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Editorial

Urological care for healthy aging of male: An emergence of awareness

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Bangladesh is experiencing a demographic shift with a rapidly growing elderly population who are ≥60 years of age. According to the census 2011 and 2022, they constituted 7.47% and 9.29% of the total population respectively and in 2050 it is predicted to be double. With the advancement of age, risk of developing all systemic diseases increases and urological problems are not exceptions. It hampers not only their physical health, also mental and social welling and is closely associated with the quality of life irrespective of gender. People often neglect and hide the problems as these are mostly not life threating but they continuously suffer and pay financial penalty to get management at the later stages.

Several risk factors are responsible for urological health issues. These are aging, medical conditions like diabetes, hypertension, stroke, heart diseases and lifestyle issues like smoking, excessive alcohol intake, obesity, sedentary lifestyle and lack of physical activity, poor diet and hydration, stress etc.

Urological diseases are a broad category of illnesses, connected to the body's urine filtration and excretion mechanism. Among male population heath related urological issues may appear long before their 60 years of age.

Common urinary problems among males:

Urinary Incontinence (UI): It is involuntary urination leading to uncontrolled leakage of urine. Age-related alterations affect smooth muscle, causing weakening of urethral tone and resistance to intra-abdominal pressure. This is one of the most prevalent urinary issues among the elderly male and female. It creates an embarrassing and distressful situation and seriously affect the quality of life of the patients.

Urinary Tract Infections (UTIs): UTIs are more common in older adults and can cause symptoms such as frequent urination, burning sensations, fever, lower abdominal pain and discomfort. They can also lead to more severe complications if left untreated.

Interstitial Cystitis (IC): A type of chronic pain syndrome of bladder and pelvic floor of unknown cause characterized by chronic pelvic pain, urinary urgency and increased urinary frequency.

Benign Prostatic Hyperplasia (BPH): Many older men experience an enlarged prostate, which can lead to urinary symptoms like frequent urination, weak urine flow, and difficulty in emptying the bladder.

Overactive Bladder: This condition causes a sudden urge to urinate, which can be challenging for seniors to manage, particularly if they have mobility issues.

Kidney Stones: These are hard deposits of minerals and salts that form inside the kidneys. Symptoms include severe pain, nausea, and haematuria.

Prostatitis: This is the inflammation of the prostate gland, which can lead to pelvic pain and urinary issues.

Erectile Dysfunction (ED): This refers to the inability to maintain an erection adequate for sexual intercourse, impacting both physical and psychological health and conjugal disharmony.

Urological Cancers: These include prostate, bladder, and kidney cancers. Many serious conditions, including prostate cancer, bladder cancer start quietly. Early detection through regular screenings can significantly improve outcomes.

Managing urinary issues in the elderly:

Proactive screening and lifestyle modification is a powerful duo of thumb rules that prevents a spectrum of urological conditions, from benign prostatic hyperplasia (BPH) to urinary incontinence, kidney stones, and erectile dysfunction (ED). These issues might not be life-threatening, but they can still produce disability and affect quality of life. Identifying them early opens the door to management strategies before they become severe.

Interventions for prevention and proper management:

1. Routine yearly screening tests

Includes urine analysis and urine culture, Blood for creatinine and uric acid, Prostate-specific antigen (PSA) test, Digital Rectal Examinations (DRE), Imaging tests like CT scans, Ultrasounds etc.

2. Healthy lifestyle

- **Diet:** A balanced diet rich in fruits, vegetables, and whole grains supports overall health. Vegetables and fruits rich in fibers have impacts on regular bowel movements. Drinking Cranberry juice of taking cranberry pills can prevent UTIs and reduce urinary incontinence.
- **Physical activity and exercise:** Regular physical activity helps to maintain a healthy weight and reduces the risk of conditions like BPH and ED.

Practice Pelvic floor exercises, also known as Kegel exercises. It strengthens the muscles that control urination and increase vascularity and neurophysiological function of urology system. These exercises can be easily incorporated into daily routines

- **Hydration:** Drinking plenty of water helps to flush the urinary tract and reduces the risk of kidney stones and UTIs.

3. Managing chronic conditions

Proper management of chronic conditions such as diabetes and hypertension is crucial as they are risk factors for many urological problems. Medications to control blood pressure and blood sugar can help protect kidney function.

4. Medications

Depending on the specific urinary issue, medications may be prescribed. For example, antibiotics are commonly used to treat UTIs, while medications like alpha-blockers can help manage BPH symptoms in men. Additionally, drugs that reduce protein in the urine (e.g., ACE inhibitors or ARBs) can be beneficial.

5. Avoidance of bladder irritants

Tobacco, alcohol, caffeine etc. are bladder irritants. Cessation of smoking of all forms, avoiding excessive caffeine and alcohol are important for control as they can exacerbate urinary problems.

6. Maintenance of healthy body weight

Prevention of obesity and maintenance of healthy weight is essential.

7. Scheduled bathroom breaks

Establishing a routine for bathroom breaks, even if the urge to urinate is not present, can help manage overactive bladder symptoms and prevent accidental urinations.

8. Assistive devices

For seniors with mobility issues, assistive devices such as bedside commodes, raised toilet seats, or grab bars in the bathroom can provide much needed support and prevent falls.

9. Catheterization:

In some cases, individuals with severe urinary retention may require intermittent catheterization under medical supervision.

10. Surgery:

For certain conditions like BPH or severe incontinence that do not respond to conservative treatments, surgical interventions may be considered. In severe cases of incontinence, surgical options such as sling procedures or bladder neck suspension may be considered.

11. Dialysis and Transplantation

In advanced stages of CKD, dialysis or kidney transplantation may be necessary. However, these options are less common in very elderly patients due to associated risks and complications.

12. Patient education and empowerment

In medical practice patient education is a critical component. An informed patient is an empowered patient for decisions and actions to take regarding when, where, whom and why to seek care! So fostering suitable environment of open communication is also very important.

Conclusion:

Aging is a natural phenomenon. But we must remember every age is unique and could be productive for thyself, family and community. Very often our aging population are neglected and under cared in the family, even they also give up to live a healthy and graceful life.

However, with proper care, attention, and medical guidance, these challenges and shortcomings of the society can be effectively managed. Aging gracefully involves taking proactive steps towards maintaining urological health. Seniors and their caregivers need to be proactive in seeking medical evaluation and adopting lifestyle modifications that can make a substantial difference in managing urinary problems. It is our collective responsibility to ensure that our aging population receives the care and support they need to live comfortably and with dignity.

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Management of hypotension in patients undergoing surgery for inguinal hernia under spinal anesthesia: Comparison between intravenous phenylephrine and ephedrine

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Abstract

Background: Hemodynamic instability after spinal anesthesia (sub arachnoid block) is one of most common complications that an anesthesiologist has to deal with clinical practice. Ephedrine and phenylephrine are the two potent drugs to manage hypotension during spinal anesthesia.

Objective: To compare the effectiveness of ephedrine and phenylephrine on hypotension and heart rate after spinal anesthesia

Methods: This randomized control study was conducted on 100 male patients scheduled for surgery for inguinal hernia in a medical college hospital. Patients were selected according to inclusion criteria. They were divided equally into two groups where group A (n=50) patients received intravenous ephedrine 5mg bolus and group B (n=50) received intravenous phenylephrine 50 microgram bolus dose. The hemodynamic parameters were compared at a regular interval. The main outcome variables were systolic arterial pressure (SAP), diastolic arterial pressure (DAP) and Heart rate (HR).

Results: This study showed that phenylephrine and ephedrine both are equally potent in managing hypotension. Moreover, phenylephrine has shown significantly higher systolic and diastolic arterial pressure from 12 to 30 minutes ($p<0.05$) after anesthesia and ephedrine showed considerably higher heart rate from 12 to 18 minutes ($p<0.05$) in study participants.

Conclusion: During hypotension induced by spinal anesthesia, phenylephrine has better control over systolic and diastolic arterial pressure whereas ephedrine shows increased heart rate compared to phenylephrine.

Keywords: Regional anesthesia, ephedrine, phenylephrine.

Introduction:

Herniotomy for indirect inguinal hernia is one of the common surgeries performed in Bangladesh Medical College Hospital. Hypotension while performing surgery under spinal anesthesia should be treated properly to prevent any kind of untoward complications. The administration of bolus crystalloid fluid may be effective to prevent hypotension after anesthesia.[1] But, anesthesiologist should be careful in fluid management when they deal with patient with cardiovascular and renal diseases. Elderly patients are also vulnerable for fluid overload and pose the risk for further decreases of systemic vascular resistance and cardiac output.[2] Thus, anesthesiologists use several drugs along with the fluid administration to combat hypotension during anesthesia. Ephedrine is the drug of choice as vasopressor for spinal anesthesia throughout the world.[3] It has direct effects on alpha and beta receptors. Ephedrine causes increase in systolic and diastolic arterial pressure and heart rate.[4] Phenylephrine is another alpha adrenergic agonist, with no effect on beta receptor. It causes direct arteriolar constriction resulting an increase arterial pressure.[5] Unlike ephedrine, phenylephrine is less likely cause of tachyphylaxis and supraventricular tachycardia.[6]

Materials & Methods:

This was a prospective randomized double-blind study, conducted in a medical college hospital on male patients undergoing surgery for inguinal hernia during the period of 1year.

Inclusion criteria were- 1. Patients giving informed consent; 2.

