRC)[2].

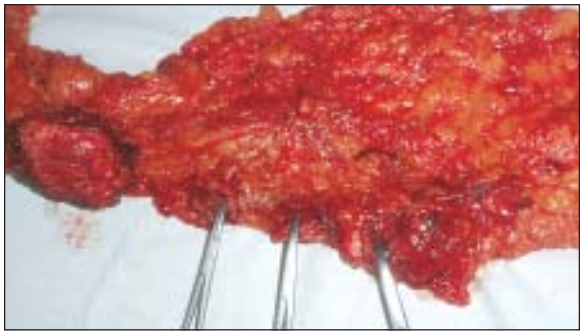

Fig.-2: *Post operative view of greater omentun shows three metastatic seedlings marked by artery forceps*

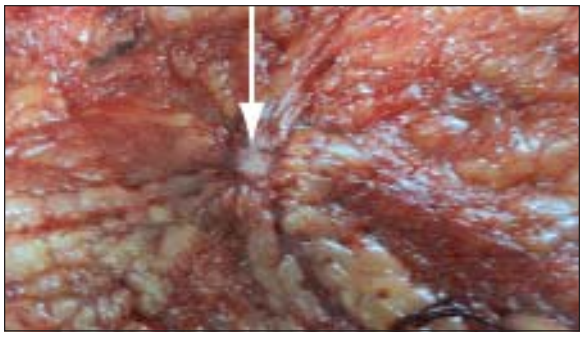

Fig.-3: *Close view of the metastatic seedling in the omentum-marked by the arrow.*

Locoregional pelvic recurrence and liver metastases are the major sites of relapse after resection of CRC. Recurrence occurs in one third of patients in the first 2 years after resection. Patterns of recurrence are different in colon cancer compared with rectal cancer. Local recurrence is more common in patients with rectal rather than colon cancer ranging from 7% to 33% and 1–19%, respectively[3],

Detection of recurrent lesions usually involves serum tumor marker level measurement, ultrasonography, CT, and MRI. In addition, the efficacy of FDG-PET has been confirmed[4]. Luboldt et al. reported that FDG-PET/CT provided promising accuracy for colorectal mass detection and that, in all carci-nomas and adenomas with high-grade dysplasia[5]. A review by Visioni and Kim stated that the sen-sitivity and specificity of PET-CT in detecting CRC recurrence were 89–95% and 83–92%, respectively[6]. One of the stud-ies evaluated in that review[7] reported a positive predic-tive value of 96.4% and a negative predictive value of 76.9% for the diagnosis of CRC recurrence by PET-CT. So PET/CT is a useful tool for the diagnosis of recurrence of colorectal cancer even in a smaller size within omentum and peritoneum. which are missed in traditional CT scan examination.. After that appropriate measures like radical surgery improves the total survival of the patient.

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LETTER TO THE EDITOR

(J Banagladesh Coll Phys Surg 2015; 33: 107-108)

To

Editor in Chief

Journal of Bangladesh College of Physicians and Surgeons

Sir,

I would like to thank you for publishing the article "Endoscopic Ultrasound: A New Hope for Patients" in Journal of Bangladesh College of Physicians and Surgeons 2015 ; 33: 23-31 . I have read the article thoroughly and discovered that the article had reviewed some important new information on standard applications of EUS that should be adopted into our clinical practice. The article is knowledgeable and is a reflection of the current trend of application of EUS, with most of the emphasis on EUS-FNA and EUS-guided interventions. The article also provides a glimpse into the future through EUS-guided new technology, like in certain treatments, e.g. draining pancreatic pseudocysts, biliary access, intramural therapy.

Although EUS has enhanced our ability to diagnose treat a variety of GI conditions, there are many queries regarding the ability of EUS. I would like to highlight a few of them in short:

(1) EUS was first introduced into clinical practice in the 1980s and has rapidly evolved into a reliable technique for diagnosis of lesions of digestive tracts. But at present one of the most common indications of EUS is esophageal cancer staging. Recently some literature has raised the question of accuracy of EUS in staging of early esophageal cancer. Prognostic and therapeutic decisions in esophageal cancer hinge on accurate tumor staging[1,2,3]. Upon evaluation of 12 studies, recent literature revealed that EUS correctly predicted the T-stage with only 65% accuracy. It concluded that pre-treatment EUS for intramucosal esophageal adenocarcinoma is unnecessary, might, in fact, be misleading.

(2) Gastric cancer staging with EUS: Does it help determine who needs neoadjuvant therapy and is it better than CT imaging? The purpose of clinical staging gastric cancer is to determine which patients have locoregional ?resectable disease versus systemic involvement. The only accepted criteria for unresectable gastric cancer are the presence of distant metastasis or invasion of major vessels such as the aorta or celiac axis (including hepatic, proximal splenic arteries)[4,5]. So for both the esophageal and gastric cancer, an "outside-in" approach is recommended. Cross-sectional imaging such as CT is a useful first step to rule out distant/ metastatic disease.

