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Contents

Editorial

Universal Health Coverage: The Key to Achieve Sustainable Developmental Goals

Yasmeen S

Health is now considered as a developmental agenda all over the world. Transition from MDGs to SDGs has generated new hopes and challenges. Eventually success will depend on country wise most feasible plan of action. In 2016, it was stated that every year 150 million people suffer from financial catastrophe & 100 million are pushed into poverty because of out of pocket expenditure on health. At least one billion people globally lack access to one or more essential health services and 56% of the global rural population has no health coverage (WHO). From the post MDGs scenario we can find out the loopholes, can learn lessons and rectify for implementation of SDGs.

In SDGs from 2016 to 2030, issues in health agenda are placed very rationally. There is inclusion of newer issues which were absent in MDGs like-non-communicable diseases including mental health, substance abuse, road traffic accident and some important elements of environmental pollution (air, water & soil pollution, chemical intoxication etc.). Role of health is framed as contributor to and beneficiary of sustainable development. The health and wellbeing goal (3rd goal in SDGs) is 'Ensure healthy lives and promote well-being for all at all ages'. Under this goal there are 9 targets and 8th target (3.8) is "achieve universal health coverage including financial risk protection, access to quality essential health care services and access to safe, effective, quality and affordable and essential medicines and vaccines for all." Under the umbrella of UHC all people will receive quality health care without financial hardship. Quality health care encompasses promotive, preventive, curative, rehabilitative health care & palliative care; quality implies to people centeredness, equitability, accessibility, effectiveness, efficiency & safety.

Basically UHC will have to ensure two types of coverage- 1) Health service coverage, 2) Financial coverage for health service. Coverage of essential services indicators are based on categories of: 1) Reproductive, maternal, newborn and child health, 2) Infectious diseases, 3) Non-communicable diseases and 4) Service capacity and access. Coverage of financial risk protection is based on indicators like 1) Incidence of catastrophic health expenditure due to out-of-pocket payments; 2) Incidence of impoverishment due to out-of-pocket payments. Therefore UHC is an essential strategy to end extreme poverty and to boost shared prosperity.

In Bangladesh significant improvement was observed in achieving MDGs especially in child health, maternal health and combating communicable diseases. The experience of MDGs showed evidence that despite limited resources Bangladesh was far ahead in achieving targets from other regional countries where per capita expenditure on health was more than Bangladesh; in Bangladesh per capita health expenditure is only 27 USD. Currently in Bangladesh direct household out of pocket health expenditure (OOP-HE) is 64% which pushes people towards poverty. According to WHO, OOP-HE more than 20% to 30% indicates problem in financial protection for healthcare. So we must generate fund for health & UHC. Bangladesh has adopted Health Care Financing Strategy (2012-2032) with a target to reduce the OOP-HE from 64% to 32%. As theoretically this 32% OOP-HE is above the cutoff value (30%) of financial risk protection, question may arise in ensuring coverage for both. Answer lies in the holistic and innovative approaches towards achieving the targets. Bangladesh is being treated as an emerging economy with aim to be a middle income country by 2021 and high income country by 2041.

Health workforce is an important component for UHC. There is shortage, inappropriate skill mix and inequitable distribution of health workforce in our country. The formal health workforces (doctors, dentists, nurses) are mostly concentrated in the urban areas. The doctor to population ratio is 1 per 1,500 people in urban areas, whereas it is 1 per 15,000 in rural areas. Retention and absenteeism are two major problems in rural areas. If health workers are not in sufficient numbers, not available and close to the people who need them, or medicines are unavailable, or if the quality of care is low- it is difficult to reach UHC. The first static facility in primary health care level is now the community clinics. People's participation and their empowerment are the two important innovative components need to be geared up and integrated with the national health system. Health services, including traditional and complementary medicine services, are to be organized around the comprehensive needs and expectations of people and communities must help to empower them to take more active role in their health and health system.

Many people think that UHC is merely an aspiration and in reality it is impossible to attain. But a strong and positive political commitment with good governance, all the concerned stakeholders including NGOs and private sectors have to own the health targets of SDGs based on UHC. Quality health care services at low and shared cost can be made available and accessible at the door step of all people. Let us come forward to work together to build a healthy nation and healthy generation.

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Original Article

Urinary Tract Infections and Pattern of Antimicrobial Sensitivity of *Escherichia coli* in Pediatric Patients in a Tertiary Care Hospital in Dhaka City

Nahar A[a], Rahman M[b], Jaigirdar MA[c]

Abstract

Background: Urinary tract infections (UTIs) remain the most common bacterial infection in childhood. Around 1% of boys and 5% of girls develop UTI during first ten years of life. To reduce the burden and also to prevent complications of UTIs appropriate use of antimicrobials is necessary.

Objectives: This study was undertaken to find out the common pathogens causing UTI in children and to assess in vitro antibiotic susceptibility pattern of *Escherichia coli* to commonly used antimicrobial agents in a tertiary care hospital in Dhaka city which may help the pediatricians to choose the appropriate antimicrobial treatment.

Methods: This cross sectional study was carried out in Bangladesh Medical College and Hospital, over a period of one year and six months from July 2014 to December 2015. Urine samples were collected from 710 children between 0 to 18 years of age admitted in inpatient department and who attended pediatric outpatient department with suspected UTI cases. Clean catch midstream and/or urine bag catch urine sample was collected into a sterile container/test tube aseptically. Growth of a single organism with a count $=10^5$ CFU/ml and >5 pus cells per high power field were considered to represent the infection. Antimicrobial susceptibility testing of the isolated bacterial species was performed by disc diffusion method following the National Committee for Clinical Laboratory Standards (NCCLS) guidelines.

Results: A total 710 suspected UTI patients were included in this study. Significant bacteriuria was found in 60 (8.45%) cases, among them 38 (63.33%) were girls and 22 (36.67%) were boys. Significant statistical association was found among the pattern of bacteria for the rate of isolation in urine culture in boys & girls. Among the 60 culture positive cases, majority of the isolates were from females. *Escherichia coli* was detected 48 (80%) of cultures, followed by *Pseudomonas* 07(11.67%), *Klebsiella* 04(6.67%) and *Staphylococcus aureus* 1(1.66%). *Escherichia coli* was 87.5% sensitive to cefuroxime, 89.58% to cefexime, 93.75% to ceftriaxone, 95.83% to nitrofurantoin, 93.75% to ciprofloxacin, 81.25% to gentamicin,75% to amikacin, 83.33% to imipenem and 87.5% to meropenem.

Conclusion: Empirical antibiotic selection should be based on the knowledge of local prevalence of bacteria and their drug sensitivity pattern rather than universal guidelines.

Keywords: Urinary tract infection, bacterial isolates, antibiotic susceptibility.

Introduction:

Urinary tract infections (UTIs) remain the most common bacterial infection in childhood.[1] Neonates, girls, young women and older men are most susceptible to UTIs. In women, bacterial cystitis is the most common bacterial infection. Around 1% of boys and 5% of girls develop UTI during first ten years of life.[2]

Majority of UTIs in children result due to ascending infection, although hematogenous spread may be more common in the first year of life. Individual differences in susceptibility to UTI may be due to host factors such as production of urethral and cervical IgA antibodies as well as other factors that influence bacterial adherence to the introitus and the urethral epithelium.[3,4] Congenital anomalies of the urinary tract such as posterior urethral valve, vesico-ureteric reflux, ureteral duplex etc., are also well known causes of UTI in children.[3]

Recognition of UTI in children should be made as early as possible to prevent from high morbidity and long term complications like renal scarring, hypertension, and end-stage renal disease. Early diagnosis is important to preserve renal function of the growing kidney. Several studies have reported varying prevalence rates of UTI in children ranging from 3.3% in USA to 37.5% in Pakistan.[5,6,7] Gram

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negative enteric bacilli, especially Escherichia coli and Klebsiella spp. are the leading pathogens though Enterococcus spp., yeasts and Staphylococcus aureus have emerged as prominent agents in recent years, many of them resistant to multiple antibiotics.[7,8]

UTI may present no urinary symptom or sign, and a delay in diagnosis and treatment may occur. In infants and young children aged < 3 years with unexplained fever, the degree of toxicity, dehydration, and ability to retain oral intake must be assessed carefully. The prognosis is usually favorable, but relies on timely administration of appropriate initial antimicrobial treatment. Appropriate treatment requires information regarding the susceptibility patterns of the current bacteria in order to give effective antibiotics in time.[9,10,11]

To reduce the burden and also to prevent the consequences like renal damage, mortality and morbidity appropriate use of antimicrobials for treatment and prevention of UTIs is important. This study was undertaken to detect the common pathogens causing UTIs in children and to assess in vitro antibiotic susceptibility pattern of *Escherichia coli* to commonly used antimicrobial agents in a tertiary care hospital in Dhaka city which may help the pediatrician in choosing the appropriate antimicrobial treatment.

Material and Methods:

This cross sectional study was carried out in Bangladesh Medical College and Hospital, over a period of one year and six months from July 2014 to December 2015. Sample collection, processing, culture & identification were done in the department of Microbiology, Bangladesh Medical College Hospital. Urine samples were collected from 710 children between 0 to 18 years of age admitted in inpatient department and who attended paediatric outpatient department with suspected UTI cases as well. Clean catch midstream and/or urine bag catch urine sample was collected into a sterile container/test tube aseptically.[12] Urine samples were processed semi-quantitatively, inoculating 0.001 ml urine by using a calibrated wire loop on Cystine-lactose-Electrolyte Deficient (CLED) agar media (Hi Media Laboratories, India) for the isolation and identification of significant uropathogens. The inoculated plates were incubated at 37^0C for 24 hours and extended to 48 hours in culture negative cases. Growth of a single organism with a count $=10^5$ CFU/ml and >5 pus cells per high power field were considered to represent the infection and were identified by using cultural morphology, Gram reactions, motility test and biochemical characteristics.[13,14,15,16,17] Antimicrobial susceptibility testing of the isolated bacterial species was performed by disc diffusion method following the National Committee for Clinical Laboratory Standards (NCCLS) guidelines.[18] All discs were obtained from Oxoid Ltd. Antibiotics used for uropathogens were ciprofloxacin (5µg), gentamicin (10µg), cefuroxime (30µg), cefexime (5µg), ceftriaxone (30µg), amikacin (30µg), nitrofurantoin (300µg), imipenem (10µg) and meropenem (10µg).

Results:

Gender distribution among culture positive cases are shown in figure 1. A total 710 suspected UTI patients were included in this study. Significant bacteriuria was found in 60 (8.45%) cases, among them 38 (63.33%) were girls and 22 (36.67%) were boys. There was a female preponderance in the culture positive cases, with an overall male to female ratio of 1: 1.7.

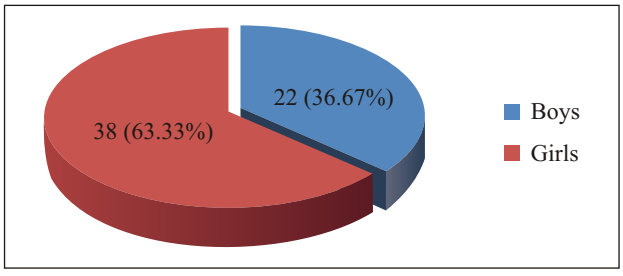

Figure 1: Gender distribution among culture positive cases (n=60)

Table 1: Pattern of bacteria for the rate of isolation in urine culture among boys & girls (n= 60)

Isolated organisms	Number of isolates		Total (%)	P value
	Boys (%)	Girls (%)		
Escherichia coli	14	34	48 (80%)	
Pseudomonas	05	02	07 (11.67%)	
Klebsiella	03	01	04 (6.67%)	P<0.05
Staphylococcus aureus	-	01	01 (1.66%)	
Total	22	38	60 (100%)	

Among the 60 culture positive cases, majority of the isolates were from females. *Escherichia coli* was detected 48 (80%) of cultures, followed by *Pseudomonas* 07(11.67%), *Klebsiella* 04(6.67%) and *Staphylococcus aureus* 1(1.66%). Statistical significant association was found among the pattern of bacteria for the rate of isolation in urine culture in boys & girls (Table 1).

Table 2: Antimicrobial sensitivity pattern of *Escerichia coli* (n= 48)

Drugs	*Escherichia coli* (n= 48)
Cefuroxime	42 (87.50%)
Cefexime	43 (89.58%)
Ceftriaxone	45 (93.75%)
Nitrofurantoin	46 (95.83%)
Ciprofloxacin	45 (93.75%)
Gentamicin	39 (81.25%)
Amikacin	36 (75%)
Imipenem	40 (83.33%)
Meropenem	42 (87.5%)

Antimicrobial sensitivity pattern of *Escherichia coli* has been shown in Table 2. In this study *Escherichia coli* was 87.5% sensitive to cefuroxime, 89.58% to cefexime, 93.75% to ceftriaxone, 95.83% to nitrofurantoin, 93.75% sensitive to ciprofloxacin, 81.25% sensitive to gentamicin,75% to amikacin, 83.33% to imipenem and 87.5% to meropenem.

Discussion:

Out of 710 children with suspected UTI, 60 had culture positive that is 8.45%. In this study UTI is more common in female children 63.33% than male children 36.67%. This might be due to short urethra in female. We found male female ratio 1: 1.7. Other such studies also showed male female ratio of 1:1.9 and 1:2.[19,20,21] A study done in Taiwan where prevalence rate of UTI was 11.3% which correlates with our study.[22] In India, prevalence rate of UTI was 20.73%.[23]

The *Escherichia coli* species was found to be the most common pathogen in our study accounting for 80% of UTI cases in the study patients, followed by *Pseudomonas* recorded in 11.67% of cases, *Klebsiella* 6.67% and *Staph. aureus* 1.66%. This was in accordance with other studies in which *Escherichai coli* was isolated from 87% to 92%.[24,25] Similar findings was also noted by different authors in various parts of the world where *Klebsiella* was isolated in 8.82%[26] and *Pseudomonas* 7.20%.[27] Our findings were different from a study in North India where *Escherichia coli* 68.30%, *Staph. aureus* 27.27%, *Klebsiella* 21.12% and *Pseudomonas* 0.70% were isolated.[28]

The initial choice of antibacterial therapy is based on knowledge of the predominant pathogen in the patient's age group, antibacterial sensitivity patterns in the practice area, the clinical status of the patient, and the opportunity for close follow up. The use of inappropriate antibiotic will delay effective treatment and increase the risks of urosepsis and renal scarring.[29]

Treatment generally begins with a broad spectrum antibiotic, but it may need to be changed based on the results of urine culture and sensitivity testing.[30] Parenteral antibiotics may be used with daily follow up until the patient is afebrile for 24 hours and complete 10-14 days of therapy with an oral antibiotic that is active against the infecting bacteria.[31] According to the American Academy of Pediatrics, the child should be reevaluated with a repeat urine culture and renal/bladder ultrasonography if clinical improvement does not occur within two days.[32]

Regarding cephalosporin group, *Escherichia coli* showed 87.5% sensitivity to cefuroxime**,** 89.58% to cefexime, 93.75% to ceftriaxone in this study. In Taiwan Escherichia coli was 94.1% sensitive to cefuroxime and 94.5% ceftriaxone which are similar to this study.[22] *Escherichia coli* was 17% sensitive to cefuroxime**,** 25.8% to ceftriaxone in India and in Pakistan ceftriaxone was 22.22% sensitive.[23,28,27] These findings differ from our study. The third generation cephalosporin should be reserved for critical cases to prevent drug resistance.