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Male patients undergoing herniotomy surgery; 3. ASA physical status I and II patients; 4. Age between 18-65 years. Exclusion criteria were-1. ASA III and IV; 2. Patients above below 18 and above 65 years; 3. Patients with hypertension, diabetes mellitus, bronchial asthma, and ischemic heart disease; 4. Morbidly obese patients; 5. Patients with any history to local anesthetic drug, ephedrine, and phenylephrine; 6. Patients having contraindication of subarachnoid block (spinal anesthesia)

In the study 100 patients, aged 18 to 65 years, who met the inclusion criteria were scheduled for elective surgery for inguinal hernia under spinal anesthesia, were selected by prospective randomized double-blind selection. All the patients were divided into two groups; Group A received ephedrine and group B was allocated phenylephrine. A proper pre-anesthetic evaluation was followed to assess the physical condition of the patients and suitability for the study. A proper fasting protocol has been following since the night before surgery for the patients in both groups.[7] After entering in the operating room, patients were applied ECG, noninvasive blood pressure, heart rate and oxygen saturation of blood with pulse oxymeter. An 18G intravenous line was introduced on each of the patients and preloaded with Ringer's lactate solution at a rate of 15ml/kg within half-life.[8] Spinal anesthesia was administered at Lumber 3-4 or 4-5 interspace with 25G needle. Heart rate, systolic and diastolic arterial pressure were monitored and recorded regularly on every patient. After confirming the level of block to T5 level, surgery was started.[9,10]

When the systolic BP less than 90 mm Hg or decrease in systolic BP more than 20% of base line whichever is lower. The patients were randomly provided two types of vasopressor drugs whenever needed. Group-A was given 5mg of intravenous bolus of ephedrine while Group-B was received 0.5ml (50 microgram) intravenous bolus of phenylephrine.[11,12] The study drugs were prepared and labeled by an anesthesiologist who did not take part in the process of data collection.

Statistical Analysis

All the patients were monitored, and systolic arterial pressure, diastolic arterial pressure and heart rate have been recorded at a regular interval up to 90 minutes after anesthesia. The data were analyzed by SPSS Windows version 20.

Results:

Table 1: Demographic characteristics of patients (N=100)

	Group A (Mean±SD)	Group B (Mean±SD)	p value
Age (years)	57.86±6.54	54.48±7.05	0.82
Height(cm)	169.08±3.91	169.72±4.16	0.14
Weight(kg)	72.78±4.39	70.50±5.48	0.55

Table 1 shows the demographic characteristics of the study patients, it was observed that mean age was found in 57.86±6.54 years in group A and 54.48±7.05 years in group B. The mean (SD) height of the study patients were found 169.08±3.91 cm in group A and 169.72±4.16 cm in group B. The mean weight of the study patients was 72.78±4.39 kg in group A and 70.50±5.48 kg in group B. The difference was not found statistically significant ($p > 0.05$)

Table 2: Pre-operative assessment of patient (ASA grading)

	Group-A Number (%)	Group-B Number (%)	p value
ASA Grade I	73	80	0.26
ASA Grade II	27	20	0.35

Table 2 shows pre-operative assessment of the study patients. The ASA grade I was 73 (%) in group A and 80 (%) in group B. The ASA grade II was 27 (%) in group A and 20 (%) in group B. The difference was not statistically significant ($p > 0.05$) between two groups.

Table 3: Comparison of systolic arterial pressure (SAP) in mmHg among the two groups

Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	p value
Base line	126.92±4.65	127.30±5.57	0.43
3 min	120.76±6.34	121.26±5.90	0.45
6 min	117.45±4.54	115.68±6.54	0.35
9 min	108.93±8.35	110.25±6.45	0.52
12 min	112.16±9.14	118.27±11.4	0.032
15 min	114.96±7.15	121.56±5.34	0.027
18 min	110.68±6.23	118.73±7.23	0.018
21 min	115.38±8.17	122.84±6.73	0.023
24 min	112.46±9.34	121.65±8.52	0.036
27 min	109.17±7.54	118.64±7.82	0.031
30 min	117.73±8.15	115.55±8.12	0.21
35 min	115.63±7.83	117.53±9.38	0.38
40 min	118.89±8.41	116.81±7.54	0.26
45 min	117.67±9.14	119.73±8.52	0.17
50 min	115.53±7.64	116.81±6.87	0.21
60 min	113.51±8.53	114.78±7.92	0.33
70 min	117.87±7.93	116.75±8.34	0.37
80 min	116.56±8.56	118.48±6.51	0.27
90 min	118.89±7.23	120.41±6.83	0.23

Table 3 shows Systolic arterial pressure (SAP) changes were not significant between the groups up to 9 minutes after anesthesia. On the other hand, SAP were significantly lower in group I than group II from 12 to 27 min after anesthesia and those values were statistically significant as p value <0.05. Though, 30 min after anesthesia SAP were not significantly different in both groups up to 90 min of operation time.

Table 4: Comparison of diastolic arterial pressure (DAP) in mmHg among the two groups

Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	p value
Base line	81.14±4.67	81.72±4.54	0.74
3 min	68.59±6.80	69.47±5.90	0.43
6 min	64.73±7.84	65.71±8.52	0.38
9 min	61.84±6.39	60.85±7.44	0.47
12 min	68.26±7.18	80.86±5.61	0.026
15 min	72.96±6.25	79.43±7.44	0.032
18 min	67.85±8.43	75.34±6.58	0.034
21 min	73.45±6.64	79.32±7.54	0.025
24 min	71.46±9.34	77.14±8.61	0.028
27 min	67.37±8.53	74.34±7.62	0.036
30 min	69.62±7.61	72.83±6.26	0.026
35 min	70.31±8.17	71.63±7.25	0.28
40 min	68.58±6.43	69.73±8.14	0.35
45 min	67.82±7.27	69.45±8.39	0.24
50 min	70.18±8.46	71.28±6.54	0.38
60 min	72.80±6.18	73.74±6.62	0.27
70 min	72.57±7.82	72.17±7.93	0.31
80 min	74.81±8.51	73.67±7.15	0.26
90 min	74.18±6.39	74.83±8.79	0.38

Table 4 shows that Diastolic arterial pressure (DAP) changes were not significant between the groups up to 9 minutes after anesthesia. On the other hand, DAP were significantly lower in group I than group II from 12 to 30 min after anesthesia and those values were statistically significant as p value <0.05. Though, 30 min after anesthesia DAP were not significantly different in both groups up to 90 min of operation time.

Table 5: Comparison of Heart Rate (beats per minute) of the patients among the two groups

Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	P value
Base line	84.46±11.59	82.64±9.48	0.64
3 min	89.15±10.47	90.41±11.19	0.56
6 min	92.37±9.38	93.74±10.26	0.48
9 min	93.64±10.18	93.15±11.71	0.64
12 min	92.64±11.42	79.53±12.82	0.018
15 min	90.73±9.81	81.63±11.54	0.037
18 min	88.74±10.74	85.26±9.42	0.028
21 min	89.26±9.37	87.43±11.61	0.23
24 min	87.72±10.31	88.72±11.75	0.14
27 min	85.14±11.14	86.64±10.41	0.21
30 min	87.51±10.24	86.11±10.63	0.37
35 min	88.27±11.26	87.51±11.41	0.23
40 min	89.63±10.36	87.42±10.24	0.18
45 min	88.76±11.42	86.82±11.63	0.23
50 min	87.42±10.63	88.73±9.42	0.31
60 min	86.54±9.26	87.32±10.42	0.24
70 min	87.32±11.72	88.54±10.26	0.35
80 min	86.63±10.25	87.52±9.64	0.41
90 min	85.41±11.78	86.41±10.82	0.32

Table 5 shows comparison of baseline heart rate of patients in both group showed no statistical difference. From 12 to 18 min mean heart rate in group II was significantly lower from group I as p value were <0.05. From 21 to 90 min intraoperative heart rate in both groups showed no significant difference.

Discussion:

Regional anesthesia has some great advantages over general anesthesia. Among the regional anesthesia, spinal anesthesia is the most common form that has been widely used for different surgical procedures. However, management of hypotension is the main challenge that the anesthesiologist must overcome during surgical procedure under spinal anesthesia. Although perioperative fluid replacement can prevent hypotension induced by spinal anesthesia, in many cases anesthesiologist must be cautious about fluid overload specially in elderly patients. In this scenario, vasopressors like ephedrine or phenylephrine may be a good option over fluid replacement to control the hypotension.

In the study, demographic data and ASA grading were comparable in two group. The baseline arterial pressure, both systolic and diastolic, did not have any significant difference in both groups. Even there was no considerable difference (p <0.05) in mean systolic and mean diastolic arterial pressure in both groups from the administration of anesthesia to 9 min of operation timeline. The study showed a statistically significant difference in systolic and diastolic pressure in group A and group B from 12 to 30 min of operation time, where phenylephrine group demonstrated considerable higher mean systolic and diastolic arterial pressure from 12 to 30 min. This findings have the similarity with the study conducted by Abbasivash R et al.[13]

When we measured the baseline heart rate in the both groups there was no significant difference. However, after introduction of anesthesia from 12 to 18 min, group A showed significantly higher mean heart rate than group B, that was similar with the findings of study conducted by Naghibi et al.[14] However, in the study conducted by Lee A et al,[15] showed higher incidence of bradycardia in the group where phenylephrine was used but in our study there was no patient who developed bradycardia in the study time. Miodrag Žunić et al[16] in their study observed that the higher heart in ephedrine group is beneficial for elderly patients to maintain the cardiac output, simultaneously increase myocardial oxygen demand. However, Hedge J et al[17] in their study showed that patients with cardiac risk factors like ischemia should be carefully monitored and drugs must be chosen meticulously where hypotension can be prevented with minimal increase of heart rate to mitigate the myocardial oxygen demand.

Conclusion:

After administration of anesthesia in this study, phenylephrine and ephedrine both showed considerable similarities in terms of managing hypotension. However, it has been observed that ephedrine showed a significantly higher heart rate after few minutes under anesthesia. This increased heart rate may have no untoward effect in healthy individuals but may jeopardize the cardiovascular function especially in elderly patients with co-morbidities. Thus, phenylephrine can be an attractive alternative to ephedrine in the management of hypotension in spinal anesthesia.