(3) Does the stylet aid or hinder the EUS-assisted fine needle aspiration?

(4) Does needle size matter in endoscopic ultrasound-fine needle aspiration of solid lesions?

(5) Contrast-enhanced harmonic EUS: is it going to be more potential in application for diagnosis of malignant potential of gastrointestinal stromal tumors? (6)

I, again thank the author for sharing such wonderful and up-to-date information with us.

Dr. Ahmedul Kabir
Associate Professor, Dept. of Medicine, Dhaka Medical College, Dhaka

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Author's Reply

To
Editor in Chief
Journal of Bangladesh College of Physicians and Surgeons

Sir,

I am thankful to the learned reader for his keen interest in the subject and inspiratory remarks.I duly honor his valuable comments

given in (1) and (2). Regarding his queries (3), (4) and (5) I shall try to give the best answer.

Stylet doesnt hinder EUS FNA but having said that evidence have not shown it to be any better either.

Needle with stylet is stiffer than without it.Expert opinion differs regarding its use.In our centre it was used regularly.

Stylet helps to exclude obstructing tissue plugs inside the needle tip by reintroducing it when the needle is advanced into the lesion under ultrasonic guidance and then removed completely.

In endoscopic ultrasound-fine needle aspiration of solid lesions, technique matters rather than the needle size.

In few cases, punch technique is the only way to penetrate hard lesions.Usually, needles we use are 25 gauge for pancreas lesions, 19 gauze for wall lesions and 22 gauge for lymph nodes.

Contrast harmonic endosonography (CHEUS) is not widely available.Unlike CT and MRI examinations, contrast agents

are not routinely used to enhance images during EUS.In spite of the good performance characteristics of EUS and EUSFNA,

differentiation of malignant from inflammatory masses and assessment of tumor extent remain challenging.CHEUS uses a

second generation ultrasonic contrast agent and depicts intratumoral vessels in real time.CHEUS improved the visualization of tumor margins and vascular invasion and differentiated benign from malignant masses.It identified irregular vessels and ther by predicted GIST malignancies with a higher sensitivity, specificity and accuracy than that of high-grade malignancy GISTs by

EUS-guided FNA.In a single center study,CHEUS successfully visualized intratumoral vessels which may play an important role in predicting the malignancy risk of GISTs.

Enthusiastic and thoughtful comments as given by Dr. Ahmedul Kabir are always welcome.

Warm regards
Colonel (Prof) Shaila Perveen.
Classified Medicine Specialist and Gastroenterologist
CMH Jessore.

COLLEGE NEWS

(J Banagladesh Coll Phys Surg 2015; 33: 109-115)

College Examinations news: Results of FCPS Part-I, Part-II and MCPS examination held in January are given bellow: 4181 candidates appeared in FCPS Part-I, examination held in January, 2015 of which 471 candidates came out successful.

Subject wise results are as follows:

Result of FCPS Part-I Examination (January, 2015)

SL. No.	Subject	January-15		
		Total Candidate	Total Passed	Percentage
1.	Anaesthesiology	111	18	16.22
2.	Biochemistry	7	0	0.00
3.	Dentistry	174	17	9.77
4.	Dermatology & Venereology	68	3	4.41
5.	Family Medicine	1	0	0.00
6.	Haematology	25	3	12.00
7.	Histopathology	17	4	23.53
8.	Medicine	1412	175	12.39
9.	Microbiology	14	1	7.14
10.	Obst. & Gynae	883	117	13.25
11.	Ophthalmology	104	18	17.31
12.	Otolaryngology	137	11	8.03
13.	Paediatrics	419	38	9.07
14.	Physical Medicine & Rehabilitation	24	7	29.17
15.	Psychiatry	15	4	26.67
16.	Radiology & Imaging	50	1	2.00
17.	Radiotherapy	26	8	30.77
18.	Surgery	694	46	6.63
19	Transfusion Medicine			
	TOTAL	4181	471	11.27

The following candidates satisfied the Board of Examiners and are declared to have passed the FCPS - II Examinations held in January, 2015 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons.