In this study nitrofurantoin was 95.83% sensitive to *Escherichia coli*. A study done in Turkey also reported highest sensitivity of nitrofurantoin 97.8% against *Escherichia coli*.[24] Other studies done in Greece and United kingdom also reported 95.6% and 93% sensitivity of *Escherichia coli* to nitrofurantoin respectively.[33,25] In Nepal *Escherichia coli* was 100% sensitivite to nitrofurantoin[3] and in India sensitivity was 72%.[23] Re-emergence of nitrofurantoin sensitivity is probably due to non usage of this drug for a long period of time. This drug is less commonly used in the treatment of UTI in recent years due to more adverse effects.

In this study *Escherichia coli* was 93.75% sensitive to ciprofloxacin. *Escherichia coli* was 95.2% sensitive to ciprofloxacin in Nepal.[34] An Indian study found that *Escherichia coli* was 22.6% sensitive to ciprofloxacin.[28] Ciprofloxacin has increased adverse events related to joints and surrounding tissue, so better not to use this drug though its sensitivity pattern was good. For this reason pediatrician do not use this drug in our hospital.

Regarding aminoglycoside our study showed that *Escherichia coli* was 81.25% sensitive to gentamicin and 75% to amikacin**.** In India Gupta found *Escherichia coli* was 72% sensitivite to amikacin and 41% sensitivite to gentamicin.[23] In Taiwan E coli was 86% sensitive to gentamicin and 100% sensitive to amikacin.[22]

In case of carbapenem group, *Escherichia coli* was 83.33% sensitive to imipenem and 87.5% to meropenem in this study. E. coli was 78.3% sensitive to meropenem in India.[28] In Pakistan and in Taiwan *Escherichia coli* was 100% sensitive to imipenem.[27,22] Generally, meropenem is four-to sixteen fold more active than imipenem against Enterobacteriaceae.[35,36,37,38,39] So, UTIs caused by Gram negative rods can be treated by meropenem.

Conclusion:

Our study shows that prevalence rate of UTI are observed higher in girls. *Escherichia coli* was found to be the most common pathogen that causes UTI. From the findings of our study we conclude that cefuroxime, ceftriaxone, cefexime, gentamicin and amikacin are effective therapeutic agents for pediatric UTIs. These above drugs can be started as empirical therapy after sending urine culture and sensitivity. So, empirical antibiotic selection should be based on the knowledge of local prevalence of bacteria and their antibiotic sensitivity pattern rather than Universal guidelines.

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Original Article

Laparoscopic Evaluation of Female Factors of Subfertility: A Study of 150 Cases at a Selected Hospital

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Abstract

Background: The Laparoscopic procedure has revolutionized the management of infertility cases. It is a necessary armamentarium for the diagnosis, treatment and prognosis of patients and guides the specialist to plan the surgery & other modalities of treatment.

Objective: This study was conducted to highlight the importance of Laparoscopy in evaluation of female factor of infertility and to evaluate different factors associated with infertility.

Methods: This retrospective cross-sectional study was conducted in Combined Military Hospital (CMH) during the period of July 2015 to June 2016. A total of 150 patients complaining of infertility of different age group were included in this study who underwent diagnostic laparoscopy in CMH Dhaka during the study period. All the information was collected on predesigned questionnaire and different parameters were evaluated. Data analysis was done in SPSS version 16.0.

Results: Among 150 patients 94 (62.7%) had primary infertility while 56 (37.3%) presented with secondary infertility. Eighty (53.3%) patients had infertility of more than 5 years. Majority 120 (80%) infertile patients were in the age group of 23-32 years. Sixty cases (40%) had some irregularities of menstrual cycle. When Laparoscopic findings were evaluated 22 (14.7%) patients had normal pelvic organs while 62 (41.3%) had polycystic ovaries, 10 (6.7%) had chocolate cyst, 16 (10.7%) had endometriosis and 16 (10.7%) had extensive pelvic adhesion. When tubal patency was evaluated by chromotubation, 36 (24%) patients had healthy and patent tubes while 100 (66.7%) patients had blocked tube. In 88 (58.7%) patients there were no spill of dye seen indicating bilateral blockade while 12 (8%) patients had dye spill on one side showing unilateral blockade of tube. About 64 (42.7%) patients of primary infertility had one or both blocked tube while 36 (24%) patients of secondary infertility had blocked tubes. Bilateral block was more than unilateral block both in primary and secondary infertility as 38.7% and 20% respectively. When Laparoscopy associated complications were noted, 20 (13.3%) patients had minor complication and major complication was nil.

Conclusion: Diagnostic Laparoscopy is a valuable technique and is a mandatory invasive investigation for complete assessment of female infertility before the couple progresses to infertility treatment especially where assisted reproductive techniques were not available.

Keywords: infertility; primary infertility; secondary infertility; diagnostic laparoscopy.

Introduction:

Infertility and uncontrolled fertility are two major problems affecting women's health and quality of life leading to social and psychological maladjustments.[1] Infertility is defined as one year of frequent, unprotected intercourse during which pregnancy has not occurred.[2] Primary infertility denotes those patients who have never conceived. Secondary infertility indicates previous pregnancy but failure to conceive subsequently.[3] Approximately 8% (52-80 million) of couples in worldwide are suffering from infertility. Major causes of infertility include male & female factors.[4] In 20-30% of cases the problem rests with the male, in 20-35% with the female, 20-45% with both partners and in a further 10-20% of cases, the cause is unknown.[3] Fertility problems strike one in three women over 35 years of age.

The appropriate selection of investigations based on problem areas identified by history and physical examination would guide the physician in the management of the infertile couple.[5]

In general, an infertility evaluation is initiated after 12 months of unprotected intercourse during which pregnancy has not been achieved.[6] Diagnostic Laparoscopy is not recommended as a first line screening test, however, it should be considered in patients with a history suggestive of endometriosis, previous pelvic inflammatory disease or previous pelvic surgery. Furthermore, if the hystero salphingography reports an

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abnormal result, verification should be carried out with diagnostic Laparoscopy. Some clinicians hold the view that to diagnose unexplained infertility, both peritoneal factor and endometriosis should be excluded, even in patients with normal hysteron-salphingography, by carrying out Laparoscopic examination.[7]

As a result, diagnostic Laparoscopy is a valuable technique and is a mandatory invasive investigation for complete assessment of female infertility in many clinics before the couple progresses to infertility treatment[1] and making a decision to go to assisted reproductive technology.[8]

On visual Laparoscopic inspection the appearances of the ovaries are suggestive of certain clinical condition.[1] Most ovarian abnormalities can be managed Laparoscopically and often a Laparoscopic examination of the adnexa will enable the gynecologist to decide if Laparotomy is indicated. Laparoscopy is an ideal procedure for diagnosing and staging of endometriosis because the magnification offered by the Laparoscope. [9] It is generally accepted that it is the gold standard in diagnosing tubal pathology and its etiology.

Every procedure has its own benefits or drawbacks but Laparoscopy provides a panoramic view of anatomy of pelvis and magnifies view of pelvic organs. It is generally accreted that diagnostic Laparoscopy is the gold standard in diagnosing tubal pathology and other intra-abdominal causes of infertility.[10] Diagnostic Laparoscopy is an endoscopic procedure and now it has become an indispensable tool while evaluating infertile patients. It is a relatively safe procedure with minimal morbidity.[11]

Materials and Methods:

This is a retrospective study done on 150 patients who underwent Laparoscopy during investigation for primary and secondary infertility. Laparoscopy were conducted between July 2015 to June 2016 in Obstetrics and Gynecology department of Combined Military Hospital, Dhaka.

Before Laparoscopy women had satisfied inclusion criteria: History of regular physical relation, clinical examination, hormonal assay, cervical smears, ultrasound report and semen analysis of husband.

After obtaining thorough history and detailed examination, patients were informed about the procedure and written informed consent was taken. Laparoscopy was performed in follicular phase of menstrual cycle under general anesthesia using the same method and the same principle in reporting the result. During the Laparoscopy, pelvis was inspected for any adhesions or endometriotic spots. Any structural abnormalities of uterus and tubes were noted and dye studies performed by injecting a dilute solution of methylene blue through cervix via Rubin's canula.

Statistical Analysis:

All data were entered and analyzed using SPSS venison 16. The variables were stratified. Quantitative variables like age and duration of infertility has been presented as mean. Qualitative variables like type of infertility, previous history, Laparoscopic findings of chromotubation, complication of Laparoscopy were presented us frequency and percentage.

Results:

Table 1: Distribution of infertility and types of Infertility by age group (N=150)

Age Group	No. of patients	Percentage
18-22	16	10.7
23-32	120	80
33-37	12	8
38-42	2	1.3
Total	**150**	**100**
Age Group	**Primary infertility No. (%)**	**Secondary infertility No. (%)**
18-22	14 (9.3)	2 (1.3)
23-32	78 (52.0)	42 (28)
33-37	2 (1.3)	10 (6.7)
38-42	0	2 (1.3)
Total	**94 (62.7%)**	**56 (37.3%)**

Table 1 shows most of the infertility cases 120 (80%) were in the age group of 23-32 years with mean age of 28 years. Only 2 cases (1.3%) were in 38-42 years age group. Majority of the patients with primary infertility (52%) and secondary infertility (28%) were in age group 23-32 years.

Table 2: Distribution of patients according to duration of infertility (N=150)

Duration (years)	No. of patients	Percentage
< 2 years	15	10
2-5 years	55	36.7
> 5 years	80	53.3
Total	**150**	**100**

Table 2 shows 80 (53.3%) patients had h/o infertility of > 5 years, 55(36.7%) patients had h/o infertility of 2-5 years and 15(10%) had h/o infertility <2 years. Mean duration of infertility was 4 years.

Table 3: Distribution of patients according to previous history of related factors (N=150)

Previous history	No. of patients	Percentage
Irregular menstrual cycle	60	40
H/O PID	20	13.3
H/O Endometriosis	20	13.3
H/O Previous Laparotomy	31	20.7
H/O Septic abortion	4	2.7
H/O D&C	15	10
Total	**150**	**100**

Table 3 shows 60 (40%) patients had h/o irregular menstrual cycle, 20 (13.3%) had h/o PID, 20 (13.3%) had h/o endometriosis, 31 (20.66%) had h/o previous laparotomy, 4 (2.66%) had h/o septic abortion and 15 (10%) had h/o D & C. There was no presence of multiple factors in one patient.

Table 4: Different findings associated with infertility on Laparoscopy (N=150)

Laparoscopic findings	No. of patients	Percentage
Normal pelvic organs	**22**	**14.7**
Abnormal tubo-peritoneal factors		
Extensive pelvic adhesion	16	10.7
Endometriosis	16	10.7
Features of Tuberculosis	6	4.0
Hydrosalpinx	6	4.0
Total	**44**	**29.4**
Ovarian pathology		
Polycystic Ovarian Diseased (PCOD)	62	41.3
Chocolate cyst	10	6.7
Simple cyst	6	4.0
Total	**78**	**52.0**
Uterine factors		
Congenital anomalies of uterus (bicornuate uterus, partially developed tube)	4	2.7
Fibroids	2	1.3
Total	**6**	**4.0**
Grand Total	**150**	**100**

Table 4 shows 22 (14.7%) patients had normal pelvic organ. Sixteen (10.7%) had endometriosis & extensive pelvic adhesion each, 6 (4%) had features of TB & hydrosalpinx each, 78 (52%) had ovarian pathology and 6 (4%) had uterine factor. Among ovarian pathology 62 (41.33%) had PCOD, 10 (6.66%) had chocolate cyst, 6 (4%) had simple cyst. Among uterine factor 4 (2.7%) had congenital anomaly of uterus and 2 (1.3%) patient had fibroid.

Table 5: Findings of chromotubation (N=150)

Tubal factors	No. of patients	Percentage
Tubal block		
Bilateral block	88	58.7
Unilateral block	12	8.0
Total	**100**	**66.7**
Peritubal adhesions (but spill seen)	**14**	**9.3**
Healthy & patent tubes	**36**	**24**
Grand Total	**150**	**100**

Table 5 shows findings of chromotubation. In this study total 100 (66.7%) patients had blocked tube while 36 (24%) patients had healthy and patent tube; 88 (58.7%) had bilateral block, 12 (8%) had unilateral block and in 14 (9.3%) patients there were peritoneal adhesions but spillage were seen.

Table 6: Frequency of tubal blockade detected in primary & secondary infertility

Type of infertility	No. of patients with blocked tubes	Percentage
Primary infertility	Unilateral 6 Bilateral 58	4.0 38.7
Total	64	42.7
Secondary infertility	Unilateral 6 Bilateral 30	4.0 20.0
Total	**36**	**24**

Table 6 shows 64 (42.7%) patients of primary infertility and 36 (24%) patients of secondary infertility had blocked tube. Bilateral block were more in both group 38.7% and 20% in primary and secondary infertility respectively. Unilateral block were same as 4% in each group.

Table 7: Complications of Laparoscopy

Complications of laparoscopy	No. of patients	Percentage
Pyrexia	12	8
Nausea & vomiting	6	4
Wound infection	2	1.3
Damage to bowel	0	0
Damage to blood vessels	0	0
Total	**20**	**13.3**

Table 7 shows total 20 (13.3%) patients had minor complications. Among them 12 (8%) developed pyrexia,

6 (4%) developed nausea and vomiting, 2 (1.3%) had wound infection. No patient had any major complication.