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Original Article

Influencing factors of caesarean section among married women in Bangladesh

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Abstract

Background: The worldwide increase in cesarean sections (CS) has a significant impact on maternal and newborn health, particularly in a developing nation like Bangladesh. In Bangladesh, the CS rate, which comprises institutional and community-based births, increased from approximately 3% in 2000 to 33% in 2018.

Objective: To assess the influencing factors of cesarean section in Bangladesh among married women.

Methods: This research was conducted to identify potential socioeconomic, demographic, Institutional, and social network factors influencing the provision of CS in Bangladesh. Data from the Bangladesh Demographic and Health Survey (BDHS) 2017-18 were utilized for this study.

Result: This study used data from 4,929 mothers who gave birth in healthcare institutions three years before the survey. The prevalence of CS delivery among Bangladeshi mothers was 33.37 % (43.65 % in urban areas and 28% in rural areas). Logistic regression showed that mothers aaged30-49 years (AOR = 1.19, 95% CI = 0.94 to 1.50), wealth index average (AOR = 1.79, 95% CI = 1.37 to 2.55), and richest (AOR = 3.13, 95% CI = 2.32 to 4.21), antenatal visit >3 (AOR = 2.03, 95% CI = 1.76 to 2.33) were significantly more prone to CS delivery. Also, the education and occupation of mothers and husbands, as well as mobile phone use, were potentially associated with CS delivery.

Conclusion: Bangladesh has a far higher rate of C-sections than the WHO recommends. As a result, Bangladesh's high CS delivery rates may not be associated with better birth outcomes. It is essential to decrease such a phenomenon, making the mothers aware of the risks of cesarean delivery and establishing counseling sessions.

Keywords: Cesarean Sections, Married women, Bangladesh.

Introduction:

A surgical procedure performed during childbirth is called Cesarean Section (CS). This process occurs through the abdominal and uterine incisions during a CS. It is a potentially dangerous procedure that can result in hemorrhage, Infection after CS, pre-eclampsia, repeated CS, other organ injuries, and other maternal-fetal factors. [1,2] CS should only be performed when medically required. Otherwise, life-threatening complications can occur for the mother and newborn.[3,4] Furthermore, CS-delivery infants have a higher risk of respiratory problems such as transient tachypnea, low APGAR score, surgical injury, allergic rhinitis, childhood asthma, and type 1 diabetes onset in childhood than VB-delivery infants (Vaginal Birth).[5] Over the last three decades, CS rates have increased, and the World Health Organization (WHO) says that up to 15% rates are acceptable. However, it climbed above the expected rate of all births in 2015 in numerous middle- and high-income countries.[6] In Bangladesh, between 2014 and 2017–18 BDHS, the proportion increased from 23% to 33%. According to Save the Children, among the CS rates in Bangladesh, the number of medically unnecessary CS has climbed by 51% in recent decades. This rate is nearly four times higher than the World Health Organization (WHO) recommends.[7] An earlier study of data from 26 South Asian and sub-Saharan nations found that rates were highest among the "urban rich" in every nation and lowest among the "rural poor" across every region.[8]

CS is now carried out for financial, commercial, or self-desirable motives. A few adequately specified maternal reasons, such as severe antepartum hemorrhage, significant cephalo-pelvic disproportion, failed progress of labor, repeated CS, etc., justify the use of CS.[9] Despite being a reasonably safe delivery procedure, CS has complications.[10] The government of Bangladesh has established and put into practice various maternal health-related measures throughout the last few decades.[11]

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We are all familiar with the medical rationale for cesarean sections. So many studies have demonstrated how factors associated with pregnant mothers and the healthcare system raise the demand for CS services.[12] Numerous studies have shown that women desire CS in addition to the on-demand CS because they fear labor pain, interference with future sexual performance, and discomfort during labor.[13] Many doctors believe this is a significant role in the rise of non-medical cesarean sections.[14] Many studies have found that socio-demographic, socioeconomic, cultural, family, and facility characteristics are linked to a high rate of CS.[15] This study aimed to identify the recent determinants of CS in Bangladesh. This will help us to take the necessary action by emphasizing responsible factors for reducing unnecessary CS.

Materials & Methods:

A descriptive cross-sectional study was conducted among married women in Bangladesh**.** This study used data from the BDHS for 2017–2018 to assess the variables affecting cesarean section. A two-stage stratified cluster sampling methodology based on enumeration areas (EAs) and households were used for the BDHS 2017–18. This study's sample size was 4,929, and 1645 were delivered through CS. The factors connected with CS delivery are divided into six major categories: Socio-demographic factors, socioeconomic factors, Institutional factors, Social-network factors, medical causes for CS-related factors, and non-medical causes for CS-related factors. The statistical analysis used the software program Stata (version 14). A descriptive analysis was used to view the characteristics of the participants. A Bivariate (Chi-square test) and multivariate analyses (Logistic regression) were done to understand the association of outcome variables and determinants of CS.

Results:

In this study among 4,929 mothers who delivered in health facilities, 1,645 **(**33**.**37%**)** delivered through CS**.**

Table 1 shows that most of the mothers were 20**-**24 (34.52%) and 25**-**29 **(**33**.**33%) years old**.** Two**-**fifths **(**43**.**65%; p<0**.**0001**)** of the urban mothers underwent CS**.** CS delivery was higher among the higher**-**educated mothers **(**61**.**59**%),** statistically significant, and CS was statistically prevalent among unemployed mothers (38.38%). Women who own a mobile phone went through CS more, which is statistically significant (40.85%; p<0.0001). Mothers from the richest wealth quintile also experienced CS more (63.37%; p<0.0001). CS was more prevalent among higher-educated husbands (61.74%). In contrast, CS was conducted more for mothers whose husbands were professionals (64.11%; p<0.0001), and the CS risk is equally higher when the mother or both of them choose. Those with more than four visits had a chance of a higher rate of CS (50.81%; p<0.0001). Most mothers with CS went to the private sector (65.22%), which is statistically significant.

Table 1: Socio-demographic Characteristics of participants (N=4929)

Variable	Delivered by CS n_1 (%)	Normal delivery n_2 (%)	P-value (Difference in proportions)
Total	1645(33.37)	3,284(66.63)	
Mother's age in year			
15-19	247(29.16)	600(70.84)	0.030
20-24	603(34.52)	1144(65.48)	
25-29	429(33.33)	858 (66.67)	
30-49	366(34.92)	682(65.08)	
Residence			
Urban	739(43.65)	954(56.35)	<0.0001
Rural	906(28.00)	2,330(72.00)	
Mother educational level			
No education	47(15.46)	257(84.54)	<0.0001
Primary	244(17.85)	1,123(82.15)	
Secondary	804(34.00)	1,561(66.00)	
Higher	550(61.59)	343(38.41)	
Mother currently working			
No	1144(38.38)	1,837(61.62)	<0.0001
Yes	501(25.72)	1,447(74.28)	
Owns a mobile telephone			
No	408(21.46)	1,493(78.54)	<0.0001
Yes	1237(40.85)	1,791(59.15)	
Economical background			
Poorest	136(12.76)	930(87.24)	<0.0001
Poorer	221(22.19)	775(77.81)	
Middle	281(31.68)	606 (68.32)	
Richer	372(38.04)	606 (61.96)	
Richest	635(63.37)	367(36.63)	
Husband education level			
No Education	110(16.22)	568(83.78)	<0.0001
Primary	365(22.07)	1,289(77.93)	
Secondary	578(35.44)	1,053(64.56)	
Higher	589(61.74)	365(38.26)	
Don't know	3(25.00)	9(75.00)	
Husband occupation			
Professional	293(64.11)	164(35.89)	<0.0001
Business	64(18.99)	273(81.01)	
Services	706(32.67)	1,455(67.33)	
Others	565(29.26)	1,366(70.74)	
Unemployed	17(39.53)	26(60.47)	
Decision maker for respondent's health issues			
Respondent alone	131(35.12)	242(64.88)	0.005
Respondent and husband	1115(34.62)	2,106(65.38)	
Husband/partner alone	298(29.30)	719(70.70)	
Someone else	94(33.81)	184(66.19)	
Other	7(17.50)	33(82.50)38	
Number of antenatal visits during pregnancy			
No antenatal visit	19(4.76)	0(95.24)	<0.0001
1-4 visits	721(26.23)	2028(73.77)	
More than 4 visits	905(50.81)	876(49.19)	

Table 2: Reasons for choosing cesarean section delivery
*Multiple response observed

Reason	Mother side (n)%	Doctor side (n)%
Convenience	109(67.70)	52(32.30)
Water broke/dried up	12(21.82)	43(78.18)
Didn't want labor pains	80(76.19)	25(23.81)
Mal presentation	90(22.50)	310(77.50)
Premature baby	01(6.67)	14(93.33)
Cord prolapsed	04(26.67)	11(26.67)
Multiple births	07(36.84)	12(63.16)
Progress in labor	129(32.74)	265(67.26)
Pre-eclampsia	08(23.53)	26(76.47)
Diabetes	01(7.69)	12(92.31)
Previous C-section	195(48.99)	203(51.01)
Complications during delivery	150(27.57)	394(72.43)

Table 2 shows that in most cases, doctors made the ultimate decision regarding CS. Therefore, convenience (67.70%), avoiding labor discomfort (76.19%), and previous CS (48.99%) were the most frequently cited reasons for having a cesarean section from the mother's side. Diabetes and premature babies were the leading reasons for conducting CS on the doctor's side.