Roll No.	Name	From where graduated	Subject
025-8905	Marufa Mustari	Rangpur Medical College, Rangpur	Endocrinology and Metabolism
025-8906	Sadiqa Tuqan	Mymensingh Medical College, Mymensingh	Endocrinology and Metabolism
025-8908	Ariful Basher	Chittagong Medical College, Chittagong	Infectious Disease and Tropical Medicine
025-8909	Mohmmad Mozibur Rahman	MAG Osmani Medical College, Sylhet	Neonatology
025-8914	Muhammed Shariful Islam	Comilla Medical College, Comilla	Plastic and Reconstructive Surgery
025-8915	Rafiq Uddin Ahmed	Comilla Medical College, Comilla	Plastic and Reconstructive Surgery
025-8916	Ayesha Hanna	Chittagong Medical College, Chittagong	Plastic and Reconstructive Surgery
025-8917	Md. Mahbubur Rahman	Rajshahi Medical College, Rajshahi	Plastic and Reconstructive Surgery
025-8918	Kazi Imran Ahmed	Sher-E-Bangla Medical College, Barisal	Plastic and Reconstructive Surgery

Roll No.	Name	From where graduated	Subject
025-8919	M. A. Hamid	Dhaka Medical College, Dhaka	Plastic and Reconstructive Surgery
025-8920	Md. Ashif Chowdhury	Rajshahi Medical College, Rajshahi	Urology
025-8921	Muhammad Zia Uddin	Comilla Medical College, Comilla	Urology
086-7013	Mohammad Asadullah	Rajshahi Medical College, Rajshahi	Anaesthesiology
086-7016	Fahd A.A. Karim	Bangladesh Dental College, Dhaka	Conservative Dentistry and Endodontics
086-7018	Jahan Tabassum	Sapporo Dental College, Dhaka	Conservative Dentistry and Endodontics
086-7029	Kaniz Rahman	Medical College for Women and Hospital, Dhaka	Dermatology and Venereology
086-7032	Farzana Afroz	Dinajpur Medical College, Dinajpur	Dermatology and Venereology
086-7033	Humaira Afreen	Dhaka Medical College, Dhaka	Dermatology and Venereology
086-7034	Md. Shirajul Islam Khan	Rangpur Medical College, Rangpur	Dermatology and Venereology
086-7037	Md. Humayun Kabir	Sir Salimullah Medical College, Dhaka	Dermatology and Venereology
086-7039	Tahmina Akter	Ibrahim Medical College, Dhaka	Dermatology and Venereology
086-7041	Tasnuva Ashraf	Rangpur Medical College, Rangpur	Dermatology and Venereology
086-7044	Shafiqul Islam	Khulna Medical College, Khulna	Haematology
086-7047	Mosammat Samira Taufique Rashma	Chittagong Medical College, Chittagong	Haematology
086-7048	Salina Haque	Rangpur Medical College, Rangpur	Haematology
086-7106	Md. Soroar Hossain	Rajshahi Medical College, Rajshahi	Medicine
086-7177	Shamsun Nahar	Mymensingh Medical College, Mymensingh	Medicine
086-7228	Eshita Biswas	Mymensingh Medical College, Mymensingh	Medicine
086-7244	Aminul Islam	Rajshahi Medical College, Rajshahi	Medicine
086-7245	Md. Rashedul Hasan	MAG Osmani Medical College, Sylhet	Medicine
086-7259	Md.Rabiul Awal	Sir Salimullah Medical College, Dhaka	Medicine
086-7300	Mohammed Maksudul Karim	Chittagong Medical College, Chittagong	Medicine
086-7327	Md. Bellal Hossain	Sir Salimullah Medical College, Dhaka	Medicine
086-7335	Mohammad Jahidul Hasan	Faridpur Medical College, Faridpur	Medicine
086-7339	Md. Yousuf Ur Rahman	Rangpur Medical College, Rangpur	Medicine
086-7343	Mirza Nurul Karim	MAG Osmani Medical College, Sylhet	Medicine
086-7359	Susmita Islam	Sher-E-Bangla Medical College, Barisal	Medicine
086-7362	Sanzida Akter	Chittagong Medical College, Chittagong	Medicine
086-7373	Sheikh Mohammad Samsuzzaman	Dhaka Medical College, Dhaka	Medicine
086-7416	Muhammad Saiyedur Rahman	Sir Salimullah Medical College, Dhaka	Medicine
086-7418	K.M. Nazmul Islam Joy	Dhaka Medical College, Dhaka	Medicine
086-7425	Habib Iftekhar Ahmad	Chittagong Medical College, Chittagong	Medicine
086-7432	Sukanta Chandra Das	Dhaka Medical College, Dhaka	Medicine
086-7450	Taslima Akter	Dhaka Medical College, Dhaka	Medicine
086-7497	Probal Sutradhar	Rangpur Medical College, Rangpur	Medicine
086-7499	Suraiya Nazneen	Sir Salimullah Medical College, Dhaka	Medicine
086-7502	Tasnuva Saiful	Sher-E-Bangla Medical College, Barisal	Medicine
086-7520	Ismet Nigar	Sir Salimullah Medical College, Dhaka	Microbiology
086-7521	Jayanti Bachhar	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7533	Nasrin Jahan Baker	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7538	Razia Akter	Jahurul Islam Medical College, Bajitpur	Obst and Gynae
086-7541	Shafeya Khanam	Mymensingh Medical College, Mymensingh	Obst and Gynae
086-7568	Ruma Akter	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7581	Sadia Jerifa	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7585	Rabeya Khanom	Medical College for Women and Hospital, Dhaka	Obst and Gynae
086-7586	Mosammat Amina Begum	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
086-7591	Shohana Shikder	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
086-7601	Muhammad Jasim Uddin	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
086-7607	Nihar Bala	Mymensingh Medical College, Mymensingh	Obst and Gynae