Discussion:

Laparoscopy is considered as a gold standard method in assessment of female factors of infertility. About 20 to 35 percent of infertile couples have some contribution of female factors in their infertility.[3] In our study, we have collected data at Combined Military Hospital, Dhaka, where huge number of infertility patients visit for their diagnosis and subsequent management. Laparoscopy is a mandatory procedure for full assessment of infertile couple.

In our study 62.7%, n =94, patients were suffering from primary infertility and 37.3%, n=56 patients with secondary infertility. A study by Shetty Sk. showed that there were 68% cases of primary infertility & 32% cases of secondary infertility.[12]

In a similar study, 65.7% were for primary infertility and 34.3% were for secondary infertility.[13]

In our study 16 patients were in age group of 18-22 years, 120 were between 23-32 years & 12 were 33-37 years of age and 2 patients were 38-42 years of age. Mean age of infertility was 28 years. These results were similar to study by Sajeda Parveen where mean age of patients was 28.4 year.[14]

About 53.3% patients with > 5 years of active married life followed by 36.7% with 2-5 years of active married life. A similar study showed that the maximum member (45.7%) of patients presented after 2-5 years of failure to conceive and 54.3% of patients had duration of infertility of more than 5 years.[13]

In our study 40%, n= 60 patients had some or other menstrual irregularity, 13.3%, n = 20 patients had history of PID, 13.3%, n = 20 patients had history of endometriosis, 20.66%, n=31 patients had some kind of Laparoscopy in their past, 2.66%, n=4 patients had H/O septic abortion & 10%, n=15 patients had underwent D&C. In a study conducted in Hyderabad (Pakistan) among all who presented with secondary infertility 16.6% had h/o pelvic pain and 16% had history of irregular cycle and 50% of patients who presented with primary infertility had previous D&C, while in secondary infertility 44% had previous LSCS.[15]

In our study, we observed a wide range of findings on Laparoscopy. In 14.7%, n = 22 cases, pelvic organs were found to be normal, which are the candidates of other investigations. The findings of other authors varied from 37.93% to 66.7% in case of primary infertility [16] & 14% to 52.6% in case of secondary infertility.[17]

Tubal block (66.7%, n =100) was the highest abnormality seen among infertility patients followed by ovarian factor 52%, n=78; endometriosis 10.6%, n=16, extensive pelvic adhesion 10.7%, n=16. hydrosalpinx 4%, n=6, Tuberculous salphingitis 4%, n=6. Congenital anomalies of uterus 2.6%, n=4 & uterine fibroid 1.3%, n=2, Our findings were corroborated by the study by Maheshwari A that the most common cause observed by Laparoscopy was tubal occlusion (26%), this was followed by polycystic ovaries (15.6%), endometriosis (12.5%) in case of primary infertility while peritubal and periovarian adhesions (22%) and PID (16.7%) were second most common causes in cases of secondary infertility.[18]

Polycystic ovary was the common findings among ovarian pathology 41.3%, n=62 followed by chocolate cyst 6.7%, n =10 & simple cyst of ovary 4%, n=6. It is somehow similar to Usmani et-al study.[19]

In study conducted by Mehmood incidence of endometriosis was 16.16%.[1]

Extensive pelvic adhesions was seen in 10.7% of cases which corresponds with other studies.[20,21]

In our study 2.7% n=4, patient was observed having congenital anomaly of uterus. [1] In a study, incidence of congenital tubal anomalies and hypoplasia of uterus and fallopian tube was about 3%.[1]

Uterine fibroids (1.3%) diagnosed in infertility patients in this study was much lower than in other studies where was seen in 7.14%19 & 15.15%[1,20] which could be explained by the difference in racial and environmental factors between the studies.

In our study out of 94 patients who presented with primary infertility 64 (42.7%) had blocked tubes. In patients with secondary infertility out of 56 patient 36 (24%) had blocked tubes.

Most tubal factor cases of primary and secondary infertility were diagnosed to have bilateral blockage (38.7%, n =58,20%,n=30) respectively. Our findings correspond with Mehmood study[1] and Usmani et-al study [19] where the tubal factor was the most common cause and constituted 35.85% and 37.6% of cases respectively while ovarian factor was seen in 32.83% and 26.08% of cases respectively.

Bilateral tubal blockage constituted 58.7% of patients with tubal blockage which corresponds with Vasijevic et-al study [22] where was seen in 50.94% of cases and lower than 78.57% seen among infertile Nigeria women.[20]

In this study, among patients with tubal blockage, it appears mainly due to pelvic inflammatory disease, endometriosis, tuberculosis which is seen in Jamal study.[23]

When Laparoscopic associated complication were noted, there were 12 (8%) patients who had pyrexia, 6 (4%) patients had nausea & vomiting, 2 (1.3%) had wound

infection and none of our patients had any damage to bowel and blood vessels.

In a large finish follow up study the complication rate of diagnostic Laparoscopy was 0.6 per 1000 procedures and the most common complication were pyrexia, shoulder tip pain, nausea and vomiting.[24]

Conclusion:

Laparoscopy is an effective diagnostic total for evaluation of pelvic pathologies and is a mandatory invasive investigation for complete assessment of female infertility. Laparoscopy and chromotubation test should be performed as a first step in investigation of infertile women especially those with history of PID and pelvic surgery. Laparoscopy not only help in identification of unsuspected pathology, but also help us in future decision making regarding treatment of infertile patients.

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Original Article

Histopathological Analysis of Five Years Specimen of Breast Lump in a Selected Medical College Hospital

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Abstract

Background: Various types of lesion from inflammation to carcinoma can affect the breast. Some lesions are common in young females while others are more common in elderly. Early presentation and prompt diagnosis is essential to relieve anxiety of non-neoplastic conditions, and in case of carcinoma, it can save the patient from metastases.

Objective: This study was done to assess the histopathological pattern of breast lumps among patients in a selected medical college hospital.

Methods: A retrospective cross-sectional study was conducted for the period of five years from 2012 to 2016 in the Pathology Department of Jahurul Islam Medical College and Hospital. Histopathology samples were received, processed, reported and recorded in the Pathology laboratory. Demographic and clinical data were collected from the hospital records. Data were analyzed by Microsoft Excel software. Descriptive statistics was used to present the variables.

Result: A total of 228 samples of breast tissue sent for histopathology were studied. Peak incidence of benign lesions was in between 21-30 years and malignant lesions in between 31-50 years. No breast lesions were seen in the first decade of life. Cancer of the breast was seen in 12.2% of cases. Among 228 cases majority were benign fibroadenoma (39.4%) and infiltrating ductal carcinoma was 7.9% which was highest among all malignant cases.

Conclusion: Majority of the breast lumps are benign either fibroadenoma or fibrocystic disease. Benign lesions were common in second to fourth decade and malignancy in fourth and fifth decades. Therefore self examination of breast for screening specially for young adult women and periodic mamogram after 40 years of age are universally recommended for early detection and treatment of any kind of breast diseases.

Keywords: Breast lump, Carcinoma, Fibroadenoma, Biopsy.

Introduction:

The human breast is paired mammary glands composed of specialized epithelium and stroma in which both benign and malignant lesions can occur. Benign breast diseases (BBD) however constitute the greater of the breast lesions.[1] These BBD are diverse, ranging from disorders of development, inflammatory lesions, proliferative diseases of the epithelium and stroma to different types of neoplasms.[2] Though most of the available literature shows that breast lumps are mostly benign and non proliferative epithelial lesions, it is known that certain benign breast diseases (BBD) are important risk factors for breast cancers which can develop in either breast later.[3] Breast cancer is one of the commonest cancers among women and commonly presents with a lump in breast to the physician. It is related to morbidity and mortality worldwide among women. In Asia, the incidence of breast cancer is increasing and may occur in younger age group. About 25% of breast cancer occurs in younger patients in developing Asian countries as compared to developed Asian or Western countries.[4] As breast lump can be the cause of different benign and malignant lesions, the management of the patients varies. Though clinical examination of the breast lump and the age of the patient can provide information about the nature of the lump, histopathological examination is necessary to establish the diagnosis. The aim of the present study is to see the spectrum of conditions/ lesions in breast lump specimens in a selected medical college hospital situated at a rural and partially semi-urban environment.

Materials and Methods:

This is a retrospective cross sectional study of breast tissue specimen received from 2012 to 2016 at the Department of Pathology, Jahurul Islam Medical College and Hospital. The specimens were labelled, entered in the data system of the lab and kept for fixation in 10% Formalin overnight. After grossing, it was processed in the tissue processor, made up to blocks and cut into sections of 0.5 micron

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thickness. After staining with hematoxylin and eosin, slides were examined by pathologists. All the findings were recorded in the database. All the original request forms and histopathological reports on the breast specimens received within this study period with their slides were retrieved from the archives and reviewed. From the request forms and histopathological reports, information on age, sex, nature of specimen, hospital numbers, laboratory numbers and histopathological diagnosis were extracted. New slides were made from formalin fixed, paraffin-embedded tissue blocks and stained with Haematoxylin and Eosin (H&E) where necessary for appropriate diagnosis and classification. Male breast tissues, cases of breast lesions with incomplete data and cases unable to trace slides or blocks were excluded from the study.

Statistical Analysis:

Microsoft Excel software was used to generate tables. The descriptive statistics were used to infer results.

Results:

A total of 228 breast tissue specimens were examined in the five years period, which were around 6.5% of the total specimens received for histopathological examination in this hospital.

Table 1: Age distribution of patients of breast tissue specimen (n=228)

Age group (in years)	Number of cases	Percentage
11-20	18	7.9
21-30	66	28.9
31-40	88	38.6
41-50	38	16.7
51-60	12	5.2
61-70	06	2.6
Total	228	100

The age of the cases ranged from 11 to 70 years. Most of the patients were in the age group 31 to 40 years group (38.6%) as shown in Table 1.

Table 2: Type of presenting complain of the patients (n=228)

Presenting complain to Physician	No. of cases	Percentage
Lump	78	34.2
Pain	67	29.3
Tenderness	40	17.5
Lumpiness with heaviness	25	10.9
Skin redness with rash	10	4.4
Pain in the axilla and hand	08	3.5
Total	228	100

The presenting complain of the patients coming to the hospital was feeling of lump in 78 (34.2%) cases, pain in the breast 67(29.3%) cases, tenderness 40 (17.5%) cases, feeling of heaviness in the breast 25 (10.9%) cases as shown in Table-2.

Table 3: Distribution of benign and malignant breast lesion by age group (n=228)

Age group (in years)	Benign lesions	Malignant lesions
11-20	18	0
21-30	64	02
31-40	80	08
41-50	28	10
51-60	08	04
61-70	02	04
Total	200 (87.7%)	28 (12.2%)

The average age of the patients' was around 34 years. The average age for benign and malignant breast diseases were found as 30 years and 52 years respectively. The benign lesions and malignant lesions were most common in the age group of 31-40 years and 41-50 years respectively. Benign breast lesion were 87.7% and malignant cases were 12.2%. The ratio between benign and malignant cases is 7:1 as shown in Table-3.

Table 4: Histopathological diagnosis of breast lump (n=228)

Histopathological Finding	Number of cases	Percentage
Fibroadenoma	90	39.4
Fibrocystic disease	42	18.4
Breast abscess	36	15.9
Duct ectasia	10	4.3
Granulomatous lesion	06	2.6
Intraductal papilloma	06	2.6
Fat necrosis	08	4.4
In-situ carcinoma (DCIS)	06	2.6
Invasive ductal carcinoma	18	7.9
Invasive lobular carcinoma	04	1.7
Total	228	100

Among histopathological diagnosis, majority benign cases were fibroadenoma 90 (39.4%), followed by fibrocystic diseases 42 (18.4%), and breast abscess 36 (15.9%). Other benign lesions included duct ectasia 10 (4.3%), granulomatous lesion 06 (2.6%), fat necrosis 10 (4.4%) and intraductal papilloma in 06 (2.6%) cases. The carcinoma cases were found in 12.2% cases. There were 18 (7.9%) cases of invasive ductal carcinoma, 04 (1.7%) cases of Invasive lobular carcinoma. In situ carcinoma (DCIS) was found in 06 (2.6%) cases as shown in Table-4.

Discussion:

The average number of breast tissue specimens received (6.5%) in our study is almost similar to that shown by Singh and Thakur (2.3%).[5] The peak incidence of benign lump was found in 21 to 30 years age group and peak incidence of malignant lumps 31 to 50 years which is younger compared to the study of Prakash S.[6] No breast tumors were seen in the first decade of life. The youngest patient in this study was 14 years similar to that seen in other parts of Nepal.[7] The rarity of breast disease in the first decade of life is also reported by others.[8] Most common complain of the patients of breast tissue specimen was lump (34.2%), pain (29.3%) and tenderness (17.5%) similar to other study.[9]

Fibroadenoma (39.4%) followed by fibrocystic disease (18.4%) formed the majority of breast lesions sent for histopathology, which is similar to that seen by Khanna et al. from Banaras- India.[10] Singh and Thakur in their study showed similar incidence as 28.28% and 21.71% respectively for fibroadenoma and fibrocystic changes.[5] The real incidence of fibrocystic disease is difficult to estimate and diagnosis depends a great deal on individual clinician or pathologist acumen. Ten (4.3%) cases of duct ectasia were present in this study. Duct ectasia of the breast (or mammary duct ectasia) is a condition in which there is an obstruction of the lactiferous duct. Mammary duct ectasia can mimic breast cancer. It is a disorder of premenopausal age. Signs of duct ectasia can include nipple retraction, inversion, pain, and sometimes bloody discharge.[11] Microglandular adenosis is widely known as a benign breast lesion that can produce a mass. The main importance of this lesion is that it is usually considered as a precursor for malignancy. Four (1.75%) of breast lesions in our study was diagnosed as microglandular adenosis.[12] The benign to malignant ratio was 3:1 in a study in Calcutta and 7:1 in our study. In that study the percentage of malignancy was higher (24.44%) as compared to our Study.[4] Benign lesions were common in the second to fourth decade and malignant lesion in fourth and fifth decades, which is similar to that seen in other parts of the world.[13] Eight cases of traumatic fat necrosis and six case of granulomatous lesion were also found in our study.