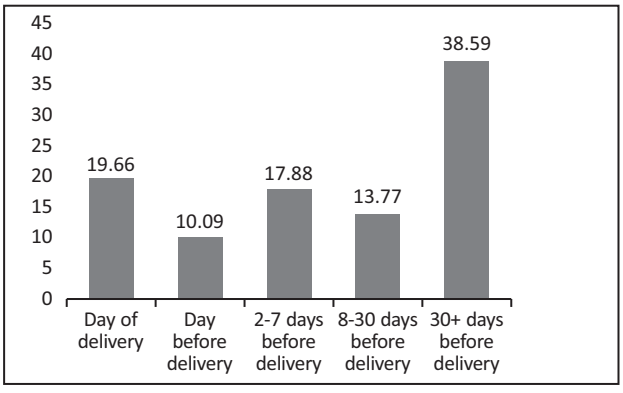

Figure 1: Timing of decision-making on cesarean section

In Fig 1 most of the cases (38.59%), the decision for CS was made 30 days before the delivery, which is called elective cesarean section. The decision was made the day before the delivery date in about one-fifth of the cases (10.09%). The decision for CS was made on the day before delivery (19.66%) and 2 to 7 days before delivery (17.88%), which are almost equal.

Table 3: Association between cesarean delivery and selected socio-economic, demographic, biological, and institutional factors and social network factors based on logistic regression.

Characteristic	Adjusted Odds Ratio	P value	95% CI of AOR
Mother Age			
20-24	0.94	0.581	0.75-1.16
25-29	1.04	0.683	0.85-1.27
30-49	1.19	0.142	0.94-1.50
Residence			
Rural	0.98	0.826	0.83-1.15
Mother Education			
Primary	0.82	0.294	0.55-1.19
Secondary	1.19	0.373	0.81-1.72
Higher	1.70	0.013	1.11-2.59
Mobile			
Yes	1.29	0.001	1.10-1.50
Economical background			
Poorer	1.36	0.018	1.05-1.75
Middle	1.68	0.000	1.28-2.17
Richer	1.79	0.000	1.37-2.34
Richest	3.13	0.000	2.32-4.21
Husband Education			
Primary	1.13	0.374	0.86-1.46
Secondary	1.28	0.076	0.97-1.67
Higher	1.86	0.000	1.35-2.55
Don't know	0.99	0.989	0.24-3.97
Mother Occupation			
Yes	0.70	0.000	0.60-0.81
Husband Occupation			
Business	1.16	0.480	0.77-1.73
Services	0.92	0.528	0.70-1.20
Others	0.79	0.090	0.60-1.03
None	0.93	0.845	0.44-1.94
Person who usually decides on respondent's health			
Respondent and husband	0.99	0.921	0.76-1.27
Husband alone	0.79	0.114	0.59-1.05
Someone else	0.87	0.459	0.59-1.26
other	0.69	0.410	0.27-1.68
Number of antenatal visits during pregnancy			
more than 4 visits	2.03	0.000	1.76-2.33

Table 3 shows that older mothers aged 25-29 years and 30-49 years had higher odds of delivery by CS [OR=1.04; CI=0.85-1.28 and OR=1.19; CI=0.94-1.50], respectively, than adolescent mothers aged 15–19 years. Mothers who lived in rural areas had lower odds of CS delivery [OR= 0.98; CI=0.83-1.15] than mothers who lived in urban areas. The chance of CS increased with the mother's level of education. Similarly, those husbands with higher education had more chance of having their wives CS [OR=1.86; CI=1.35-2.55]. Mothers with mobile phones had more odds

of conducting CS [OR= 1.29; CI= 1.10-1.50]. Mothers who belonged to higher wealth quintiles had more chance of getting CS, e.g., richest [OR = 3.13; CI = 2.32-4.21]. Mothers who were employed had fewer odds of CS done [OR = 0.70; CI= 0.60-0.81] than those who were not employed. Mothers who received more antenatal care visits had a higher chance of CS delivery than those with less than three visits [OR= 2.03; CI=1.76-2.33].

Discussion:

Bangladesh's rising population-based CS rates may be attributable to the various maternal health programs currently in place.[16] In 2014, the all-cause CS rate in Bangladesh was 9% higher than the WHO recommendation range of 10%-15%(34).[17] The message that CS is convenient, less painful, and comparatively simple was conveyed to mothers, which increases the demand for elective cesarean sections.[18] In our study, we can see that more than 60% of mothers chose CS for convenience and to avoid labor pain, and when the patients are the principal decision-makers, these two reasons dominate. On the other hand, in most cases, physicians went for CS when the pregnancies were complicated, e.g., malpresentation of the fetus (22.0%), other complications (72.43%), and failure to progress in labor (67.26%). Physicians' incentives for performing CS include both saving time from prolonged normal vaginal deliveries and making more money at the same time.[19]

The timing of the decision on CS also shows that the decision on 38.59% of CS was taken 30 or more days before the delivery date. This signifies that a big proportion of CS was decided way before the indication for CS, e.g., complications, arose. Educated, affluent, and urban mothers have shown a significant increase in the prevalence of CS.

During the past few decades, important social determinants of health in Bangladesh, particularly female education, have improved significantly.[20] The findings of this study indicate that mothers gained more education than their spouses. In this study, the education and occupation of the husband have a significant effect on the CS rate, as they are the main influence on respondents' health. With increasing urbanization, rising average income, and greater coverage of private facilities, the CS rate will continue to rise in Bangladesh.[21,22] A greater proportion of mothers have received multiple prenatal visits, indicating an increase in ANC service utilization. The outcomes of this study reveal that having a CS is less likely if you have your ANC visits and births done in a public hospital. The age of the mother also plays an important influence in CS performance.[23]

In this study, older mothers have a greater likelihood of undergoing a cesarean section than younger mothers. Awareness developed due to increased exposure mobile phone, and due to increasing socialization, mothers nowadays embark on pregnancy at a later age. Therefore, the chances of CS are high for their delivery. In this study, we found that the majority of reasons to have CS are due to complications during pregnancy or suggested by doctors.

One of the limitations of the study is the recall period. The three-year BDHS recall period was insufficient to describe all birth-related events. Therefore, the probability of recall bias should be quite low. Except for a few questions, almost all of the information collected in BDHS surveys was susceptible to reporting biases. Another limitation is that data was obtained solely from the responses of mothers. The accumulation of data from physicians would allow us to conduct a more thorough analysis. However, the strength of this paper is that it utilizes very recent data to demonstrate the cesarean section indicators.

Conclusion:

The study found that mothers from urban areas, affluent families, and private facilities were more likely to have a cesarean section. Higher maternal age, mother education, and occupation, number of ANC visits, spouse education, and occupation were all significant predictors of cesarean section delivery.

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Calculation of arch index from footprint of male medical students in Bangladesh: A cross- sectional study

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Abstract

Background: Foot dimension has great use in anatomy, physiology, ergonomics, forensic science, plastic surgery, radiology, podiatry, archaeology, anthropology, nutrition science and diagnostic knowledge between patient and normal population. The data of foot dimensions of Bangladeshi male is essential for the anatomist for normative reference, to the radiologist and podiatrist for diagnosis of diseases in their respective fields.

Objective: This cross sectional analytic study was planned to determine the arch index of foot from the footprint of Bangladeshi male medical students.

Methods: Two hundred (200) male medical college students of 18-25 years of age were selected from different medical colleges of Dhaka city and their age was determined by their national ID cards. Both feet of the same individual were painted with ink with the help of the brush. After ensuring that toes and sole were inked properly, footprints were taken at the same time for both feet while the ink was still wet. The footprints were scanned after putting a graph paper on top which was photocopied on a transparent paper. Arch index was calculated from the footprints superimposed by a transparent photocopied centimeter calibrated graph paper. Calculations were done by counting the big boxes first (1 cm^2) then the small boxes in the centimeter calibrated graph sheet which was scanned along with the footprint.

Results: In the present study, normal foot arch was found in 41 % and 39.5% individuals on the right and left foot respectively. High arch was observed in 22% and 23% on right and left foot respectively while low arch on right and left foot were found in 37% and 37.5% individuals respectively.

Conclusion: On calculation of the arch index, maximum distribution of foot arch was normal (41% on the right foot and 39.5% on the left foot) in type, the low foot arch type was 37% on the right foot and 37.5% on the left foot and the high foot arch showed minimum distribution (22% on the right foot and 23% on the left foot) among adult Bangladeshi male medical students.

Keywords: Arch index, foot print, male medical students

Introduction:

The foot dimensions derived from footprint can provide definitive information on many physical characteristics of the individual as morphology of human foot shows variations due to the combined effects of heredity, lifestyle and climatic factors. The partial or complete footprints can be found on rain covered areas, newly waxed floors, freshly cemented surfaces, moistened surfaces, in dust, mud, sand, oil, paint and can be left in blood at the crime scenes.