Roll No.	Name	From where graduated	Subject
086-7667	Mst. Nilufar Jahan	Rangpur Medical College, Rangpur	Obst and Gynae
086-7673	Taslima Akter	Mymensingh Medical College, Mymensingh	Obst and Gynae
086-7685	Fahmida Sultana	North-East Medical College, Sylhet	Obst and Gynae
086-7700	Papiya Sultana	Dhaka Medical College, Dhaka	Obst and Gynae
086-7723	Sharmin Akter	Chittagong Medical College, Chittagong	Obst and Gynae
086-7734	Rushdana Rahman	Mymensingh Medical College, Mymensingh	Obst and Gynae
086-7740	Sanzida Huda	Khulna Medical College, Khulna	Obst and Gynae
086-7756	Zannat Ara Begum	Chittagong Medical College, Chittagong	Obst and Gynae
086-7767	Susmita Sarker	MAG Osmani Medical College, Sylhet	Obst and Gynae
086-7770	Sohelee Nargis	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7774	Sanjida Parveen	Rajshahi Medical College, Rajshahi	Obst and Gynae
086-7797	Sharmina Jalil	Rajshahi Medical College, Rajshahi	Obst and Gynae
086-7802	Farzana Chowdhury	Chittagong Medical College, Chittagong	Obst and Gynae
086-7804	Mst. Rokeya Satter	Shaheed Ziaur Rahman Medical College, Bogra	Obst and Gynae
086-7807	Lipy Bakshi	Rangpur Medical College, Rangpur	Obst and Gynae
086-7821	Kumer Tanshen	Mymensingh Medical College, Mymensingh	Obst and Gynae
086-7828	Samira Haque	Rangpur Medical College, Rangpur	Obst and Gynae
086-7833	Runa Laila	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7835	Chhanda Majumder	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7839	Mst. Nofisa Khatun	Rangpur Medical College, Rangpur	Obst and Gynae
086-7840	Jesika Rizvi Tamanna	Holy Family Red Crescent Medical College, Dhaka	Obst and Gynae
086-7842	Aliya Amin Ul Haq	Chittagong Medical College, Chittagong	Obst and Gynae
086-7853	Farhana Tanzin	Mymensingh Medical College, Mymensingh	Obst and Gynae
086-7862	Kazi Khadeza Farhin	Z.H. Sikder Women's Medical College, Dhaka	Obst and Gynae
086-7869	Mst. Assamsunnahar Begum	Dhaka Medical College, Dhaka	Obst and Gynae
086-7901	Selina Begum	Jahurul Islam Medical College, Bajitpur	Obst and Gynae
086-7907	Fahima Akter	MAG Osmani Medical College, Sylhet	Obst and Gynae
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086-7962	Dilruba Akhter	Dhaka Medical College, Dhaka	Obst and Gynae
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086-7967	Novera Islam	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7973	Amina Jannat Peea	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-7982	Shirin Shabnam	Rangpur Medical College, Rangpur	Obst and Gynae
086-7983	Mushammat Farzana Khondker	Comilla Medical College, Comilla	Obst and Gynae
086-7991	Lipika Das	Rangpur Medical College, Rangpur	Obst and Gynae
086-7995	Nisfun Naher	Mymensingh Medical College, Mymensingh	Obst and Gynae
086-8033	Mohammad Altaf Hossain	MAG Osmani Medical College, Sylhet	Ophthalmology
086-8034	Muhammad Humayun Kabir	Rangpur Medical College, Rangpur	Ophthalmology
086-8035	Md Forhad Hossain	Rajshahi Medical College, Rajshahi	Ophthalmology
086-8037	Jewell Ilias Rab	Sher-E-Bangla Medical College, Barisal	Ophthalmology
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086-8042	Md. Rahmat-E-Khuda	Dhaka Medical College, Dhaka	Ophthalmology
086-8044	Mohammad Kamrul Hasan	Mymensingh Medical College, Mymensingh	Ophthalmology
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Roll No.	Name	From where graduated	Subject
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086-8052	Mossammat Shoheli Nasrin	Mymensingh Medical College, Mymensingh	Ophthalmology