Cancer was seen in 12.28% of our cases. Singh and Thakur found the incidence of cancer as 18.42%.[5] The percentage of carcinoma in this study appears to be slightly closer to the west (10.5%) and lower than that of Africa (21%).[14] Among the cases of breast carcinoma, Invasive ductal carcinoma was the commonest malignancy seen (7.89%) in our study. Singh and Thakur[5] in their study found invasive ductal carcinoma in 18.48% cases which is similar to that reported by Ali et al[15] and is higher than the present study. There were 6 cases of In-situ carcinoma (DCIS), 4 cases of lobular carcinoma and 18 cases of invasive ductal carcinoma in our study. Prakash et al. reported the incidence of malignancy as 2.5% for age group 30 years and below and 97.5% for age group above 30 years. She therefore pointed out the necessity of investigating all patients with breast lumps to rule out malignancy especially in women above 30 years.[6]

Conclusion:

Majority of the breast lumps are benign either fibroadenoma or fibrocystic disease. Benign lesions were common in second to fourth decade and malignancy in fourth and fifth decades. Ductal carcinoma is the commonest sub-type in this study. It is thus recommended that all women above the age of 40 presenting with a palpable breast lump or a suspicious non-palpable abnormality on screening mammogram to have their lump excised. However, women below 30 years should also have the lump excised in the presence of risk factors such as a family history of breast cancer.

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Original Article

Serum Magnesium in Gestational Diabetes Mellitus: A Cross-sectional Comparative Study

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Abstract

Background: Gestational Diabetes Mellitus (GDM) is associated with perinatal mortality, stillbirth, neonatal & intrauterine death, prematurity and congenital malformations. There is a greater risk of developing recurrent GDM in subsequent pregnancy. Magnesium (Mg) deficiency may interfere carbohydrate metabolism at different level by impairing the action of insulin.

Objective: The objective of the study was to assess the serum Mg level and its association with GDM .

Methodology: This cross sectional comparative study was conducted in the Department of Biochemistry, Dhaka Medical College, Dhaka for a period of one year. Total 50 cases were selected purposively. In Group-I there were 30 GDM cases and in Group-II there were 20 uncomplicated normal pregnant women. GDM patients were diagnosed by OGTT (Oral Glucose Tolerance Test) according to WHO criteria & subsequently measurement of serum Mg level was performed in all cases. Unpaired "t" test and correlation were done to determine the statistical association between serum Mg level and GDM.

Results: Among 50 cases 30 were GDM (Group-I) and 20 were uncomplicated normal pregnancy (Group-II). Mean serum Mg concentration of Group-I was 0.71 0.10 mmol/L and Group-II value was 0.77 0.10 mmol/L and significant association was found between Mean serum Mg concentration and GDM ($p<0.05$). In Group-I, mean serum concentration of Mg (0.71 mmol/L) was inversely correlated with mean of fasting plasma glucose (8.3mmol/L) at $p<0.05$ level when 'r' value was - 0.577.

Conclusion: Hypomagnesaemia might be a risk factor for GDM.

Keywords: GDM (Gestational Diabetes Mellitus), Magnesium (Mg), Insulin receptor substrate (IRS), OGTT (Oral Glucose Tolerance Test)

Introduction:

Gestational diabetes mellitus (GDM) is glucose intolerance of variable severity developed first ever during pregnancy. GDM is associated with perinatal mortality, stillbirth, neonatal & intrauterine death, prematurity and congenital malformations. Birth trauma is more common due to macrosomic babies. There is a greater risk of developing recurrent GDM in subsequent pregnancy. So, it is imperative to take care of mother's and the baby's health.[1] Insulin is a peptide hormone secreted by cell of pancreas. For performing action, insulin binds with specific insulin receptor in the cell membrane of the most tissues. Insulin receptor has two subunits and . First of all insulin binds with -subunit, which causes autophosphorylation of tyrosine kinase of -subunit. This phosphorylated tyrosine-kinase promotes phosophorylation of a peptide known as insulin receptor substrate (IRS). Then phosophorylated IRS ultimately interacts with a number of intracellular proteins and provokes a wide range of biological action of insulin.[2] Magnesium is one of the most prevalent intracellular cation. It has got a role in the mechanism of insulin action. It activates phosophorylation of tyrosine kinase of insulin receptor.[3] Normal serum Mg range between (0.7-1.05 mmol/l). Magnesium is a critical co-factor for a multitude of enzymatic reactions that are important for the generation of energy from ATP. Cellular magnesium deficiency can alter the activity of glucose transport. This defective glucose transport and receptor activity may ultimately influence the intracellular signaling and processing of insulin responsible for the impairment of insulin action.[4] Mg deficiency thus may interfere carbohydrate metabolism at different level by impairing the action of insulin. Experimental studies in animal and cross-sectional studies in humans have suggested that low Mg intake play a role in the development of diabetes and in the development of insulin resistance. Magnesium supplementation has a beneficial

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effect on insulin action and glucose metabolism.[5] Although exact pathophysiological mechanism of GDM is not yet fully established but impaired insulin action is associated with development of GDM. Principal source of Mg is diet. Diets rich in magnesium are- green leafy vegetables, unrefined grains, citrus fruit, other vegetables and fishes. Meat is a poor source. So due to modern dietary habits and ignorance, pregnant women may have magnesium deficiency before pregnancy. Increased need during pregnancy can aggravate the deficiency leading to GDM. As Mg rich foods are easily available in our country and they are not much expensive, childbearing and pregnant women can be encouraged to take Mg rich diets or Mg supplements to prevent GDM. Therefore the purpose of the present study was designed to explore the association between serum Mg level and GDM in Bangladeshi women.

Methodology:

This cross sectional comparative study was conducted in the Department of Biochemistry, Dhaka Medical College, Dhaka. Total 50 subjects with no past history and family history of diabetes participated in this study. Serum Mg of GDM cases were measured and compared with serum Mg in uncomplicated normal pregnancy**.** GDM patients were diagnosed as diabetic by OGTT according to WHO criteria. The subjects did not start anti-diabetic therapy and patients were selected from Gynae outpatient department of BIRDEM by purposive sampling. Uncomplicated normal pregnant women with normal blood glucose level were selected from antenatal check-up room of DMCH. Obese, hypertensive & pre eclamptic patients were excluded in this study. Permission for the study was taken from relevant authority. Informed written consent was taken from study subjects. Data were collected through a preformed and pretested questionnaire. BMI was calculated by the recorded weight during 1st antenatal check-up in 1st trimester. Fasting blood amounting of 10 ml was collected and 3 ml of blood was transferred in a test tube containing anticoagulant for the measurement of plasma glucose. Rest of the blood was taken in another test tube. Serum was separated by centrifugation and stored at 2-8^{0}C for maximum two days before analysis. Serum was analyzed for magnesium. Data were analyzed using SPSS software and MS Excel of Office 2000. Mean values of serum Mg of two groups were compared by unpaired't' test. 95% confidence limit ($p<0.05$) was taken as level of significance.

Results:

Table 1: Grouping of Study Subjects (n=50)

Groups	Status	No. of cases
Group I	GDM	30
Group II	Uncomplicated normal pregnancy	20

Among 50 subjects 30 were GDM (Group I) and 20 were uncomplicated normal pregnancy (Group II) cases as shown in Table 1.

Table 2: Fasting plasma glucose concentration of study subjects

Groups	Number of subjects (n)	Plasma glucose concentration (mmol/L)	
		Mean ±SD	Range
Group I (GDM)	30	8.3±0.34	8.3-0.34
Group II (Uncomplicated Normal pregnancy)	20	4.5±0.56	4.5-0.56

In Group I (GDM), mean SD value of plasma glucose concentration was 8.3 0.34 mmol/L with range 8.0-8.7 mmol/L, in Group II (Uncomplicated normal pregnancy) mean SD value was 4.5 0.56 mmol/L with range 3.9-5.0 mmol/L as shown in Table-2.

Table 3: Serum magnesium concentration of study subjects

Groups	Number of subjects (n)	Serum Mg concentration (mmol/L)	
		Mean ±SD	Range
Group I (GDM)	30	0.71±0.10	0.60-0.81
Group II (Uncomplicated Normal pregnancy)	20	0.77±0.10	0.66-0.87

Mean serum Mg concentration of Group-I (GDM) was 0.71 0.10 mmol/L with range 0.60-0.81 mmol/L and in Group-II (Uncomplicated normal pregnancy) value was 0.77 0.10 mmol/L with range 0.66-0.87 mmol/L as shown in Table-3 .

Table 4: Comparison of serum Mg concentration between Group-I and Group-II

Parameter	Group I (GDM) n=30	Group II (Uncomplicated normal pregnancy) n=20	Level of Significance ('p' value)
Serum Mg (mmol/L	0.71±0.10	0.77±0.10	<0.05*

Unpaired't' test done * Significant

To find out the statistical significance, mean values of serum Mg concentration between group I and group II were compared and it was observed that Group I (GDM) showed significantly lower ($p<0.05$) level of serum Mg compared to group II (Uncomplicated normal pregnant women) as shown in Table 4.

Table 5: Correlation of fasting serum Mg with fasting plasma glucose level in GDM cases

Fasting plasma glucose (mmol/L) Mean±SD	Fasting serum Mg (mmol/L) Mean±SD	'r' value	'p' value
8.3±0.34	0.71±0.10	-0.577	<0.05*

* Significant

Table 5 showed correlation of fasting serum Mg with fasting plasma glucose level in group I (GDM), where mean serum concentration of Mg (0.71 mmol/L) was inversely correlated with mean plasma glucose (8.3 mmol/L) at $p<0.05$ level when 'r' value was - 0.577

Discussion:

In this cross sectional comparative study we had measured the serum Mg and fasting plasma glucose in GDM cases and uncomplicated normal pregnant women to assess the association of serum magnesium with GDM. Mean serum Mg concentration found in group I (GDM) was 0.71 0.10 mmol/L with the range 0.60-0.81 mmol/L and in group II (uncomplicated normal pregnant women) mean was 0.77 0.10 mmol/L with the range 0.66-0.87 mmol/L. GDM patients of this study hade shown serum Mg concentration significantly lower in comparison to uncomplicated normal pregnant women. Same phenomenon was observed in many other studies around the world.[6,7]

The reasons why low Mg is common in GDM patients are not clear but may include lower dietary intake, increased demand during pregnancy, increased urinary loss, impaired absorption and treatment with diuretics.[8] Magnesium is involved in glucose metabolism as it is involved in phosphorylation and is a cofactor for ATPase & adenylate cyclase enzyme.[9] It is required for both proper glucose utilization and insulin signaling. The suppressed magnesium concentration may result in defective tyrosine kinase activity and modify insulin sensitivity. Magnesium deficiency may affect the development of insulin resistance and alter the glucose entry into the cell.[10] Low serum Mg is the common finding of GDM & decreased insulin sensitivity or insulin resistance (IR) is the underlying pathophysiology of GDM.[11] Reports of some studies however pointed out some doubts regarding the association between serum Mg & glucose metabolism. They found low Mg in diabetes but failed to show significant correlation between them.[12] Hypomagnesaemia can be consequence rather than cause of hyperglycemia as seen in increase urinary Mg excretion with glycosuria.[13] But several other studies showed that though diabetes can induce decreased serum Mg but Mg deficiency can also be a risk factor for GDM.[14,15]

In the study serum magnesium level had significant inverse correlation with plasma glucose level (r value -0.577) in GDM patients. This finding indicates that serum Mg is a predictive risk factor of GDM. This result is consistent with the other studies.[5,9] A number of prospective study suggested that diet supplementation with Mg significantly reduces risk of diabetes and its complication by improving insulin sensitivity.[16]

Conclusion:

The prevalence of GDM is increasing in developing country like Bangladesh. It has got some well-established risk factors. Decreased serum magnesium has been claimed as a risk factor of GDM as decreased serum magnesium can reduce insulin sensitivity. In light of above mentioned results and discussion it can be concluded that hypomagnesaemia might be a risk factor for GDM. However prospective study to find out the effect of Mg supplementation in reversing GDM by decreasing blood glucose level can be carried out.

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Original Article

Histological Scoring for Nonalcoholic Fatty Liver Disease- A study of 55 Cases

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Abstract

Background: Steatohepatitis is a pattern of liver injury that may be seen in alcoholic or nonalcoholic liver disease. This morphological changes may occur with obesity, diabetes, the use of certain drugs or it may be idiopathic. The main histopathological features of nonalcoholic steatohepatitis (NASH) include hepatocellular steatosis and ballooning, mixed acute and chronic lobular inflammation and fibrosis. The recently developed histological scoring system for nonalcoholic fatty liver disease (NAFLD) by the NASH Clinical Research Network (NASH CRN) is becoming increasingly popular and followed for histopathological diagnosis

Objective: This study was done to analyze the histological spectrum and to develop grading and staging system in patients with nonalcoholic fatty liver disease.

Methods: This cross sectional study was carried out at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh. A total of 55 patients with non-alcoholic fatty liver disease (NAFLD) who fulfilled the inclusion criteria were selected for the study. Liver biopsy was done at hepatology department of BSMMU, and different clinics of the Dhaka City for a period of one year. The data of patients along with histopathological reports were studied.

Results: Out of total 55 cases of nonalcoholic fatty liver diseases 33 were male (60%) and 22 were female (40%). Mean age of the patients was 37.81±10.80 years. Among 55 patients of NAFLD, NAFL (steatosis) were diagnosed in 6 patients (10.90%) and NASH in 49 patients (89.1%). Histopathological findings revealed that among 46 definite NASH cases all (100%) had steatosis and hepatocyte ballooning, 97.8% and 91.3% had lobular inflammation and fibrosis respectively. NAS in majority for steatosis was score 3 (50.90%), for lobular Inflammation score 2 (58.18%) and for hepatocyte ballooning score 2 (89.1%). About 91.3% of the 46 Definite NASH biopsy specimens exhibited fibrosis and 6.52% had cirrhosis in M. T stain.

Conclusion: Grading and staging of NAFLD are to be considered as a very useful diagnostic tool. It is also important for dealing with the management of NAFLD and follow up later on.

Keywords: Nonalcoholic fatty liver, Steatohepatitis, Fibrosis, Cirrhosis, Nonalcoholic activity score.