Arch-height estimation though practiced usually in supine posture; is neither correct nor scientific as referred in literature, which favour for standing x-rays or arch index as yardstick. In fact, the standing x-rays can be excused for being troublesome in busy OPD, but an ink-footprint on simple graph-sheet can be documented, as it is easier, cheaper and requires almost no machineries and expertisation.[1]

Measurement of the height of the arch of foot deserves immense importance so far its clinical aspects are concerned and for this purpose since middle of the past century several methods were used by pioneer researchers. Practically the height of the medial longitudinal arch provides acceptable outlook of the arch-height. Some researchers have classified the foot arch type by only visual impression, which was quite practiced till the end of last

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century.[2-4] On the other hand a few of them carried on such a classification based on palpation of the navicular tuberosity.[5] In late nineties researchers approached with the help of radiography in parallel with footprint. Radiographically parameters like the 'taller height', 'navicular height' and recently the 'normalized navicular height' obtained from standing weight bearing lateral view x-ray of foot, were accepted as yardsticks to predict the arch height.[6-9]

Procurement of, and processing the footprint being easier and cheaper, is more acceptable for the patient than radiography. Hence, in spite of the fact that radiography is still important in establishing the arch height, footprint procedures are preferred to it.[10-11] It was previously disclosed that the foot-print obtained on a graph sheet by conventional ink is better that the electronic foot-print obtained by special soft-ware system, so far determination of the sole contact area was concerned.[12] This can be conveniently taken on a graph paper and the Arch Index can be calculated thereafter to ascertain the height of the arch of foot. The concept of Arch Index was first described by Cavanagah et al. (1987) as the ratio of the area of the middle third of the foot to the entire foot area excluding the toes. An arch index of less than 0.21 has been said to be indicative of a cavus foot, while it greater than 0.26 is indicative of planus foot whereas Arch Index between 0.21~0.26 corroborates normal arch height. Importance of "arch-index" as a sensitive podographic indicator was later on confirmed in different studies[13]. Later it has been established that arch index, derived from footprint to show a significant negative correlation with the navicular height.[9, 14-16] But unfortunately almost no studies have inter-related mathematically the foot-print derived arch-index values with the radiographically[17] evaluated standing arch-height measurements with an acceptable equation, by which one can interpret directly the standing navicular or taller height with the help of arch index without proceeding through actual maneuver. Especially such information lacks in pertinent literature so far.

The values of different dimensions of foot of Bangladeshi males may be helpful to the anatomists for a normative reference. For the radiologist and the podiatrists the normative values may be helpful in diagnosis of diseases in their respective fields. This knowledge of different foot dimensions and its correlation with the arch of foot is of extreme importance for the forensic scientists to establish the identity of an individual. For proper designing of a prosthetic foot by the ergonomists and for surgical reconstruction by the plastic surgeons foot dimension data is essential.

With the above perspective, the present study was carried out to give the overview of foot dimensions of Bangladeshi males and to calculate the arch index of foot of the same age group of Bangladeshi males**.**

Materials and Methods:

This cross sectional analytic study was carried out at Department of Anatomy of Dhaka Medical College, Dhaka from July 2012 to June 2013. The study was performed on 200 male Bangladeshi medical students of age ranging from 18-25 years' individuals with congenital anomaly of feet, any deformity of feet from any disease, endocrine diseases like acromegaly, gigantism, those who have encountered any road traffic accident or burn injury affecting feet and tribal population were excluded from the study.

The feet of the individual were washed with liquid soap before inking. Feet were washed in order to remove oily or greasy substance and dirt from the foot. The feet were then wiped with a towel. Two legal size white papers were fixed on a clip board with double clips to take print of right and left foot which was placed on an even floor as footprints of both feet were collected at the same time. A small amount of ink was poured into a clean and dry flat box with a wide base. The individual was asked to sit on a chair and rest his legs on a low stool with extended knee so that his feet were placed beyond the stool for proper painting of the soles. A wide paint brush was moved in the ink over flat surface of the wide based box until the ink spread thinly and homogenously in the brush.

The right and left foot were painted with ink with the help of the brush. After ensuring that toes and sole were inked properly, footprints were taken at the same time for both foot while the ink was still wet. The feet were carefully removed from the stool and the soles were placed slowly on the paper from proximal to distal end while the individual still remained seated. The individual was then asked to stand from sitting position with his feet placed on the papers on the clipboard without moving the feet. After ensuring that the feet were placed properly the individual was asked to stand erect without any support while putting equal pressure on both foot without moving their position on the papers. The individual was then asked to sit. The feet were then lifted from the paper at the same time so that there was no overlapping of the already imprinted footprint.

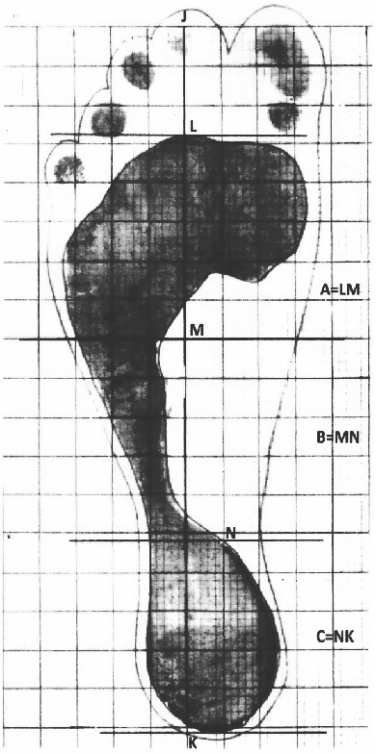
For statistical analysis, SPSS version 16 was used. The relationship between the right and left footprint measurements were determined by paired t- test. The study was approved by Ethical Committee of Dhaka Medical College.

Measurement of arch index

Arch index was calculated from the footprints superimposed by a transparent photocopied centimeter calibrated graph paper. The footprints were scanned after putting a graph paper on top which was photocopied on a transparent paper. Then the linear distance of the centre of the heel (point K) and the tip of the second toe, which is the axis of the foot (point J) was measured (Fig:1). Next a perpendicular line was drawn tangential to the most anterior point of the main body of the footprint (footprint without the toes). Their point of intersection was marked (point L). The line LK was then divided in three equal parts (LM, MN, NK). Ultimately the whole body of the footprint was divided in three areas from those points with the perpendiculars from the foot axis (Fig:1). The anterior,

middle and posterior areas were marked as A, B and C which represented the areas of forefoot, midfoot and hindfoot respectively (Fig:1). Calculations were done by counting the big boxes first (1 cm^2) then the small boxes in the centimeter calibrated graph sheet which was scanned along with the footprint. Further calculations were carried out by using the foot index formula:

Arch index (AI) = B ÷ [A + B + C][1]



J- tip of 2nd toe

K- centre of heel

L- most anterior point of main body footprint

A=LM area of forefoot

B=MN area of midfoot

C=NK area of hindfoot

Arch index (AI)= B÷[A+B+C][1]

Fig 1: Showing graph paper on footprint for calculation of arch index of foot

Table 1: Arch index of right and left footprints of adult male medical students

Index (cm^2)	Right foot (n_2=200) **No**. (%)	Left foot (n_2=200) **No**. (%)
High (<0.217)	44 (22.0)	46 (23.0)
Normal (0.217-0.261)	82 (41.0)	79 (39.5)
Low (>0.261)	74 (37.0)	75 (37.5)

Table 1 shows Arch index of the right and left foot ranged from 0.117-0.379cm^2 and 0.123-0.469 cm^2 respectively and the mean(±SD) was 0.251±0.047cm^2 and 0.250±0.047cm^2 respectively.

High arch index (< 0.217) of the right and left foot were found in 44 and 46 individuals respectively and the percentage were 22% and 23% respectively.

Normal arch index (0.217- 0.261) of the right and left foot were found in 82 and 79 individuals respectively and the percentage were 41% and 39.5% respectively.

Low arch index (> 0.261) of the right and left foot were found in 74 and 75 individuals respectively and the percentage were 37% and 37.5% respectively.

Discussion:

The present work was undertaken to study arch index from footprint of 200 (two hundred) medical college students of Dhaka city and describes a statistically based analysis that illustrates the usefulness of the footprint of an individual. The footprints of the present study were collected from the Department of Anatomy of Dhaka Medical College, Dhaka and other non-government medical colleges in Dhaka city. The main aim of the study is to calculate the arch index of foot from footprint of Bangladeshi males. The findings of this study were statistically analysed and revealed important information about variations in foot dimensions of Bangladeshi males.

The reason of dissimilarities might be due to racial variation as he used sample from tribal population who are mostly engaged in the agricultural work all the time, putting more strain on their feet while working in the fields and therefore it is most natural that the foot is more used for walking or weight bearing becomes physically better developed**.** Another reason of dissimilarity is also supported by Rao and Kotian,cited by Kewal Krishan[18] as they suggested that the difference between left and right footprints in the same individual is not a coincidence but may be explained on the basis of the **"**dominant foot**".** Most of the individuals have dominant foot, usually the left one, which always support the body to a greater extent while in standing or in walking**.** The shoe of this foot wears off at a faster rate than the shoe on the other foot**.** The bones in the dominant foot are regularly subjected to stronger stress forces like weight bearing pressures, than the bones of the other foot**.** This in turn enlarges the bones of the dominant foot and therefore produces a footprint of a larger dimension**.** The medial longitudinal arch also serves to distribute the weight of the body and absorb shock.[19] Damage to the normal biomechanics of the foot, caused by abnormal function of the subtalar joint and medial longitudinal arch, can result in flatfoot.[20]

Hironmoy et al. (2012) conducted study on 103 adult males and females of North Bengal.[1] The findings of their study were higher in percentage both in case of high and normal arch than the present study. Another study was conducted by Xiong S. et al.[21] on 48 (24 males and 24 females) Hong Kong Chinese adults. The findings of their study were also higher in percentage than the present study.

Hironmoy Roy[1] cited, Values of Arch Indices in respective sex-group were also calculated out to be finalized with mean of 0.22 ±0.04 and 0.23 ±0.03 among males and females. Following the classification-system as described

by McCroy et al.(1997)[15] based on the arch index, in the present population 59.8% had normal arch, whereas 35.3% and 4.9% had high and flat arches respectively.

In present study, normal foot arch was found in 41 % and 39.5% individuals on the right and left foot respectively. High arch was observed in 22% and 23% on right and left foot respectively while low arch was observed on right and left foot were found in 37% and 37.5% individuals respectively.

Conclusion:

In the present study, on calculation of the arch index, maximum distribution of foot arch was normal (41% on the right foot and 39.5% on the left foot) in type, the low foot arch type was 37% on the right foot and 37.5% on the left foot and the high foot arch showed minimum distribution (22% on the right foot and 23% on the left foot) among adult Bangladeshi male medical students.