086-8053	Tania Rahman Chhara	Kumudini Womens' Medical College, Tangail	Ophthalmology
086-8054	Rubina Akther	Jahurul Islam Medical College, Bajitpur	Ophthalmology
086-8058	Md. Almasul Islam	Chittagong Medical College, Chittagong	Ophthalmology
086-8059	Bipul Kumer De Sarker	Rajshahi Medical College, Rajshahi	Ophthalmology
086-8060	Shifat Toufique	Armed Forces Medical College, Dhaka	Ophthalmology
086-8061	Mohammad Ibn Abdul Malek	Sir Salimullah Medical College, Dhaka	Ophthalmology
086-8069	Abul Hasnat	Chittagong Medical College, Chittagong	Oral and Maxillofacial Surgery
086-8072	Newaz Mohsina	Rangpur Medical College, Rangpur	Oral and Maxillofacial Surgery
086-8074	Md. Ariful Islam	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
086-8079	A.S.M. Didar Alam Khan	Pioneer Dental College, Dhaka	Oral and Maxillofacial Surgery
086-8081	Md. Emdadul Haque	Rajshahi Medical College, Rajshahi	Oral and Maxillofacial Surgery
086-8083	Muhammad Mizanur Rahaman	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
086-8089	Luthfun Nahar	City Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
086-8093	Moniruzzaman	Dhaka Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
086-8094	Md. Asaduzzaman Sheikh	Dhaka Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
086-8112	Md. Abdur Razzak	Rangpur Medical College, Rangpur	Otolaryngology
086-8117	Md. Mahmudul Amin Sakik	Faridpur Medical College, Faridpur	Otolaryngology
086-8136	Sharmin Afrozy	Rajshahi Medical College, Rajshahi	Paediatrics
086-8156	Md. Abdullah Al Mamun	Chittagong Medical College, Chittagong	Paediatrics
086-8163	Mohammad Showkat Ali	Sher-E-Bangla Medical College, Barisal	Paediatrics
086-8199	Shirina Yasmin	Dhaka Medical College, Dhaka	Paediatrics
086-8212	Md. Sazzad Haider Shahin	Rangpur Medical College, Rangpur	Paediatrics
086-8216	Mohammad Mohsin	Sher-E-Bangla Medical College, Barisal	Paediatrics
086-8223	Aparup Kanti Das	MAG Osmani Medical College, Sylhet	Paediatrics
086-8232	Mrinal Kanti Das	Dinajpur Medical College, Dinajpur	Paediatrics
086-8240	Taskina Mosleh	Rangpur Dental College, Rangpur	Paediatrics
086-8243	Chaity Barua	Dhaka Medical College, Dhaka	Paediatrics
086-8246	Shakila Sharmin	Rajshahi Medical College, Rajshahi	Paediatrics
086-8299	Gule Tajkia	Sir Salimullah Medical College, Dhaka	Paediatrics
086-8303	Rubaba Sharmin	Dhaka Medical College, Dhaka	Paediatrics
086-8322	Simu Saha	Comilla Medical College, Comilla	Paediatrics
086-8323	Md. Tauhidul Islam	Mymensingh Medical College, Mymensingh	Paediatrics
086-8328	Rasheda Akther	Mymensingh Medical College, Mymensingh	Paediatrics
086-8332	Farzana Afroze	MAG Osmani Medical College, Sylhet	Paediatrics
086-8343	Mobashshera Rahman	Dhaka Medical College, Dhaka	Paediatrics
086-8347	Muhammad Alamgir Mandal	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
086-8349	Md. Alauddin All Mamun	Khulna Medical College, Khulna	Physical Medicine & Rehabilitation
086-8352	Prasanta Kumar Chakraborty	Rangpur Medical College, Rangpur	Physical Medicine & Rehabilitation
086-8353	Dilir Jamal	Faridpur Medical College, Faridpur	Physical Medicine & Rehabilitation
086-8356	Md. Nurul Hoque Miah	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
086-8357	Rayhan Hamid	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
086-8359	Abul Khair Ahmad Zaman	Community Based Medical College, Mymensingh	Physical Medicine & Rehabilitation
086-8368	Mahbuba Shirin	Dhaka Medical College, Dhaka	Radiology & Imaging
086-8372	Farzana Rahman	Mymensingh Medical College, Mymensingh	Radiology & Imaging