Introduction:

Nonalcoholic fatty liver disease (NAFLD) is a morphological pattern of liver injury due to fat accumulation within hepatocytes in the absence of significant alcohol use or other known liver diseases. NAFLD encompasses a wide spectrum of liver diseases ranging from simple fatty liver (steatosis) to non-alcoholic steatohepatitis (NASH) and eventually cirrhosis (irreversible advanced scarring of the liver).[1]

Non-alcoholic fatty liver disease (steatosis) refers to accumulation of fat within liver hepatocytes that usually causes no liver damage. Non-alcoholic steatohepatitis (NASH) on the other hand is associated with accumulation of fat in the liver cells as well as inflammation of the liver. NASH can ultimately progress to cirrhosis or to hepatocellular carcinoma.[2]

In Japan, and China NAFLD is now recognized as one of the common liver disease.[3] In a small epidemiological study in India among healthy controls, prevalence of fatty liver in absence of alcohol intake was found to be 9-32%.[4]

The grading and staging approach have been applied to asses the severity of fatty liver disease. According to The Pathology Committee of the NASH Clinical Research Network a NAFLD activity score (NAS) system that addresses the full spectrum of lesions of NAFLD and proposed a NAFLD activity score (NAS) for use in clinical trials. Four features were evaluated semi-quantitatively: Steatosis (0-3), Lobular inflammation, (0-2), Hepatocellular ballooning (0-2), and Fibrosis (0-4)

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(Kleiner et al 2005). The individual scores then added to produce an overall "NAFLD Activity Score" (NAS).[5]

There is also recognition of portal fibrosis as a separate pathway for disease progression.

With this view the present study was carried out to determine scoring which includes probable or definite NASH (score =5), Uncertain (score 3-4) and Not NASH (= 2). The cases were collected from different institutes and pathologic laboratories of Dhaka city.

Materials and Methods:

This is a cross sectional study carried out at the Department of Pathology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka for a period of one year. A total of 55 patients were selected for the study. These patients were clinically, biochemically and sonologically diagnosed as suffering from non-alcoholic fatty liver disease. Liver biopsy was done at hepatology department of BSMMU and different clinics of the Dhaka City. Histopathological evaluation of liver biopsy was done to see the lobular architecture, hepatic cell plate, steatosis, lobular inflammation, hepatocyte ballooning and fibrosis.

Grading:

Individual scores of steatosis, lobular inflammation and hepatocyte ballooning added to produce an overall NAFLD Activity Score: (NAS). NAFLD activity score is =5 then the histological diagnosis will be Probable or Definite NASH; 3-4 is Uncertain and = 2- Not NASH.

Staging:

Staging was used to evaluate the intensity of necro-inflammatory activity and extent of fibrosis.

The staging score is as below;

Stage I : Perisinusoidal or periportal fibrosis which is further divided into as;

IA- Mild, zone 3, perisinusoidal;

IB-Moderate, zone 3, perisinusoidal and

IC- Portal/periportal fibrosis only.

Stage II : Perisinusoidal and portal/periportal fibrosis;

Stage III : Bridging fibrosis;

Stage IV : Cirrhosis.

Fibrosis scores for Stage-I were extended to include a distinction between delicate (Stage IA) and dense (Stage IB) perisinusoidal fibrosis and to detect portal-only fibrosis, without perisinusoidal fibrosis (Stage IC).

Results:

Table 1: Age and sex distribution of NAFLD cases (n=55)

Age (years)	Male		Female		Total (%)
	NAFL No.	NASH No.	NAFL No.	NASH No.	
10-20	1	2	0	0	3 (5.45)
21-30	0	8	1	3	12 (21.81)
31-40	4	10	0	6	20 (36.36)
41-50	0	6	0	10	16 (29.09)
51-60	0	2	0	1	3 (5.45)
61-70	0	0	0	1	1 (1.81)
Grand Total	**5**	**28**	**1**	**21**	**55 (100)**

Table 1 shows that total of 55 cases were included of whom 33 were male (60%) and 22 were female (40%). Among 55 patients NAFL (steatosis) were diagnosed in 6 patients (10.90%) and NASH in 49 patients (89.1%). Most of the cases (36.36%, n=20) of NAFLD were in age group 31-40 years, followed by 41-50 years (29.09%, n=16). Mean age of the patients was 37.81±10.80 years.

Table 2: Histological parameter of NASH

Histological Diagnosis		Histological Parameter			
		Steatosis No. (%)	Lobular Inflammation No. (%)	Hepatocyte ballooning No. (%)	Fibrosis No. (%)
NAFL (N=6)	Definite NASH (N=46)	6(100)	0	0	0
NASH		46(100)	45(97.8)	46(100)	42(91.3)
	Uncertain NASH (N=4)	2(50)	3(75)	2(50)	3(75)
	No NASH (N=5)	5(100)	0	0	0

Liver biopsy was done in all fatty liver patients. Among 55 patients NASH was diagnosed in 49 patients (89.1%) and NAFL (steatosis) in 6 patients (10.90%). Among 46 definite NASH cases all had steatosis and hepatocyte ballooning, 97.8% and 91.33% had lobular inflammation and fibrosis respectively.

Table 3: Histological scoring for NAFLD cases (n=55)

NAS-score Component	NAS score	
	No.	%
Steatosis		
0=<5%	02	3.64
1=5%-33%	09	16..36
2=33%-66%	16	29.09
3=>66%	28	50.90
Lobular Inflammation		
0=none	06	10.90
1=<2 foci per x 200 field	15	27.27
2=2-4 foci per x 200 field	32	58.18
3=>4 foci per x 200 field	02	4.34
Ballooning		
0=none	06	10.9
1=Few ballooned cells	0	0
2=many cells/Prominent balloning	49	89.1

Steatosis: Scoring was done more precisely for steatosis. Among 55 NAFLD cases majority had score 3 (50.90%, n-28), followed by score 2 (29.09%, n=16).

Lobular Inflammation: Majority had score 2 (58.18%, n=32), followed by score 1 (27.27%, n=15).

Hepatocyte ballooning: About 89.1% (n-49) had score 2 and only 6 cases had 0 score as shown in Table 3.

Table 4: Distribution of Fibrosis Stage

Fibrosis Component in Staging	As scored in H & E stain	As scored in M. T stain
	No. (%)	No. (%)
Stage I: Perisinusoidal or periportal		
IA. Mild, zone 3,perisinusoidal	0	4 (8.69)
IB. Moderate, Zone 3,perisinusoidal	0	1(2.17)
IC. Portal/periportal fibrosis only	23 (50)	5 (10.86)
Stage II: Perisinusoidal & portal/periportal Fibrosis	1 (2.17)	26 (56.52)
Stage III: Bridging fibrosis	4 (8.69)	6 (13.04)
Stage IV: Cirrhosis	2 (4.34)	3 (6.52)

Fibrosis & Cirrosis: Fibrosis was evaluated by H & E and M.T stains. Total 42 of the 46 definite biopsy specimens exhibited fibrosis (91.3%) stage I to stage III. Stage IV (Cirrhosis) was seen in 6.52% in M.T stains as shown in Table 4.

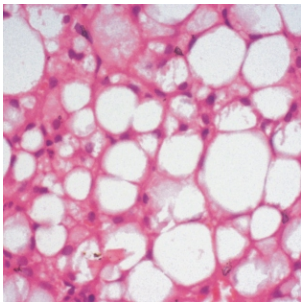

Fig. 1: Macrovesicular steatosis (H & E stain)

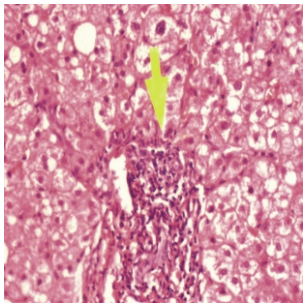

Fig. 2: Lobular Inflammation (H & E stain)

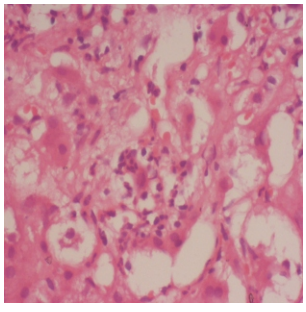

Fig. 3: Hepatocyte ballooning (H & E stain)

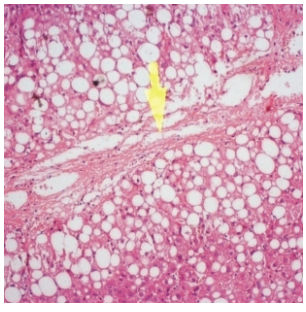

Fig. 4a: Bridging Fibrosis (H & E Stain)

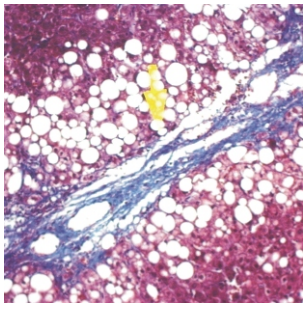

Fig. 4b: Bridging Fibrosis (M.T Stain)

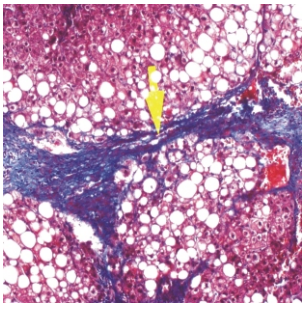

Fig. 5: Cirrhosis (M.T stain)

Discussion:

There has been great interest and anxiety regarding non-alcoholic fatty liver disease (NAFLD). It is a growing problem in the Western hemisphere. In USA the prevalence among general population is 20%.[6] Awareness about NAFLD is increasing in the Asia-Pacific region. A prevalence of NAFLD in this area has been estimated ranging from 20-30%.[7] NAFLD represents a spectrum of clinico-pathologic syndromes and represents with varying risk of cirrhosis.[8] Bangladesh is not excluded from this crisis. The aim of the present study was to evaluate the histomorphological features of NAFLD with grading and staging of the disease. A total of 55 NAFLD cases were selected on the basis of clinical, biochemical and sonological features of the liver disease. Among 55 patients, NAFL (simple steatosis) was diagnosed in 6 (10.9%) and

NASH in 49 (89.1%) cases. In developing countries like Bangladesh the incidental finding of NAFLD is increasing because of change in life style, food habit, and lack of consciousness. In Bangladesh many children and adolescents with suspected liver disease are seen by hepatologists. A significant number of cases of "cryptogenic cirrhosis" are found which may represent end stage of NAFLD.[9]

During the 12 month of the study, the subject's liver biopsy specimens were submitted to the laboratory as "routine" cases were not specified as "study biopsies" to avoid possible biased interpretation. One Hematoxylin & Eosin stained slide and one M. T' stained slide were studied. Different studies when reporting the histologic findings in patients with NASH, only steatosis was universally present as a diagnostic features. In the present study out of 55 liver biopsy specimens all i.e. 100% biopsies had steatosis. Steatosis as part of NAFLD is usually macrovesicular in type, perivenular in distribution. Steatosis are scored according to NAS score, modified by Kleiner *et al* in 2005.[5] In the present study 2 (3.64%) biopsies reveal steatosis score 0 (<5% hepatocytes), score 1 (5%- 33% hepatocytes) in 9 (16.36.%) biopsies, score 2 (33-66% hepatocytes) in 16 (29.09%) biopsies and score 3 (>66% hepatocytes) in 20 (50.90%) biopsies (Table 3).In the present study at BSMMU, lobular inflammation was present in all but 6 (10.90%) patients. Fifteen (27.27%) biopsies had score 1, 32 (58.18%) had score 2 and 2 (4.34%) had score 3 (Table 3). In a similar study it was observed that lobular inflammation was identified in most patients and score 2 was present in more than half biopsies.[10]

Regarding ballooning score 1 was not found in this study. Score 2 was seen in 49 (89.09%) patients. Ballooning was not seen in 6 (10.90%) patients (Table 3). In a similar study ballooning was seen in 100 biopsies.[11] A hallmark of NASH is the development of a specific form of fibrosis where a collagenous matrix is deposited along the hepatocytes in the space of Disse. In the most prominent cases, individual hepatocytes appear to be outlined by a rim of collagen, giving the liver a chicken wire appearance.

In present study 42 of the 46 definite NASH patients exhibited fibrosis. When using Masson's Trichrome stained slides, scored stage 1A was 8.69% (n-4), stage IB was 1.8% (n=1), stage IC was 10.86% (n=5) and stage 2 was seen in 56.52% (n-26). But in H & E stain these score were evaluated as stage 0 or IC. Stage III was 13.04% (n-6) in M.T stain and in H & E stain they were evaluated as either 0, 1C or 2. Stage IV was 6.52% (n=3). In H&E staining stage 4 was 4.34% (n=2). But cirrhosis was 6.52% (n=3) as revealed in Table 4. In the study by Brunt *et al* 2004 19 of the 30 biopsy specimens exhibited fibrosis (63%), scored as stage I in 7 (23%), stage II in 4 (13%) and stage III in 8 (27%).[11]

Conclusion:

NAS is useful for diagnosing NAFLD with grading and staging of histological features. The system is simple and require only routine histochemical stains (H & E and M. T stain). From the present study it was concluded that M. T stain is required for detection of collagen deposition in NAFLD cases. The staging in M.T stain has more predictive value in comparison to staging in H&E stain. The clinical, biochemical and sonological findings may me correlated with the histopathological findings for evaluation of disease for effective management of NAFLD and in its subsequent follow up.

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Review Article

Subclinical Hypothyroidism-a Comprehensive Overview

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Abstract

Subclinical hypothyroidism is biochemically defined as an elevated serum thyroid stimulating hormone (TSH) level in combination with a serum free T4 level that is within the reference range. The relationship between serum TSH& free T4 is such that a small decrease in free T4 can result in a relatively large increase in serum TSH level that is above the reference range, while the free T4 level is still within the reference range. In case of progression to overt hypothyroidism, the TSH level typically continues to increase and the free T4 level falls below the reference range. In this respect subclinical hypothyroidism can be seen as a mild form of thyroid failure that is caused by autoimmune thyroid disease in the majority of cases. Approximately 75% of patients with subclinical hypothyroidism have a TSH level of less than 10mlU per liter.

Keywords: Subclinical Hypothyroidism, Thyroxine, Thyroid stimulating hormone.

Introduction:

The incidence of sub-clinical hypothyroidism varies among populations and ranges from 3 to 15% with a higher incidence associated with increasing age, female gender and a suboptimal iodine status.[1,2] A TSH cutoff level of 10 mlU per liter is continuously used to distinguish between mild and more severe subclinical hypothyroidism.[3,4,5]

Subclinical hypothyroidism is defined biochemically as a normal serum free Thyroxine (T4) concentration in the presence of an elevated serum TSH concentration. Some patients with subclinical hypothyroidism may have vague, non-specific symptoms suggestive of hypothyroidism, but attempts to identify patients clinically have not been successful.[6,7] Thus, this disorder can only be diagnosed on the basis of laboratory test results. This topic will review the diagnosis and management of subclinical hypothyroidism. The clinical manifestations, diagnosis and management of overt hypothyroidism, as well as subclinical & overt hypothyroidism during pregnancy are reviewed separately.