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Original Article

Distinguishing sellar and suprasellar masses: A CT scan approach

Mahmood S

Abstract

Background: The differential diagnosis of sellar and suprasellar masses by CT scan is pivotal due to the anatomical complexity and varied pathologies involved. Accurate characterization through radiological imaging plays a crucial role in guiding management decisions and improving patient outcomes.

Objective: To distinguish sellar and suprasellar masses through CT imaging in symptomatic patients, emphasizing key radiological features, diagnostic considerations, and management implications.

Methods: This study was carried out in the Department of Radiology and Imaging with collaboration with the Department of Neurosurgery, in tertiary care hospital from July 2004 to June 2005. Patients with suprasellar masses who had undergone CT scan before surgery and histopathological examination post operatively were included in this study irrespective of age and sex. Total 60 patients, age ranging between 7 and 55 years were included in the study.

Results: Majority of people presented with headache 93.3% followed by visual problem in 78.3% cases. Among radiologically diagnosed sellar/suprasellar masses 60% were pituitary adenoma and histopathological data 51% pituitary adenoma.

Conclusion: CT scanning is indispensable in distinguishing sellar and suprasellar masses, aiding in precise diagnosis and management planning. Awareness of characteristic imaging features facilitates timely intervention, ensuring optimal patient care and outcomes in this complex anatomical region.

Keywords: Sellar mass, Suprasellar mass, CT scan, Pituitary adenoma

Introduction:

The evaluation of sellar and suprasellar masses poses a significant diagnostic challenge due to the wide array of potential differential diagnoses and the critical anatomical structures involved. These masses can arise from various origins including pituitary adenomas, craniopharyngiomas, meningiomas and metastatic lesions among others, each necessitating distinct management strategies and prognostic implications. The advent of computed tomography (CT) scanning has revolutionized the detection and characterization of these lesions, providing crucial insights into their size, morphology, and surrounding tissue involvement.

By examining specific characteristics such as per description size, enhancement patterns, calcifications, and surrounding tissue involvement, CT imaging plays a crucial role in narrowing down the differential diagnosis and facilitating timely intervention. Though the superiority of MRI over CT is well known, MRI is expensive and limited availability, hence CT remains the most widely used form of neuroimaging for diagnosis of brain tumours.[1]

CT scanning is a fundamental imaging modality in the initial evaluation of sellar and suprasellar masses due to its widespread availability, rapid acquisition time, and ability to provide detailed anatomical information. Contrast-enhanced CT scans enhance the delineation of vascular structures and can highlight certain features of masses that aid in the differential diagnosis. The interpretation of these scans requires a nuanced understanding of normal anatomy, pathological processes, and imaging artifacts to accurately diagnose and guide subsequent management. Subdividing sellar/juxtasellar lesions into intra, supra and juxtasellar masses facilitates diagnosis although some disease processes involve more than one area.[2]

Pituitary adenomas represent the most common sellar masses encountered clinically, with varying hormonal activity and growth patterns influencing their radiological appearance on CT scans. These benign neoplasms can range from microadenomas (<10 mm) to macroadenomas (>10mm), often demonstrating homogeneous enhancement with contrast due to their vascular supply.

In contrast, craniopharyngiomas, derived from remnants of Rathke's pouch, typically exhibit calcifications and cystic components on CT imaging, distinguishing them from other sellar masses.

Meningiomas, arising from arachnoid cap cells, may also involve the sellar and suprasellar regions, displaying dural tail sign and hyperostosis on CT scans.

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Differentiation of suprasellar masses by computed tomography criteria depends on intrasellar extension, patterns of calcification and enhancement, associated bony reaction, and cystic changes. In the case of a homogeneously enhancing suprasellar mass that does not exhibit any of these differentiating findings, separating a suprasellar meningioma from a large suprasellar pituitary adenoma has been quite difficult.[3]

Sellar and suprasellar tumours constitute about 25% of total intracranial mass lesions, 50% of them are pituitary adenomas, 25% are craniopharyngiomas and 10% meningiomas.[4]

Metastatic lesions to the sellar and suprasellar regions represent a diagnostic challenge, often presenting with variable enhancement patterns and multiple lesions in different anatomical locations. CT imaging plays a critical role in identifying these lesions, guiding further workup for primary malignancies elsewhere in the body. Other less common entities such as lymphomas, germ cell tumors, and infectious processes may also manifest as sellar and suprasellar masses, each necessitating a tailored diagnostic approach based on CT findings.

CT imaging serves as an indispensable tool in the differential diagnosis of sellar and suprasellar masses, offering valuable insights into the anatomical localization, structural characteristics, and tissue involvement of these lesions. By carefully analyzing radiological features and integrating clinical data, radiologists and clinicians can collaboratively formulate an accurate diagnosis and initiate appropriate management strategies. This study will delve into the nuances of sellar and suprasellar masses on CT scans, highlighting key diagnostic considerations and management implications to enhance clinical decision-making and patient outcomes.

Materials and Methods:

The study was carried out in the Department of Radiology and Imaging with collaboration of the Department of Neurosurgery, in tertiary care hospital from July 2004 to June 2005. Total 60 patients were included in this study.

Inclusion criteria included patients with suprasellar masses who have undergone CT examination before surgery and histopathological study post operatively. Patients were selected irrespective of age and sex. Patients who have not undergone operative treatment, those cases where histopathological reports were not available and drop out cases were excluded from this study.

The patients were evaluated by CT scan with contrast and expert opinion taken in every cases. Patient was followed up to completion of surgery. Specimen was sent for histopathological examination.

Results:

Table 1: Distribution of patients of CT scan with symptoms (N=60)

Presenting symptoms	Frequency	Percent
Headache	54	90
Visual problem	47	78.3
Amenorrhoea,galactorrhoea	3	5.0
D.I	4	6.7
Seizure	2	3.3
Ptosis and Ophthalmoplegia	2	3.3
Impaired consciousness	2	3.3
Poor development of secondary sexual character	7	11.6
Growth retardation	6	10.0
Gynaenocomastia and erectile dysfunction	4	6.7
To and fro movement of head	1	1.7
Diplopia	2	3.3
Hypothalamic feature	3	5

Majority of people presented with headache (90%) and visual impairment (78.3%) followed by endocrine deficiency (11.6%). Seizure, Ptosis and ophthalmoplegia, impaired consciousness, diplopia each were present in 3.3% cases as shown in Table 1.

Table 2: Distribution of radiological diagnosis of tumours (N=60)

Radiological Diagnosis	Frequency	Percent
Pituitary adenoma	36	60
Craniopharyngioma	10	16.67
Supra and parasellar meningioma	8	13.34
Hypothalamic glioma	2	3.3
Suprasellar arachnoid cyst	1	1.67
Suprasellar epidermoid	1	1.67
Parasellar schwannoma	0	0
Metastasis	1	1.67
ICA aneurysm	1	1.67
Total	60	100

Table 2 shows among radiologically diagnosed sellar/suprasellar masses, 60% were pituitary adenoma,16.67% craniopharyngioma, 13.34% meningioma, 3.3% hypothalamic glioma and 1.67% of each arachnoid cyst, epidermoid, metastasis and ICA aneurysm each.

Table 3: Distribution of histopathological data (N=60)

Histopathology	Frequency	Percent
Pituitary adenoma	31	51.6
Craniopharyngioma	11	18.3
Adamantinomatus	8	13.3
Papillary	2	3.3
Normal brain tissue	1	1.6
Meningioma	10	16.6
Meningiotheliomatous	5	8.3
Transitional	2	3.3
Fibrous	2	3.3
Psammomatous	1	1.6
Hypothalamic glioma	3	5
Low grade	2	3.3
Oligodendroglioma	1	1.67
Arachnoid cyst	1	1.67
Epidermoid cyst	1	1.67
Schwannoma	1	1.67
Metastasis	1	1.67
ICA aneurysm	1	1.67
Total	60	100

Among the 60 patient's histopathological data 51.6% pituitary adenoma which was highest, 18.3% craniopharyngioma, 16.6% meningioma 13.3% adamantinomatus, 8.3% meningiotheliomatous, Hypothalamic glioma 5%, papillary, Transitional, Fibrous, Low grade are 3.3% each and rest Psammomatous, Oligodendroglioma, Arachnoid cyst, Epidermoid cyst, Schwannoma, Metastasis and ICA aneurysm 1.6% each.

Discussion:

Majority of people presented with headache (93.3%) and visual impairment (78.3%) followed by endocrine deficiency. Seizure, Ptosis and ophthalmoplegia, impaired consciousness, diplopia each were present in 3.3% cases. The endocrine deficiencies include poor development of secondary sexual character, amenorrhoea, galactorrhoea, growth retardation, gynaecomastia and erectile dysfunction (Table 1).

Among radiologically diagnosed sellar/suprasellar masses, 60% were pituitary adenoma,16.67% craniopharyngioma,13.34% meningioma,3.3% hypothalamic glioma and 1.67% of each arachnoid cyst, epidermoid, metastasis and ICA aneurysm (Table 2).

Among the 60 patient's histopathological data 51% were pituitary adenoma which was highest, 18% craniopharyngioma, 13% Adamantinomatus, Papillary 3.3%, Normal brain tissue 1.6%, 16% meningioma,8.3% Meningiotheliomatous, 3.3% Transitional, 3.3% Fibrous, Low grade and rest Psammomatous 1.6%, Oligodendroglioma (1.6%), Arachnoid cyst (1.6%), Epidermoid cyst (1.6%), Schwannoma (1.6%), Metastasis (1.6%) and 1.6% ICA aneurysm (Table 3).