Roll No.	Name	From where graduated	Subject
086-8379	Rowshon Ara Begum	Sir Salimullah Medical College, Dhaka	Radiotherapy
086-8380	Md. Nizamul Haque	Dhaka Medical College, Dhaka	Radiotherapy
086-8381	Asma Siddiqua	MAG Osmani Medical College, Sylhet	Radiotherapy
086-8392	Mohammad Saief Uddin	Chittagong Medical College, Chittagong	Surgery
086-8402	Md. Mahbubur Rahman	Shaheed Ziaur Rahman Medical College, Bogra	Surgery
086-8415	Abu Faisal Md. Ariful Islam	Khulna Medical College, Khulna	Surgery
086-8437	Shah Raihanur Rahman	Mymensingh Medical College, Mymensingh	Surgery
086-8440	Md Kamrujjaman	Faridpur Medical College, Faridpur	Surgery
086-8456	Mohammad Mainul Islam	Dhaka Medical College, Dhaka	Surgery
086-8457	Asadullahil Galib	Khulna Medical College, Khulna	Surgery
086-8471	Md. Mizanur Rahman	Rangpur Medical College, Rangpur	Surgery
086-8473	Mohammad Jahirul Islam	Khulna Medical College, Khulna	Surgery
086-8480	A. K. M. Shamsul Haque	Rajshahi Medical College, Rajshahi	Surgery
086-8499	Mohammed Omar Faroque	Rangpur Medical College, Rangpur	Surgery
086-8508	A.H.M. Tanvir Ahmed	Jahurul Islam Medical College, Bajitpur	Surgery
086-8519	Dipak Chandra Kirttania	Sher-E-Bangla Medical College, Barisal	Surgery
086-8522	Md. Shahidul Islam	Rajshahi Medical College, Rajshahi	Surgery
086-8523	Hasan Shahriar Md. Nuruzzaman	Dhaka Medical College, Dhaka	Surgery
086-8529	Nasim-E-Tasnim	Armed Forces Medical College, Dhaka	Surgery
086-8539	Rana Jahangir Alam	Chittagong Medical College, Chittagong	Surgery
086-8543	Md. Ahsan Habib	Rangpur Medical College, Rangpur	Surgery
086-8548	Subal Chandra Paul	Rajshahi Medical College, Rajshahi	Surgery
086-8564	Reza Ahmad	Dhaka Medical College, Dhaka	Surgery
086-8568	Muhammad Iqbal Hossain	Khulna Medical College, Khulna	Surgery
086-8569	Avisak Bhattacharjee	Chittagong Medical College, Chittagong	Surgery
086-8578	Muhammad Faruk Hussain	Mymensingh Medical College, Mymensingh	Surgery
086-8587	Nasima Akhter	Comilla Medical College, Comilla	Surgery
086-8588	Farjana Haque Shumi	Mymensingh Medical College, Mymensingh	Surgery
086-8589	Muhammad Mamun Miah	Rangpur Medical College, Rangpur	Surgery
086-8602	Muhammad Enamul Haque	Sher-E-Bangla Medical College, Barisal	Surgery
086-8611	Pradip Kumar Nath	Comilla Medical College, Comilla	Surgery
086-8612	Muntasir Faisel	Bangladesh Medical College, Dhaka	Surgery
086-8614	Farhad Uddin Ahmed Bhuiyan	Comilla Medical College, Comilla	Surgery
086-8616	Muhammad Nuruzzaman	Shaheed Mansur Ali Medical College, Uttara, Dhaka.	Surgery
086-8623	Syeda Shahnaz Nasrullah	Sir Salimullah Medical College, Dhaka	Surgery
086-8624	Saimun Naher	Sir Salimullah Medical College, Dhaka	Surgery
086-8630	Md. Tafiqul Islam	Rajshahi Medical College, Rajshahi	Surgery
086-8633	Sharmin Akter Sumi	Mymensingh Medical College, Mymensingh	Surgery
086-8634	Md. Obaidul Islam	Dhaka Medical College, Dhaka	Surgery
086-8637	Sarwar Ahmed Sobhan	Dhaka Medical College, Dhaka	Surgery
086-8641	Mohammad Azim Uddin	Mymensingh Medical College, Mymensingh	Surgery
086-8646	Md. Ashek Mahmud Ferdaus	Mymensingh Medical College, Mymensingh	Surgery
086-8656	Nazia Farid	Dhaka Medical College, Dhaka	Surgery
086-8657	Md. Kamrul Huda	Rajshahi Medical College, Rajshahi	Surgery
086-8659	Sobhana Iftekhar Tani	Bangladesh Medical College, Dhaka	Surgery
086-8666	Ashrafun Nessa	Sir Salimullah Medical College, Dhaka	Surgery
086-8667	G. M. Nuruzzaman	Rajshahi Medical College, Rajshahi	Surgery
086-8669	Krishna Pada Saha	Sher-E-Bangla Medical College, Barisal	Surgery
086-8673	Md. Saiful Islam	Sir Salimullah Medical College, Dhaka	Surgery
086-8680	Sonia Shormin	MAG Osmani Medical College, Sylhet	Transfusion Medicine