Subclinical hypothyroidism, also referred to as mild hypothyroid failure, is diagnosed when peripheral thyroid hormone levels are within the normal range, but thyroid stimulating hormone is mildly elevated. It is common, occurring in 3-8% of the population and carries a risk of progression to overt hypothyroidism of 2-5 % per year. There is no absolute consensus on which patients to treat, although there are some clear recommendations. Estimation of serum TSH is both specific & sensitive test for thyroid disease. However, its sensitivity causes a dilemma, as some patients are found to have elevated TSH levels and may also be asymptomatic. Most of the circulating T3 is generated by peripheral conversion from T4, mainly by the liver through enzymatic removal of an iodine atom from T4. Very little T3 is produced by the thyroid gland itself.

Studies from different countries have shown upward trend in the prevalence of subclinical hypothyroidism. Nutrition plays a critical role in thyroid hormone synthesis.[8,9,10] The production of thyroid hormones depend on adequate amounts of tyrosine, a non-essential amino acid synthesized in the body from the essential amino acid phenyl alanine and dietary iodine. Iodine deficiency is the most common cause of hypothyroidism worldwide. We get iodine from food and is found in commercially processed iodized salt and preservatives used in bread products. Adequate iron is also required for thyroid metabolism as are zinc and copper. Vitamin A, vitamin C, vitamin D, vitamin E and balanced essential fatty acids (2:1 to 1:1, Omega 6 to Omega 3) are required for optimal thyroid hormone production. One study showed reduced incidence of hypothyroidism with supplementation of vitamin A and iodine deficient children in developing countries, suggesting that vitamin A supplementation improves iodine efficacy.[10,11]

Measurement of serum TSH is generally considered the best screening test for thyroid disease (Table 1).[12,13] Increased values indicate hypothyroidism. Serum TSH concentrations have a logarithmic relationship with serum thyroxine, so that doubling in thyroxine produces a hundred fold change in TSH. TSH is thus a much more sensitive test. The population reference normal ranges for thyroxine are set wide compared to the normal individual, so that fall in thyroxine levels at the lower end of the range may elevate the TSH above normal. Levothyroxine treatment in patients with serum TSH level 4.5 to 10.0 mIU/L related to certain clinical condition (Table 2).[14,15,16] There are certain factors favouring levothyroxine therapy in TSH level 5 to 10 mIU/L (Table 3).[17,18]

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Table 1: Reference ranges for serum Thyroid hormones and TSH

Hormones	Reference range(SI units)
TSH	0.2-4.5mlU/L
Thyroxine(freeT4)	9-21 pmol/L
Triiodothyronine(T3	0.9-2.4nmol/L

Table 2: Quality of evidence on the strength of association and risks/benefits of levothyroxine treatment of subclinical hypothyroidism for patients with a serum TSH level of 4.5 to 10.0 mlU/L[a]

Clinical condition	Benefits of treatment	Strength of association
Progression to overt Hypothyroidism	Good	Variable[b]
Adverse cardiac end points	Insufficient[c]	No evidence
Elevation in serum total Cholesterol and LDL-C levels	Insufficient[c]	Insufficient
Cardiac dysfunction	Insufficient[c]	Insufficient

[a] LDL-C= low density lipoprotein cholesterol; TSH = Thyroid stimulating hormone

[b] Thyroid hormone therapy normalizes the serum TSH level at any TSH concentration. Overt

hypothyroidism occurs earlier in untreated patients with a serum TSH level of >10mlU/L than in those with a serum TSH level of 4.5 to 10mlU/L.

[c] Available data do not distinguish between serum TSH concentrations of 4.5 to 10 mlU/L and of more than 10mlU/L.

Table 3: Factors favoring levothyroxine therapy in patients with a Thyroid Stimulating Hormone (TSH) level of 5 to 10 mIU/L

Factors Favouring Levothyroxine therapy in patients with a thyroid stimulating hormone (TSH) level of 5 to 10 mlU/L
Pregnancy or intention of pregnancy
Goiter
Therapeutic trial for possible hypothyroid symptoms
Childhood and adolescence
Depression
Infertility

However, some patients are found to have elevated TSH levels, but have normal free thyroxine hormone levels and may also be asymptomatic. One study evaluated the natural history of mild thyroid failure in 154 female patients over a 10 year period , 57% of patients continued to have mild thyroid failure, 34% of patients progressed to overt hypothyroidism and 9% of patients reverted to a normal TSH level.[19,20]

Proponents of the view that most subclinical hypothyroid subjects should be treated with thyroxine advocate early initiation of therapy to prevent the future morbidity associated with development of overt hypothyroidism. The Whickham survey estimated that the progression of subclinical hypothyroidism to overt hypothyroidism occurs in 2-5% of subclinical hypothyroidism subjects per year, an increased risk those with thyroid autoantibodies.[21]

A number of studies do illustrate interesting physiological abnormalities associated with subclinical hypothyroidism but the reversibility of such dysfunction with levothyroxine therapy remains to be proven. If it is true that most subjects with subclinical hypothyroidism should not be treated, then the recommendations, advocating systematic screening of individuals for thyroid disease. Subclinical hypothyroidism occurs in the clinical setting of a serum TSH level above the upper limit of normal despite a normal serum free thyroxine concentration. Initiating levothyroxine replacement therapy is recommended for all patients with a TSH greater than 10ml/L, even if the free thyroxine concentration is within normal laboratory range. However treatment of patients with a serum TSH level between 5 and 10 mlU/L remains controversial. The strongest arguments for levothyroxine therapy are the high risk of progression to overt hypothyroidism. The possible improvement of quality of life, and the possibility that subclinical hypothyroidism is a cardiovascular risk factor.[22,23] Recent evidence shows that any possible increased cardiovascular risk would be to persons younger than 70 years; those aged 70 and 80 years may actually enjoy a protective benefit. Large-scale, government-sponsored, multicenter, randomized, placebo-controlled studies are urgently needed to assess the efficacy of levothyroxine therapy in the sub group with TSH levels less than 10mlU/L. Meanwhile, therapy of this subgroup should be individualized by taking into account patient preference, presence of symptoms, age, and associated medical conditions.

Etiology:

Causes are the same as those of overt thyroid diseases:

1. Chronic autoimmune thyroiditis-Hashimoto's thyroiditis. This is by far the most common cause, accounting for over 90% of cases. Treatment of hypothyroidism most commonly after radioactive iodine treatment.
2. Hypothyroidism can occur in 5-25% patients with surgery or antithyroid drugs.
3. Less common causes are medications- eg. Lithium or Amiodarone
4. Other causes include head & neck surgery or radiotherapy.

Epidemiology:

Subclinical hypothyroidism is a common condition. Prevalence is 3-8%, increasing with age and being more common in women. Iodine deficiency is the most common cause of hypothyroidism worldwide. In persons living in iodine-replete areas, causes are congenital, spontaneous because of chronic autoimmune disease (atrophic autoimmune thyroiditis or goitrous autoimmune thyroiditis [Hashimoto's thyroiditis]) or iatrogenic because of goitrogens, drugs or destructive treatment for thyrotoxicosis. Screening for congenital hypothyroidism exists and its use prevents mental retardation. The prevalence of spontaneous hypothyroidism is between 1% and 2% and is more common in older women and 10 times more common in women than in men. A significant proportion of subjects have asymptomatic chronic autoimmune thyroiditis and 8% of women (10% of women over 55 years of age) and 3% of men have subclinical hypothyroidism. Approximately one third of patients with newly diagnosed overt hypothyroidism have received destructive therapy for hyperthyroidism and indefinite surveillance is required.[24,25] There is not much that can be done to prevent the occurrence of spontaneous autoimmune hypothyroidism, but if identified early, something can be done to prevent progression to overt disease. Controversy exists as to whether healthy adults would benefit from screening for autoimmune thyroid disease because a significant proportion of subjects tested will have evidence of mild thyroid failure. Case finding in women at menopause or visiting a primary care physician with nonspecific symptoms appears justified. 80% of these patients have a serum TSH of less than 10 mlU/L, and 80% have antithyroid antibodies.[26]

Differential Diagnosis:

There are a few other causes of a raised TSH in the presence of normal thyroxine levels.

1. Recovery from acute (non-thyroidal) illness.
2. Assay variability.
3. Heterophile antibodies interfering with the TSH assay (heterphile antibodies are weak antibodies with multi specific activities, which can cause significant interference immunoassays).
4. Central hypothyroidism: In these patients there is hypothalamic or pituitary low serum T4 & T3 with overt (but no goiter). It is rare around 1in 100,000 and usually associated with other pituitary axis abnormalities. Causes include pituitary microadenoma and pituitary infarction.

Clinical Features:

The term subclinical is at times inaccurate, as some patients have symptoms. In the elderly, diagnosis of hypothyroidism may be delayed by wrongly attributing the symptoms, for example fatigue and constipation to ageing. Clinical manifestation can be explained by assuming that T4 level of 6-7 mcg/dl, although inside the normal range may represent a significant decrease from a previous normal of 10mcg/dl and is low for this particular patient. Some studies have suggested that if symptoms are present then treatment with thyroxine will resolve them.

Common Clinical Features of Hypothyroidism include: Depression & fatigue, hyperlipidemia & hyperhomocysteinaemia, goiter, coarse hair, cold intolerance, constipation & weight gain, hoarseness, hearing loss, menorrhagia, low return phase in knee reflexes, bradycardia, coronary artery disease & cardiac risk factors

Investigations:

Recommendations about thyroid screening have been inconsistent. The American Thyroid Association (2001) recommended that adults be screened for thyroid dysfunction by measurement of the serum thyrotropin concentration, beginning at age 35 years and every five years thereafter.

The US Preventive Services Taskforce slightly more recently (2004)-stated that a case had not been made for routine screening.[27,28] In the UK, screening is not felt to be warranted although case finding in women at the menopause or if visiting a doctor with non-specific symptoms may be justified in view of the high prevalence of mild and subclinical thyroid failure. The practical approach may be to measure TSH in those patients who have persistent, non-specific complains women in particular and the elderly.[29] Borderline results may need to be repeated at a consistent time of day, persistent, non-specific complains in women with consistent fasting status.

Management:

If the elevation of TSH level is large and it has been so for a long period of time anti-thyroid antibodies will also have been present. This situation carries a greater likelihood of progression to overt hypothyroidism and therefore a greater potential benefit from treatment.

Treatment Criteria:

All patients with TSH >10mlU/L, or clinical features of hypothyroidism, should be treated.[30] All patients who are pregnant or contemplating pregnancy should be treated to decrease the risk of pregnancy complications and of cognitive impairment in the baby. Controversy remains regarding the treatment of non-pregnant adult patients with serum TSH <10mlU/L. In this sub group treatment should be considered in symptomatic patients, patients with infertility, and patients with goiter or positive antithyroid peroxidase (TPO) antibodies. Limited evidence suggests that treatment of subclinical hypothyroidism in patients with serum TSH<10mlU/L should probably be avoided in those aged >85 years.

Medication:
Levothyroxine is the drug of choice as it has a long half life (seven days) and is partially converted to T3 in the body resulting in a constant physiological level of both T3 and T4 with a single daily dose.[31,32,33]

Dosing:
Young starts at 50 micrograms daily; for elderly it is started at 12.5 to 25 micrograms daily. Monitoring is to be done at 6 to 8 weeks interval initially. Once the correct dose has been established, monitoring can be 6 to 12 monthly.

Aim is to lower TSH to mid normal level 1-3mlU/L.

Contraindications to treatment are osteoporosis, fracture risk etc.

Goals for treatment are a fall in LDL cholesterol or symptomatic improvement or TSH normalizing. Some clinicians believe that treatment targets should be the lower half of the reference range (below 4.0 mlU/L) but many others (including National Institute for Health and Care Excellence) continue to feel that achieving a TSH within the reference range (usually <5.5mlU/L, depending on the laboratory) is adequate in an asymptomatic patient.[34,35] There is no evidence for any benefit to long term outcomes of such disease through treating to a lower level and there may be an increased risk of osteoporosis. The exact upper limit of normal for the serum TSH level remains controversial. Lowering it to 3.0 or even 2.5mlU/L has been proposed.[36,37] However, many criticize this. The argument for lowering the upper limit of normal is the higher level of antithyroid antibodies detected in persons with a serum TSH level between 3.0 and 5.0mlU/L and the higher rate of progression to clinical thyroid disease. The argument against lowering the upper limit of normal for TSH values is that large number of patients would be diagnosed with hypothyroidism without any clinical features.

Prognosis:
Risk of progression to overt hypothyroidism rises with serum TSH level. The presence of goiter, elevated thyroglobulin and thyroid peroxidase antibodies, plus higher TSH predict a progression toward overt hypothyroidism. Replacement therapy is not recommended in asymptomatic individuals with subclinical hypothyroidism but with TSH 5-10 mlU/L, no goiter and negative anti-thyroid antibodies.

Conclusions:
Subclinical hypothyroidism should be addressed in an earlier stage for initiation of treatment. Early treatment will prevent transformation to overt hypothyroidism and its long term treatment complications such as coronary artery disease. Besides, grave complications may developed in pregnancy in the form of spontaneous abortion, gestational hypertension, pre-eclampsia. It may also lead to female infertility. As the condition is relatively asymptomatic, periodic health checkup is needed to diagnose the condition and to avoid the consequent complications.

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Case Report

Progeria with Early Manifestations: A Rare Case from Bangladesh

Ahmed S T[a], Huq M N[b], Sultana N[c], Saadi A M[d]

Abstract

Progeria, also known as Hutchinson Gilford Progeria Syndrome (HGPS), is considered a rare, severe, genetic state of premature ageing at an early age. The present incidence is one in eight million live births. Due to extreme rarity we report this case with classical clinical manifestation, radiological changes and low high-density lipoprotein (HDL).

Keywords: HGPS, Progeria.

Introduction:

Hutchinson-Gilford Progeria Syndrome (HGPS) was first described in 1886 by Jonathan Hutchinson and Hastings Gilford, henceforth named as HGPS. It also occurs sporadically as autosomal inheritance. The word progeria is derived from Greek word, Geraios, which means old. Progeria is commonly prevalent among Caucasians (97%), affecting males slightly more than females (Ratio 1.5: 1). Classically, Progeria Syndrome, is characterized by retarded growth, dwarfism along with abrupt onset of scleroderma. The clinical presentation is usually classical and necessary radiological and biochemical investigations confirm the diagnosis. Although the newborn appears to be normal at birth, growth retardation starts in 2nd year when skin becomes increasingly thin, brittle, lax, shiny and wrinkled in some cases.[1]

The following case depicts all the classical physiological and radiological findings.