The diagnosis of sellar lesions involves a multidisciplinary effort, and detailed endocrinologic, ophthalmologic and neurologic testing are essential.[5] The most prevalent cause of a tumor in the sella is pituitary adenomas. These tumors produce their symptoms by interference with hormonal functions, compression of the optic chiasma and nerves, or discovered incidentally by imaging procedures done for other reasons.[6] Pituitary adenomas can just affect the sella, but they can also spread inferiorly into the sphenoid sinus, laterally into the cavernous sinuses, and suprasellarly toward the optic chiasm. Adenomas will expand the sella in 94% to 100% of instances. The sella can expand in up to 50% of nonadenomatous tumors, including cysts with Rathke's cleft, meningiomas, and craniopharyngiomas.[7-8] Therefore, the only diagnostically useful factor for a nonpituitary lesion is the absence of sellar enlargement.

Craniopharyngiomas are slow growing tumours, in which the symptoms are related to impingement on adjacent structures, such as hypothalamus, pituitary gland or optic chiasm or the third ventricle and foramina of Monroe with the production of obstructive hydrocephalus.[8]

Meningiomas in the suprasellar and parasellar regions are found in adults. They produce bone reaction with hyperostosis and may cause expansion of underlying sphenoid sinus. They produce symptoms by compression of adjacent structures, such as optic chiasm and optic nerve.[8]

Nonpituitary sellar masses can have other wide range of differential diagnoses, such as vascular lesions, granulomatous, infectious, and inflammatory processes, gliomas, cell rest tumors, and metastatic tumors.

CT still provides some advantage over MR imaging in detecting the presence or absence of tumoral calcification and in the evaluation of bony anatomy. Calcification suggests craniopharyngiomas, meningiomas, chordomas, teratomas, gliomas, or an aneurysm, but pituitary adenomas may also contain calcifications. Erosion of the floor of the sella can be seen with adenomas, intracavernous aneurysms, meningiomas of the middle fossa, Rathke's cleft cyst, arachnoid diverticula, and elevated intracranial pressure from any source.[9] When multiple cut CT has been negative, further diagnostic studies have proved unrewarding. When CT has been positive, additional studies have been required in some cases to rule out aneurysm prior to craniotomy.[10]

Since adenoma cannot always be distinguished from another intrasellar mass, angiography to demonstrate tumor angioarchitecture may be needed to characterize some neoplasms or to confirm an intrasellar aneurysm.[11]

Headache is a common and disabling aspect of pituitary tumour. Chronic migraine, episodic migraine, stabbing headache, cluster headache are predominant complaints.[12] Increased intracranial pressure symptoms and signs could indicate tumours such as craniopharyngioma, meningioma, or germinoma. When ventricular dilatation is caused by big tumors, headache is frequently a major symptom. Patients with intrasellar or suprasellar cysts, as well as inflammatory diseases too small to increase intracranial pressure, may also experience headaches. These patients may have headaches due to diaphragmatic dysfunction or irritation of the parasellar dura.[12]

Visual loss is a common presenting complaint with sellar/parasellar lesions which is similar to present study because of the proximity of the optic nerves, chiasm, and optic tracts to the sella turcica. Because visual loss may be insidious in onset and progress slowly, severe deficits frequently are present before the patient seeks medical attention. In children, in particular, severe visual loss as a result of optic nerve compression by lesions such as craniopharyngiomas may occur before a vision problem is noticed. The particular visual field loss may provide some clue as to the nature of the lesion. Lesions anterior to the chiasm, such as meningiomas of the optic nerve sheath, can produce unilateral visual loss, whereas lesions compressing the visual system more posteriorly along the optic tract, such as meningiomas or aneurysms, can produce homonymous hemianopsias that are characteristically incongruous. Visual deficits from chiasmal tumors may manifest as visual field defects, visual loss, diplopia, nystagmus and visual hallucinations.[13]

Functioning pituitary adenoma can cause amenorrhoea, galactorrhoea, growth retardation, sterility, erectile dysfunction etc. Similar to pituitary adenomas, several nonpituitary sellar and parasellar tumors can exhibit anterior pituitary hormone failure symptoms. Gonadal dysfunction, secondary hypothyroidism, and, less frequently, clinical adrenal cortical insufficiency is among these symptoms. When lesions compress the pituitary, hypothalamus, or infundibulum, children may exhibit growth failure and absence of secondary sexual development.

Clinical diabetes insipidus at presentation is highly suggestive of a nonpituitary etiology of a sellar or parasellar mass. Diabetes insipidus may result from involvement or compression of the pituitary stalk, hypothalamus, or paraventricular region of the third ventricle by the lesion. Vasopressin deficiency may be partial or transient in some patients because regeneration of the vasopressin-containing neurohypophyseal fibers may occur. In addition, the apparent spontaneous improvement of diabetes insipidus in some patients may coincide with the development of hypopituitarism. The syndrome of inappropriate antidiuretic hormone secretion leading to potentially severe hyponatremia may also occur in patients with nonpituitary sellar and parasellar lesions.

Preoperative differentiation of the histological aetiology of masses involving the sella turcica and suprasellar region is of profound clinical importance because it determines the use of surgery versus non-surgical techniques, a transsphenoidal versus an intracranial surgical approach and the degree of resection.[14]

Conclusion:

In conclusion, the differential diagnosis of symptomatic sellar and suprasellar masses by CT scan is a complex yet vital aspect of clinical practice, necessitating a nuanced understanding of anatomical structures, pathological processes, and radiological findings. CT imaging remains integral in the initial evaluation, offering detailed insights into lesion morphology, enhancement patterns, and surrounding tissue involvement. The ability to distinguish between various entities such as pituitary adenomas, craniopharyngiomas, meningiomas, and metastatic lesions is crucial for guiding appropriate management strategies and optimizing patient outcomes. Continued advancements in imaging technology and interdisciplinary collaboration will further enhance our ability to accurately diagnose and manage these challenging conditions, ultimately improving the quality of care for patients with sellar and suprasellar masses.

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Original Article

Status of diabetes mellitus and pre-diabetes among the government civil employees in a selected secondary care hospital in Chattogram division, Bangladesh

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Abstract

Background: Diabetes mellitus (DM) is a chronic metabolic disorder. The prevalence of diabetes and prediabetes are increasing worldwide especially in urban areas.

Objective: This study's objective was to ascertain the frequency of diabetes mellitus, prediabetes, and the underlying risk factors for these conditions among Bangladesh Armed Forces personnel who were admitted to BNS Patenga (Navy Hospital), Chattogram.

Methods: This cross-sectional survey was done among the Bangladesh Armed Forces employees in the Chattogram region, who were admitted in to BNS Patenga (Navy Hospital), Chattogram. A total of 150 samples were selected by convenient sampling. A semi-structured questionnaire and check list was used for data collection. Data on age, gender, food intake, obesity, family history and blood pressure were collected from the study subjects. The ADA Guidelines 2022 were used to define pre-diabetes and diabetes mellitus. SPSS version 19 statistical software was used for data analysis.

Results: This study included 150 civil government employees, out of which 130 males (86.7%) and 20 females (13.3%); 56% were 40-60 years of age. The overall age-adjusted frequency of diabetes mellitus and pre-diabetes among 150 participants were 8% and 10%, respectively. Among them 80 (53%) had sedentary life style, 48 (32%) use to take high calorie food, 33 (22%) had the habit of smoking and family history each, 26 (17%) had Hypertension (HTN) and 17(11%) had obesity. Both prediabetes and diabetes were more prevalent in male, 66.7% and 75.0% respectively.

Conclusion: The prevention of diabetes mellitus and pre-diabetes requires population-based intervention programs and policies that promote lifestyle adjustment and enhanced knowledge on risk factors.

Keywords: Diabetes Mellitus, Prediabetes, Risk factors.

Introduction:

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia. Worldwide people with diabetes mellitus have more than doubled during the past 20 years and this rapid increase is due to emergence of type 2 diabetes in children, adolescents, and young adults. Globally, diabetes mellitus (DM) is among the leading medical conditions that cause mortality. It accounts for 30% of all fatalities.[1] Diabetes is a major cause of cardiovascular disease and also the leading cause of chronic kidney disease (CKD.[2] Data sources from 130 countries representing 382 million people had diabetes in 2013; this number is expected to rise to 592 million by 2035.[3] In South East-Asia (SEA) Region consisting of India, Sri Lanka, Bangladesh, Bhutan, Mauritius and Maldives, is expected to exceed 123 million adults with diabetes in 2035.Nearly 95% of people with diabetes have type 2 diabetes (T2DM). Numerous research studies have demonstrated the direct or indirect effects of gender, age, lifestyle, obesity, smoking, family history of diabetes, dietary habits, and other factors on the onset and course of diabetes.[4]

Diabetes and prediabetes are not as common in emerging nations as they are in wealthy nations. Our clinical experience demonstrates that the urban population of Bangladesh has experienced a sharp rise in the overall prevalence of diabetes and prediabetes in recent years.[5]

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A metabolic condition called diabetes is brought on by unusually high blood sugar levels. Type 1 diabetes, type 2 diabetes, maturity onset diabetes of the young (MODY), gestational diabetes, neonatal diabetes, and secondary causes like suspected endocrinopathies, steroid use, etc. are among the various categories of this complex illness. Type 1 diabetes is typically caused by a deficiency in insulin production and type 2 diabetes by defect in action. Type 2 diabetes usually affects middle-aged and older adults who have high blood sugar levels for an extended period because of poor dietary and lifestyle choices.