The following candidates satisfied the Board of Examiners and are declared to have passed the MCPS Examinations held in January, 2015 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons.

Roll No.	Name	From where graduated	Subject
086-9012	Kaniz Fatema	Community Based Medical College, Mymensingh	Clinical Pathology
086-9013	Tania Gaffar	Bangladesh Medical College, Dhaka	Clinical Pathology
086-9014	Mohammad Shahidul Islam	Sher-E-Bangla Medical College, Barisal	Clinical Pathology
086-9015	Sifat-E-Moyen	Armed Forces Medical College, Dhaka	Clinical Pathology
086-9022	Kazi Mohammad Khaled Huda	Dhaka National Medical College, Dhaka	Dermatology and Venereology
086-9023	Sharmin Maria	Rajshahi Medical College, Rajshahi	Dermatology and Venereology
086-9024	Farah Diba Sultana	Rajshahi Medical College, Rajshahi	Dermatology and Venereology
086-9026	Md. Alauddin Khan	Sher-E-Bangla Medical College, Barisal	Dermatology and Venereology
086-9030	Fouzia Yeasmin	Bangladesh Medical College, Dhaka	Dermatology and Venereology
086-9037	Ahmad Sadek	MAG Osmani Medical College, Sylhet	Forensic Medicine
086-9039	Sanjida Akhter	MAG Osmani Medical College, Sylhet	Forensic Medicine
086-9049	Biswas Abul Hassan	Rajshahi Medical College, Rajshahi	Medicine
086-9057	Bishnu Prosad Chanda	Comilla Medical College, Comilla	Medicine
086-9071	Dilip Kumar Sarker	MAG Osmani Medical College, Sylhet	Medicine
086-9077	Masud Karim	Sher-E-Bangla Medical College, Barisal	Medicine
086-9103	Md. Mahbubul Alam	Mymensingh Medical College, Mymensingh	Medicine
086-9137	Md. Azizul Haque	Sir Salimullah Medical College, Dhaka	Medicine
086-9142	Mohammad Jane Alam	Sher-E-Bangla Medical College, Barisal	Medicine
086-9145	A. T. M. Hasibul Hasan	Dhaka Medical College, Dhaka	Medicine
086-9160	Md. Mamun-Ur Rashid	Rajshahi Medical College, Rajshahi	Medicine
086-9164	Shankar Das Gupta	Chittagong Medical College, Chittagong	Medicine
086-9166	Md. Abul Khair Yousuf	Sir Salimullah Medical College, Dhaka	Medicine
086-9167	Mohammad Shamsul Alam	Sir Salimullah Medical College, Dhaka	Medicine
086-9168	Md. Faruk Hossen	Dhaka Medical College, Dhaka	Medicine
086-9172	S. M. Amanat Ullah	Dhaka Medical College, Dhaka	Medicine
086-9181	Muhammad Ataul Gani Osmani	Sir Salimullah Medical College, Dhaka	Medicine
086-9184	Mohammad Shahidul Islam Bhuiyan	MAG Osmani Medical College, Sylhet	Medicine
086-9186	Md. Fakhrul Islam Juwel	Dhaka Medical College, Dhaka	Medicine
086-9187	Farhana Sayeed	Dhaka Medical College, Dhaka	Medicine
086-9200	Md. Belal Hossain	Rajshahi Medical College, Rajshahi	Medicine
086-9205	Khandaker Abu Rubaiyat	Dhaka Medical College, Dhaka	Medicine
086-9212	Mehjabeen Tasnuva Aslam	Armed Forces Medical College, Dhaka	Medicine
086-9217	Md. Moin Uddin	Rajshahi Medical College, Rajshahi	Medicine
086-9234	Gobinda Gain	Sher-E-Bangla Medical College, Barisal	Medicine
086-9271	Fatema Begum	Faridpur Medical College, Faridpur	Obst and Gynae
086-9282	Farhana Ahmed Nancy	Bangladesh Medical College, Dhaka	Obst and Gynae
086-9341	Hamida Parvin	Rangpur Medical College, Rangpur	Obst and Gynae
086-9353	Fahmida Hasnat	Sir Salimullah Medical College, Dhaka	Obst and Gynae
086-9366	Shamsun Naher	Rajshahi Medical College, Rajshahi	Obst and Gynae
086-9374	Mst. Rukshana Pervin	Rajshahi Medical College, Rajshahi	Obst and Gynae
086-9385	Banani Bhowmik	Dhaka Medical College, Dhaka	Obst and Gynae
086-9396	Ayesha Siddiqua	Rajshahi Medical College, Rajshahi	Obst and Gynae
086-9406	Rina Akter	Mymensingh Medical College, Mymensingh	Ophthalmology
086-9407	Muliha Rahman	Chittagong Medical College, Chittagong	Ophthalmology
086-9411	Mohammad Rashedul Hasan	Dhaka National Medical College, Dhaka	Ophthalmology
086-9425	Susmita Sarkar	Kumudini Womens' Medical College, Tangail	Ophthalmology
086-9426	Md. Mahmudur Rahman	Dhaka Medical College, Dhaka	Ophthalmology