Case Presentation:

A lady of 32 years, para 2 delivered a baby boy normally on 22nd May, 2017. It was happened to be a precipitated labor due to early membrane rupture at 26th weeks of gestation. He was delivered at nearest hospital and his APGAR score was 7/10 at 1st and 8/10 at 5th minute.

The mother was not under antenatal checkup during pregnancy. Her pregnancy period was uneventful. There was no history of consanguinity. The siblings of the baby are a 9-years-old brother and a 7 years old sister; both are student of Madrasha and are in good health. No such type of family history was noted. He was admitted in Neonatal ICU of Bangladesh Medical College Hospital, Dhaka. On examination his vitals were: Respiratory Rate: 64/min, Heart Rate: 130/min, Body temp: Normal. Anthropometric measurements: Weiht: 850gm, Supine length: 36cm, Occipito-frontal Circumference: 23.5cm.

Appearance: Dysmorphic faces, old man like faces with large eyes, beaked nose, sticked out ears, high arched palate.

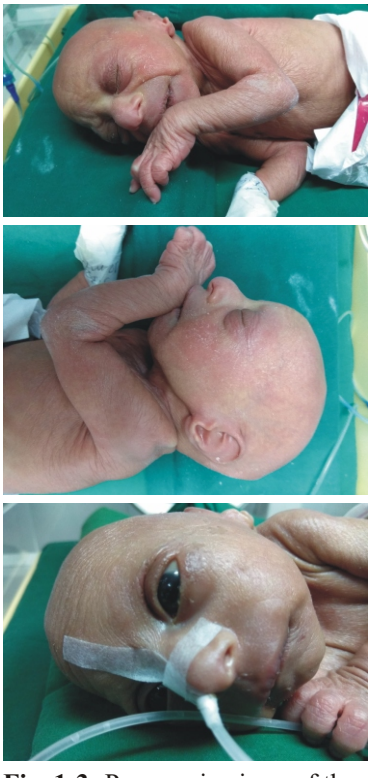

Fig. 1-3: Panaromic views of the patient

Craniosynostosis: Tower shaped head, small lower jaw.

Skin: Thin, wrinkled, alopecia, absence of eye brows and eye lashes.

Scrotum: Rugae present with bilateral undescended testes.

High pitched cry, No other congenital anomaly detected.

Clinical Diagnosis: Baby was diagnosed as a case of Pre-Term (26 weeks) baby, extreme Low Birth Weight (850gm) with Intra Uterine Growth Retardation with suspected Progeria.

Investigation Summary: Blood group: A+, Total count: WBC: 16000/cmm, Neutrophil: 95%, L: 3%, E: 01%, Hb%: 17.6%, Platelets: 250000/cmm, Blood Glucose: 2.5mmol/L, Serum Calcium: 8.8mg/dl & Electrolytes: Na:

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135mmol/L, K: 4.46mmol/L, Cl: 104mmol/L, HCO3: 24mmol/L, C- reactive protein: <6 mg/L.
Lipid Profile: Cholesterol: 113mmol/L, HDL: 11 mmol/L, LDL: 87 mmol/L, Triglyceride: 75 mmol/L, Creatinine: 0.4 mg/dl, SGPT: 31 unit/L.
Imaging: Chest X Ray in A/P view: Normal findings

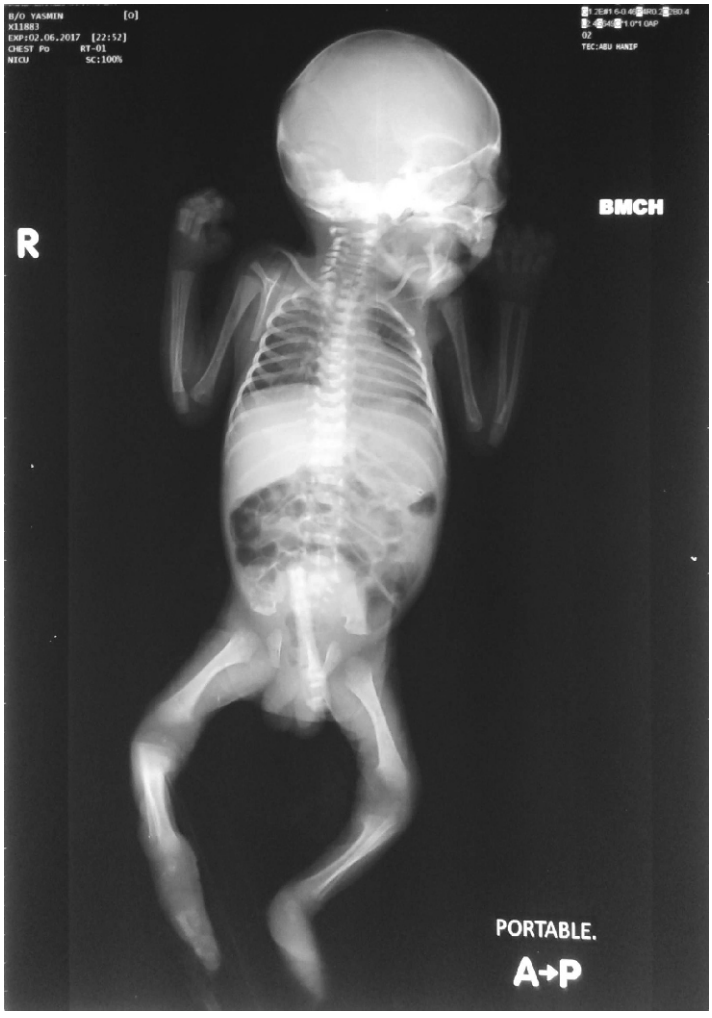

Fig. 4: Infantogram: showing long slender bones.

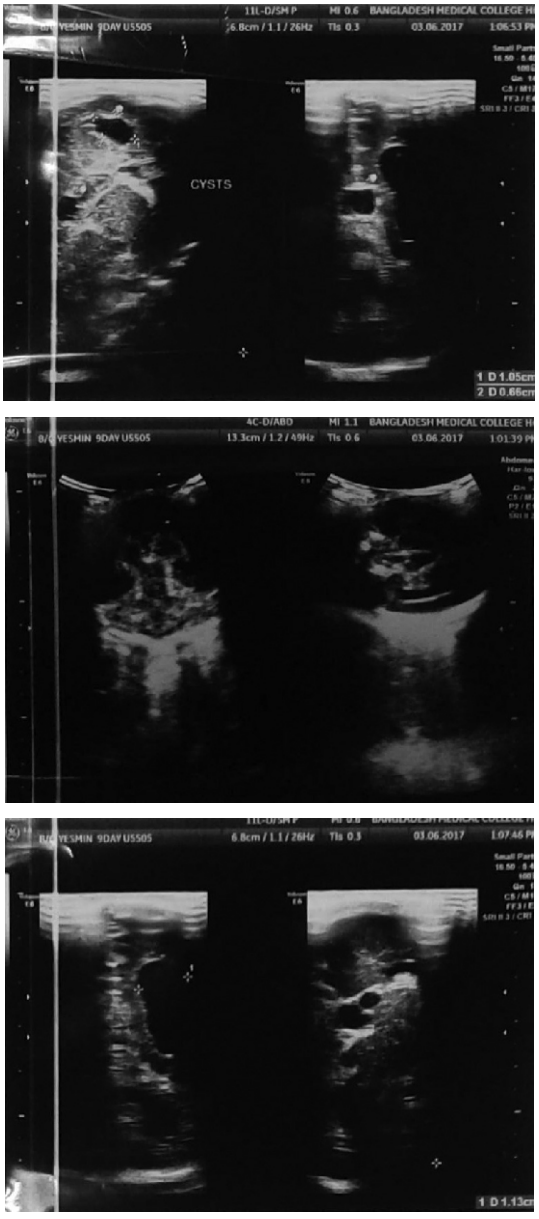

Fig. 5: USG findings

Ultra sonogram findings: Brain: Findings suggestive of internal cortical atrophy with Leukomalacia.
Abdomen: Normal Hepatobiliary system and Kidneys.

After counselling his parents, he was treated supportively and symptomatically.

On 6.6.2017, at Day 15, the baby gained weight from 850 to 955gm, tolerates feed, activity good, received all supplements and is ready for discharge.

Discussion:

The main etiology is yet unclear. Few hypotheses included dysregulation of lamin gene. The mutation of lamin probably responsible for increase in hyaluronic acid resulting sclerodermatous change, cardiovascular changes causing failure to thrive. [2]

Affected children's may be normal at birth, slowly manifests with alopecia, loss of subcutaneous fat, with scleroderma skin changes giving "Plucked Bird" appearance. De Busk later renamed the condition as Hutchinson Gilford Progeria Syndrome (HGPS).The rate of ageing in Progeria usually accelerated with an average life span of 13 years (7-27 years). Interestingly, there was a case that survived 45 years.[3] The mode of death in progeria is usually the cardiovascular complications like: Myocardial Infarction or Congestive Cardiac Failure. [4]

As the etiology is yet to be confirmed, proposed hypothesis

includes Lamin A/C gene malfunction, leading to hyaluronic acid causing sclerodermatous changes and CVS complications. The growth becomes retarded with loss of hair even eyelashes. [5]

The differential diagnosis includes, Acrogeria, Rothmund-Thompson syndrome. There are few case reports where individuals was as old as 45 years. Death caused by atherosclerosis or malignancy. Hutchinson-Gilford Progeria Syndrome (HGPS) mimics with Acrogeria: which is a progeriod condition characterized by ageing of skin and internal organs, mainly found among females known as Gottron Type. Rothmund-Thompson is a hereditary disease where cataract, baldness, teeth, bone and nails changes are more prevalent. Until now, there is no definitive treatment for Progeria, hence patients are managed conservatively. [6]

Limitations: Although Progeria is mostly diagnosed clinically, medical genetics supports its confirmation, which is not feasible in our country.

Conclusion: Progeria is a rare and atypical condition of the newborn baby and it is untreatable. Here the mother did not receive any prenatal care and on the other side it is not possible to diagnose it by prenatal screening test. But by regular prenatal examination the other associated abnormalities could be detected in relation to it and early interventions could be taken.

The parent in this situation also need social and financial support for the management of this condition and empathy to face and overcome the tremendous mental shock as well.

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Case Report

Gartner's Duct Cyst: An Uncommon Cyst of Vaginal Wall

Akter A[a], Uddin M N[b]

Abstract

Cyst of vaginal wall is rare. This case relates to a patient who presented with a polypoidal mass protruding out from vagina which could have been easily mistaken as uterovaginal prolapse, but appropriate clinical evaluation supported with investigations clinched the diagnosis easily.

Keywords: Vaginal wall, Vaginal cyst, Wolffian duct.

Introduction:

Since the vaginal epithelium is normally devoid of glands, most cysts arise from included or adjacent structures. Their nature and origin are therefore determined clinically by their position. The majority of vaginal cysts arise from Gartner's duct; their origin is deduced by their position on a line running down the lateral or anterolateral wall of the vagina from the cervix to the region of the urethra and clitoris.[1] The proportion of Gartner's duct cysts is 12.5% of all vaginal cysts. As it may arise from remnants of Wolffian duct, it may even appear in late middle age. Vaginal cyst can be histologically classified as epithelial, inclusion, mullerian, mesonephric, and urothelial in addition to other rare types.[2] It was presented with symptoms of visible palpable mass, dyspareunia, voiding disturbances, vaginal discharge, and pain.[3]

Case Presentation:

A patient, 30 years-old, para 3, presented herself in the outpatient department of Gynaecology, with the complaints of a mass protruding out from vagina for the last 3 years. On eliciting further history, she narrated that this mass initially was small in size which gradually progressed to the size of an egg. So the patient started having discomfort during micturition, defaecation and coitus. Mass was not reducible. She also gave history of regular cycle with average menstrual flow and duration.

General, physical, and systemic examinations were unremarkable. Per-vaginal examination revealed an antero-lateral vaginal wall cyst with smooth pale pink shiny surface (Fig-1). Posterior vaginal wall was normal. Uterus was normal in size, ante-verted, mobile and fornices were free and non-tender. No cystocele and rectocele were demonstrable with and without straining.

On investigations, status of hemoglobin 12.4 gm/dl, platelet count 2.9 lacs/cmm, total leukocyte count 6600/cmm, and serum creatinine was 0.7 mg/dl.

On USG, uterus was normal in size with endometrial echoes in the central region, myometrium was uniform. Both adnexa appear to be normal. No collection or mass was seen in the Pouch of Douglas.

Operative procedure and intraoperative findings: Surgery was planned under regional anaesthesia. Anterior vaginal wall cyst was excised by dissecting anterior vaginal wall and identifying the base (Fig-2). Stalk was ligated, anterior vaginal wall cyst was removed and anterior colporrhaphy was done. Urinary bladder was identified by passing metallic catheter through the urethra. Specimen was sent for histopathological examination.