Roll No.	Name	From where graduated	Subject
086-9427	Mohammad Mamunur Rashid Chowdhury	Dhaka Medical College, Dhaka	Ophthalmology
086-9439	Md. Monirul Alam	Dhaka Medical College, Dhaka	Otolaryngology
086-9440	Sanjoy Das	Sir Salimullah Medical College, Dhaka	Otolaryngology
086-9462	Suntanu Kumar Kar	Sir Salimullah Medical College, Dhaka	Paediatrics
086-9486	Saleh Mohammad Hasibul Hasan	Dhaka Medical College, Dhaka	Paediatrics
086-9487	Tasnuva Khan	Dhaka Medical College, Dhaka	Paediatrics
086-9491	Shahnaz Pervin Sumi	Armed Forces Medical College, Dhaka	Paediatrics
086-9495	Neeha Maahapara Matin	Bangladesh Medical College, Dhaka	Psychiatry
086-9497	Rubaiyat Ferdush	Z.H. Sikder Women's Medical College, Dhaka	Psychiatry
086-9498	Md. Harunur Rashid	Bangladesh Medical College, Dhaka	Psychiatry
086-9499	Ahsan Uddin Ahmed	Bangladesh Medical College, Dhaka	Psychiatry
086-9501	Nahar-E-Zannat	Chittagong Medical College, Chittagong	Radiology & Imaging
086-9502	Nikhileshwar Roy	Dhaka Medical College, Dhaka	Radiology & Imaging
086-9504	Md. Javed Mahfuz Khan	Rajshahi Medical College, Rajshahi	Radiology & Imaging
086-9505	Mukthdira	Community Based Medical College, Mymensingh	Radiology & Imaging
086-9508	Md. Al-Emran	Rajshahi Medical College, Rajshahi	Radiology & Imaging
086-9509	Mohammad Nasir Uddin	MAG Osmani Medical College, Sylhet	Radiotherapy
086-9512	Salimullah Khan	Comilla Medical College, Comilla	Surgery
086-9523	Md. Salim	Chittagong Medical College, Chittagong	Surgery
086-9535	A. F. M. Azizur Rahman Siddique	MAG Osmani Medical College, Sylhet	Surgery
086-9561	Md. Mofiz Uddin	Dhaka Medical College, Dhaka	Surgery
086-9564	A.S.M. Kutub Uddin Awal	Sir Salimullah Medical College, Dhaka	Surgery
086-9566	Sadia Imdad	Sir Salimullah Medical College, Dhaka	Surgery

FROM THE DESK OF EDITOR in CHIEF

(J Banagladesh Coll Phys Surg 2015; 33: 116)

Dear Fellows

Every good thing comes to an end and opens the door for a better replacement. This is going to be my last letter to you as the Editor in Chief of our favorite journal. I and my team have tried our level best to uphold the high standards of the Journal of BCPS. During our tenure we have gotten rid of the back log and the issues are now up-to-date. Software has been developed to keep track of all the publication and the authors along with their submissions. A new website for the journal has been installed which now fully operational. The journal committee tried to ensure publication of quality original articles. Over the years there have been an increase in the number of subscribers and the journal now has a larger group of readers. Though we haven't been able to index the journal yet, we have taken necessary steps which will make it easier for the journal to be indexed in near future. I would like to thank the fellows and my editorial team for their relentless hard work and support and wish the best in life for all. Finally I would like to welcome the new Editor In Chief.

Prof. HAM Nazmul Ahasan
Editor In Chief
Journal of BCPS

Obituary

(J Banagladesh Coll Phys Surg 2015; 33: 117)

The following fellows who died on October, 14 to April, 2015

Professor Md. Mahbub-Ul-Alam

Professor Md. Mahbub-Ul-Alam died on 15th October, 2014.He own fellowship with examination in Surgery,1984from Bangladesh College of Physicians and Surgeons (BCPS).

Dr. Dil Afroza Akhtar

Dr. Dil Afroza Akhtar died on 17th January, 2015.She own fellowship with examination in Obst & Gynae, 2005from Bangladesh College of Physicians and Surgeons (BCPS).