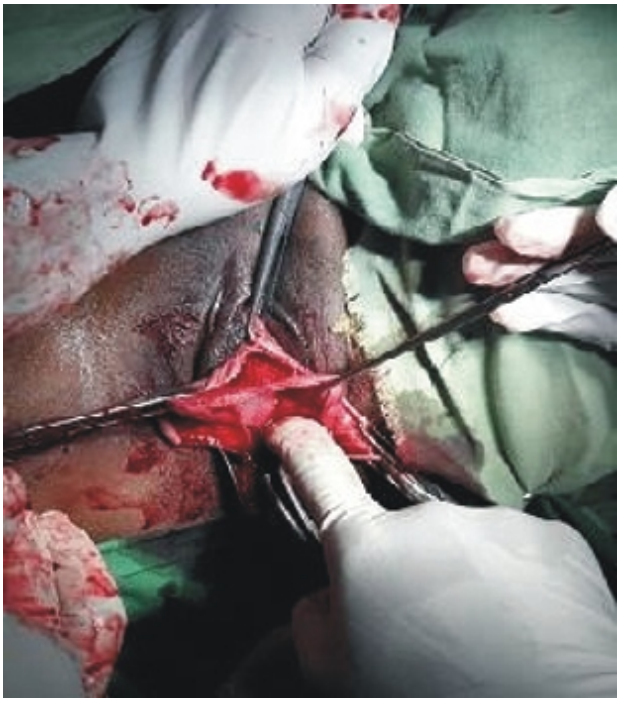

Fig 1: Anterolateral vaginal wall Gartner's duct cyst

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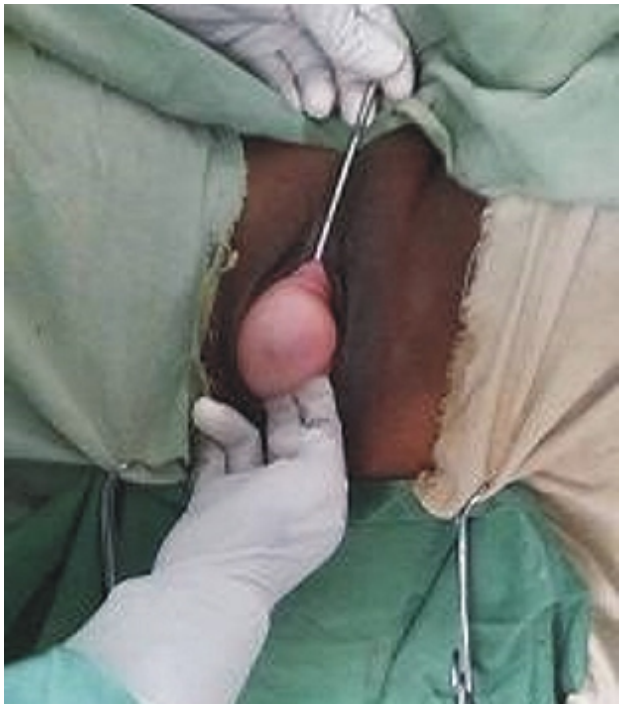

Fig 2: Dissection of Gartner's duct cyst

Histopathological finding reveals globular cyst measuring 5 cm in diameter. On cut section, unilocular cyst was identified filled with thick jelly as brownish fluid. Inner lining was smooth. Cyst wall showed tall columnar epithelial lining. The sub-epithelial tissue showed fibrocollagenous and muscle tissue. Impression stated as Benign epithelial cyst-mesonephric type.

Postoperative period was uneventful, and the patient was discharged in satisfactory condition.

Discussion:

Vaginal wall cysts are identified in approximately 25% of all adult women, and nearly 1% evolves into Gartner's duct cysts. During embryological development, the mesonephric (Wolffian) ducts develop to form their predetermined structures and later regress. Remnants often remain, however, when they develop a secretory mechanism, cause dilation of surrounding cells, and thus yield a Gartner's duct cyst, most often during and after late adolescence.

Classically, the cysts are solitary, unilateral, <2 cm in diameter and are located in the anterolateral vaginal wall of the proximal one third of the vagina.[4] Gartner's duct cysts are generally asymptomatic and most commonly diagnosed upon routine gynaecological examination, but patients' complaints can include that of skin tag, dysuria, pressure, itching, dyspareunia, pelvic pain, or protrusion from the vagina if it enlarges to a detectable size, making it a candidate for surgical removal. If large enough to cause obstetrical complications, the cyst can be drained to facilitate delivery.

To define the course of the Gartner's duct cyst and differentiate it from other pathologic considerations and structures, magnetic resonance imaging can be a useful tool. Histopathological examination may be employed to correctly identify the cellular remnants composed of nonmucin secreting low columnar or cuboidal epithelium. The differential diagnosis can be included, but is not limited to Bartholin's gland cyst or abscess, prolapsed urethra, prolapsed uterus, vaginal wall inclusion cyst, endometriosis, leiomyoma, sarcoma botryoides, malignant mass, Skene's gland cyst, or abscess and ureterocele. Only in exceptionally rare and isolated cases, there has been a malignant transformation identified.[4]

Conclusion:

Large vaginal wall cysts are always symptomatic which compels the patient to visit a gynaecologist. Not all patients presenting with mass per vagina are necessarily a case of uterovaginal prolapse. Vaginal wall cyst prolapse is a rare entity and requires proper examination. Treatment is simple and safe.

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Case Report

Ovarian Metastasis of Gastric Cancer: Krukenberg Tumour- A Case Report with Literature Review

Masud ZM[a], Tabassum SK[b], Hossain MA[c], Nuzaira Nahid[d]

Abstract

Gastric cancer, one of the most common cancers in the world, rarely metastasizes to the ovaries. Ovarian metastases of gastric signet ring cell cancer are referred to as Krukenberg tumors and account for 12% of all ovarian cancers. A 25 year old female patient, hailing from Madhubazar, Dhaka, was admitted to BMCH on 26th October, 2016 with the complaints of heaviness and pain in the lower part of abdomen for 7 months, a swelling in the lower part of her abdomen which grew rapidly in size and lumpy feeling in both of her breasts for 1 month. Histopathology of the resected ovary showed presence of metastatic adenocarcinoma and some of the tumour cells were of signet ring type, suggestive of Krukenberg Tumour. In this report, we reviewed the previous literature and discussed etiology, clinical manifestations, diagnosis, and treatment.

Keywords: Krukenberg tumour, Ovarian metastasis, Stomach cancer.

Introduction:

Krukenberg tumours are histologically defined as carcinoma within the ovary containing mucin-filled signet ring cells and represent metastasis to the ovary. Up to 30% of ovarian metastases arise from a colorectal origin, with gastric adenocarcinoma being the most common primary malignancy.[1] This is a rare metastatic tumour, accounting for 12% of all ovarian tumors. The stomach is the primary site in the majority of Krukenberg tumor cases, followed by carcinomas of the colon, appendix and breast, particularly invasive lobular carcinoma.[2] These are solid tumors and 80% are bilateral. [3] Gastric cancer metastasizes via hematogenous and lymphogenous routes, peritoneal seeding and direct invasion.[4-5]

Case Presentation:

A 25 year old female patient, hailing from Dhaka, was admitted to Bangladesh Medical College Hospital on 26th October, 2016 with the complaints of heaviness and pain in the lower part of abdomen for 7 months. The pain was gradual in onset, colicky in nature, increased on an empty stomach, decreased on intake of antiulcerants and was not associated with vomiting, fever or alterations of bowel habit. She also developed an irregular menstrual cycle, which lasted for 2 days only and occurred once in every 2 to 3 months. She suffered from generalized weakness and loss of appetite and started losing weight. About a month back, she noticed a swelling in the lower part of her abdomen which grew rapidly in size, and she had a lumpy feeling in both of her breasts.

On admission, she was very ill- looking, weak and mildly anaemic. Her vitals were stable. The abdomen was distended. Palpation revealed a huge immobile mass, hard in consistency with regular margin, extending from hypogastric region to the epigastrium. Liver, kidney, spleen were not palpable. There were lumps present in the upper and outer quadrant of both breast, which were non-tender, firm, immobile and adherent to the underlying structures. Axillary lymphadenopathy was not present. All other systemic findings were normal. CA-125 was elevated (85.80 U/ml) and USG of the lower abdomen showed large hyperechoic nonhomogeneous mass extending from the epigastric region to the pelvic region, which was 16.8 X17 cm in diameter, and vascularization was seen in Doppler study. CT scan of the whole abdomen showed left ovarian cyst with suspected uterine fibroid. FNAC of the breast lump showed fibrocystic changes in both breast with fibroadenoma in the right breast. Serum creatinine, X-ray and IVU were normal.

She underwent exploratory laparotomy with left sided salpingo-oophorectomy on 29th October 2016. Cytology of the peritoneal fluid came back negative for malignant cells. Histopathology of the resected ovary showed presence of metastatic adenocarcinoma and some of the tumour cells were of signet ring type, suggestive of Krukenberg Tumour.

About a week after the operation, the swelling in the lower part of the abdomen returned. Peripheral blood film revealed microcytic hypochromic anaemia. CA-19.9 was

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raised to 1068.71 U/L and alkaline phosphatase was raised to 465 U/L. USG of whole abdomen showed large intra-abdominal solid mass, 25 X 15 cm in diameter, with mild ascites and bilateral pleural effusion. She was readmitted to BMCH on 20th November for further evaluation and chemotherapy. She received combined intravenous chemotherapy Oxaliplatin and Capecitabin. After 2 cycles she has improved physically. On 26th January 2017 she was admitted with altered consciousness. She died on 28th January 2017.

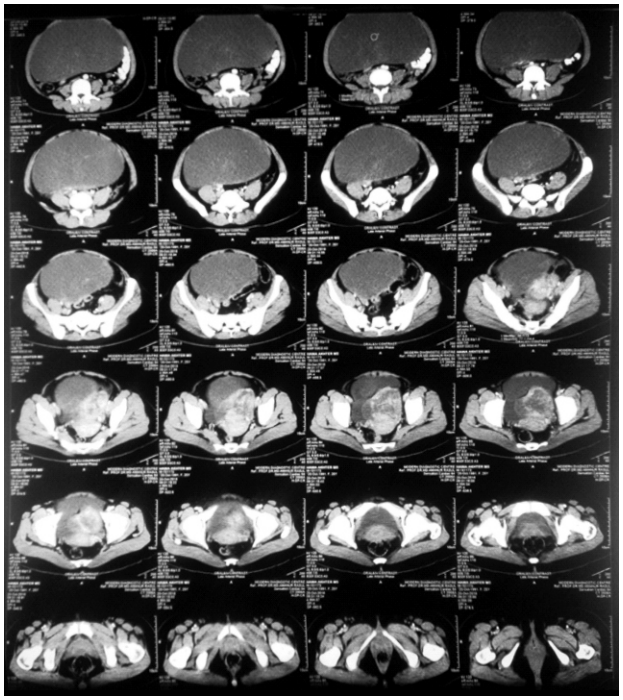

Fig. 1: CT scan shows soft tissue mass in lower abdomen and left ovarian mass

Discussion:

Krukenberg tumor is considered as a late-stage disease with poor prognosis and may account for 30-40% of metastatic cancers to the ovaries.[6] The treatment approach to these metastatic ovarian tumors remains controversial. To date, treatment mainly consists of ovarian metastasectomy, chemotherapy or radiotherapy; however, the optimal treatment has not yet been established. Surgery is the main treatment in the absence of metastases for medically fit patients. However, considering local and distant recurrence of gastric cancer, chemotherapy and radiation therapy are also included.[7] Postoperative chemo-radiotherapy is shown to decrease local recurrence in patients with locally advanced gastric cancer. Progression-free survival and OS (Overall survival) data demonstrate a continued strong benefit from postoperative chemo-radiotherapy.[8,9] Palliative options including radiotherapy may be applied for unresectable or metastatic gastric cancer.[10]

Metastatic or inoperable gastric cancer patients have a poor prognosis with a median OS, if untreated, of 3 to 5 months. Chemotherapy combined with the best supportive care can improve the survival of these patients. Despite the development of chemotherapeutic agents for treating metastatic disease, the prognosis is usually poor, with a 5-year survival rate of 5-15%.[11]

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College News

College Events:

International Mother Language Day was observed in Bangladesh Medical College and Hospital (BMC & H) on 21 February, 2017 at the campus premises.

Seminar in BMC:

A Seminar on "Prevention of Cancer and Molecular Cancer Diagnostics" was held on 7th March 2017 in BMC. The speaker was Dr. Zaheed Hosain, Ph.D, Cancer Immunologists in Beth Israel Hospital, Boston, USA.

Participation in the International Conferences/Seminars/Workshop/ Congress/Meetings:

- Prof. Md. Ashraful Islam, Professor and Head of the Dept. of ENT, Bangladesh Medical College attended the 69th Annual Conference of the Association of Otolaryngologists of India held on 2-5 February 2017 in India.
- Dr. Mushtaque Ahmad Rana, Associate Professor, Dept. of Gastroenterology, Bangladesh Medical College attended the First World Congress of Gastrointestinal Endoscopy (ENDO 2017) held at Hyderabad, India from 16-19 February, 2017.
- Prof. Dr. Sharmeen Yasmeen, Professor and Head of the Dept. of Community Medicine, Bangladesh Medical College attended the 8th SEAPHEIN Annual Meeting and International Conference on "Moving SEAPHEIN to Influence Public Health Management Education and Action" held at Jaipur, India from 13-15 February, 2017.
- Prof. Dr. Md. Fazlul Kadir, Professor, Dept. of Medicine, Bangladesh Medical College attended the 9th Diabetes Conference & Grand Rounds (DCOM 2017) held at Kuala Lumpur, Malaysia from 21-23 April, 2017.
- Dr. Kamrun Nahar, Associate Professor, Dept. of Radiology and Imaging, Bangladesh Medical College attended the International Intensive Hands on Musculoskeletal MRI and Ultrasound Course held in Malaysia from 15-18 March, 2017.
- Dr. Md. Amir Hossain, Associate Professor, Dept. of Cardiology, Bangladesh Medical College attended the 26th International Conference on "Medical, Medicine and Health Sciences" (MMHS-2017) held at Bali, Indonesia from 08-09 April, 2017.
- Prof. Dr. Sharmeen Yasmeen, Professor and Head of the Dept. of Community Medicine, Bangladesh Medical College attended International Conference on Autism and Neurodevelopmental disorders (ANDD2017) at Thimphu, Bhutan from 19-21 April, 2017.
- Prof. Dr. Md. Zahid Hasan Bhuiyan, Professor, Dept. of Urology, Bangladesh Medical College attended the "112th Annual Meeting of American Urological Association (AUA 2017) held from 12-16 May, 2017 in Boston, USA.
- Dr. A. B. M Mahbubur Rahman, Assistant Professor, Dept. of Surgery, Bangladesh Medical College attended the Advanced Laparoscopic Surgery Course, held at New Delhi, India from 16-27 April, 2017.
- Dr. Akhil Chandra Biswas, Associate Professor, Dept. of ENT, Bangladesh Medical College attended the 2nd World Congress on Endoscopic Ear Surgery held at Bologna, Italy from 27-29 April, 2017.
- Prof. Dr. M. Fakhrul Islam, Professor & Head of the Dept. of Urology, Bangladesh Medical College attended the 112th Annual Meeting on American Urological Association (AUA) held at Boston, USA from 12-16 April,2017.
- Dr. Zafrana Zahir, Lecturer, Dept. of Community Medicine, Bangladesh Medical College attended the International Conference on Public Health (ICOPH 2017)) held at Kuala Lumpur, Malaysia from 27-29 June,2017.
- Prof. Dr. Md. Mizanur Rahman, Professor and Head of the Dept. of Ophthalmology, Bangladesh Medical College attended the 7th World Glaucoma Congress 2017 held at Helsinki, Finland from June 28th to 1st July 2017.

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