Because of their distinct pathophysiology, T1D and T2D have distinct etiologies, symptoms, and courses of treatment.[6] Numerous consequences, such as microvascular, macrovascular, and neuropathic issues, are linked to diabetes. Depending on the severity and length of poorly managed diabetes, microvascular and macrovascular complications can include nephropathy, retinopathy, neuropathy, and atherosclerotic cardiovascular disease (ASCVD) events.[7] These complications are particularly dangerous when dyslipidaemia and hypertension are present. The common complication macroangiopathy in T2D concerns serious heart and vascular lesions that lead to hypertension, artery narrowing, coronary artery disease, stroke and 75% of deaths in these patients are due to underlying coronary artery disease.[8] Heart attacks or strokes claim the lives of about two thirds of diabetics. Higher fasting blood glucose levels in people with type 2 diabetes greatly raise the risk of ASCVD.[7]

In general population both overweight and obesity are increasing due to unhealthy multifaceted lifestyle factors. These are contributing as hidden public health threat to diabetes and prediabetes. Creating awareness and screening of high-risk groups combined with a tailored approach are essential for halting the epidemic of diabetes and pre-diabetes in Bangladesh.[9] The objective of our study was to ascertain the frequency of diabetes mellitus, prediabetes, and the underlying risk factors for these conditions among Bangladesh Armed Forces personnel who were admitted to BNS Patenga (Navy Hospital), Chattogram.

Materials and Methods:

A descriptive cross-sectional study was conducted in BNS Patenga (Navy Hospital), Chattogram in between 01 January 2023 to 30 June 2023 (06 months). One hundred and fifty (150) government civil employees working in Bangladesh Armed Forces at Chattogram area were selected by convenient sampling from admitted patients in BNS Patenga(Navy Hospital), Chattogram. Data were collected with pretested semi-structured questionnaire and checklist by face-to-face interview and reviewing medical and laboratory record. Variables were age, gender, food intake, obesity, physical activity, smoking, sedentary life style, family history of diabetes mellitus, blood pressure and blood sugar status.

Diabetes Mellitus and pre-diabetes were defined according to ADA Guidelines 2022.[10] Institutional approval from proper authority was taken. The participants were briefed properly and informed consent was taken from them.

Information on age, sex, sedentary life style, smoking habit, body weight in kg and Height in cm, by BMI classification (18.5-≤25=normal, 25-<30= overweight, >30= obese), stressful life (pattern of job, working environment, working time, sleeping pattern), family history (history of diabetes or prediabetes etc.) and intake of high caloric diet (>2500 kcal/day).

Population category is as follows:

a. Normal: FBS < 6.1 mmol/L, RBS < 7.8 mmol/L
b. Prediabetes: FBS 6.1-6.9 mmol/L, RBS 7.8-11.0 mmol/L
c. Diabetes: FBS ≥ 7.0 mmol/L, RBS ≥ 11.1 mmol/L

For biochemical test of all measurements were done in fasting condition in the pathology department of the above-mentioned hospital by auto analyzer machine (Model: Dimension Xpand plus with HM, Country of origin: USA).

Data were analysed by statistical software, IBM SPSS version 19. Descriptive frequency was used for data presentation and analysis.

Results:

Table 1: Age group and sex distribution of the population (n=150)

A) Age group (years)	Frequency (n)	Percent (%)
>20-30	18	12
31-40	48	32
>40-50	72	48
51-60	12	8
Total	**150**	**100**
B)Sex	**Frequency (n)**	**Percent (%)**
Male	130	86.7
Female	20	13.3
Total	**150**	**100**

Among 150 participants, 84 (56%) were above 40 years to 60 years and 66(44%) were >20 years to 40 years' age group. Male were 87% and female were 13% as shown in Table 1A and 1B.

Table 2: Distribution of normal population in contrast to prediabetes & diabetes (n=150)

Category	Frequency (n)	Percent (%)
Normal	123	82
Prediabetes	15	10
Diabetes	12	8
Total	**150**	**100**

Table 2 shows, out of 150 participants 15 (10%) were pre-diabetic and 12 (8%) were diabetic; rest 82% was non diabetic.

Table 3: Gender wise distribution of diabetes and prediabetes (n=27)

Category	Male No. (%)	Female No. (%)	Total No. (%)
Prediabete	10 (66.7)	5 (33.3)	15 (100)
Diabetes	9 (75)	3 (25)	12 (100)

Table 3 shows among prediabetics, 66.7% are male and 33.3% are female, among diabetics, 75% are male and 25% are female.

Table 4: Distribution of risk factors of diabetes and prediabetes (n=150)

Risk Factor	Normal No.	Prediabetes No.	Diabetes No.	Prediabetes and Diabetes in risk group No. (%)	Total (%)
Smoking	25	5	3	8 (24%)	33 (22%)
Obesity	11	4	2	6 (35%)	17 (11%)
High calorie food	30	10	8	18 (38%)	48 (32%)
Sedentary lifestyle	58	12	10	22 (28%)	80 (53%)
HTN	20	4	2	6 (23%)	26 (17%)
Family history	24	6	3	9 (27%)	33 (22%)

Table 4 shows among the participants, 80 (53%) had sedentary life style, 48 (32%) use to take high calorie food, 33 (22%) had the habit of smoking and family history each, 26 (17%) had HTN and 17(11%) had obesity.

Discussion:

In comparison to the other groups, majority (56%) of the participants were in the age group of 40 years to 60 years as shown in Table 1A. We observed that 130 (86.7%) of the participants in our study were male and 20 (13.3%) were female (Table 1B). Our results are consistent with a recent study that indicated men of 40 years of age and older had the highest risk of diabetes mellitus.[11]

Ten percent of the participants in this study had prediabetes, and 8% had diabetes (Table 2). One research of Bangladeshi individuals found that the prevalence of diabetes and prediabetes was nearly identical.[12]

In our study, we observed that males were more likely to have diabetes and prediabetes. Table 3 shows that 75.0% of individuals with diabetes and 66.7% of prediabetic cases were male. Some subsequent studies revealed similar results.[11,12]

Furthermore, it was discovered that individuals who used to consume high-calorie foods had the greatest percentage of diabetes mellitus and prediabetes (38.0%) as shown in Table 4. A cross-sectional study conducted at a hospital revealed a favourable correlation between diabetes mellitus and higher calorie intake.[13] According to Table 4, there is a higher prevalence of diabetes mellitus and prediabetes among those who are obese (35%), smoker (24.0%), lead sedentary lives (28%) and have a positive family history (27%). Similar findings have been discovered in several earlier Asian population studies.[14] Physical inactivity decreases insulin sensitivity with progressive loss of beta-cells, leads to impaired glucose tolerance and eventually type 2 diabetes. Physical inactivity can cause obesity which in turn is a significant risk factor for type 2 diabetes.[15] Diabetes is one of the main diseases linked to obesity and high-calorie diets in most western countries. The Bangladeshi population's dietary habits have changed because of the increasing attention and popularity of western meals over time. As a result, it contributes to both prediabetes and diabetes.[16-17] In a study, smokers are 30-40% more likely to develop type 2 diabetes compared to non-smokers.[18] When an individual smokes, the level of nicotine increases in his/her body which leads to a reduction in muscle glucose intake, developing insulin resistance and leading to type 2 diabetes.[19] Compared to the nonhypertensive group in our study, the hypertension group exhibited a higher prevalence of prediabetes and diabetes mellitus (23.0%). Furthermore, an obese individual with hypertension is at higher risk compared to a non-obese. Hypertension is associated with the development of type 2 diabetes in both men and women. However, the association is ethnicity-dependent.[15]

Family history information can serve as a useful tool for prognosis or diagnosis and public health. Family history of diabetes reflects both genetic as well as environmental factors and can lead to better prediction of incidence type 2 diabetes than only genetic factors and environmental factors alone.[20]

To recommend potential interventions based on the risk factor analysis, the study also sought to identify the numerous risk factors for diabetes and prediabetes. We emphasize that people in this age group to keep their BMI within a normal range, cut back on high-fat food intake, improve their physical activity levels, give up smoking, and regularly check their blood pressure, fasting blood sugar and 02 hours after breakfast. Thus, individuals may be able to manage their prediabetes and diabetes more effectively.

Conclusion:

According to the study's findings, prediabetes and diabetes are prevalent among Bangladesh Army officials. Additionally, high frequency of diabetes and pre-diabetes was found among those with high calorie food intake, smoking, obesity, family history of diabetes, sedentary lifestyle, and hypertension. The study's findings on diabetes and pre-diabetes condition highlighted the necessity of comprehensive and holistic policy to manage this issue for employees. To ascertain the connection between pre-diabetic prevalence and diabetes, more investigation is required. Additionally, we advise Bangladeshi government workers to maintain a low-calorie diet, stop smoking, exercise frequently, and manage their high blood pressure. Periodic survey that include the demographic and lifestyle features of the citizens will give beneficial outcome. And that oiutcome can be used along with allied health professionationals to develop a nation - wide diabetes prevention plan.

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Review Article

Metabolic dysfunction-associated steatotic liver disease: Beyond the fatty liver

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Abstract

Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as a significant global health challenge. Changing lifestyles, over-nutrition, and physical inactivity have promoted its development. Closely linked to obesity and metabolic syndrome, MASLD represents a shift in terminology from non-alcoholic fatty liver disease (NAFLD), reflecting a more precise understanding of its pathogenesis. Beyond liver-related complications, MASLD is associated with an increased risk of cardiovascular disease, chronic kidney disease, and certain extrahepatic cancers. The disease exhibits heterogeneity in its clinical manifestations due to complex molecular pathways involved in its pathogenesis. The expanding role of noninvasive techniques, such as serum biomarkers and advanced imaging modalities, have improved risk stratification and identification of individuals at high risk for fibrosis progression. This comprehensive narrative review summarizes the global incidence and prevalence rates of MASLD and its related adverse hepatic and extrahepatic outcomes. We also highlight recent nomenclature changes, the clinical relevance of early intervention and emerging pharmacological therapies.

Keywords: Metabolic dysfunction-associated steatotic liver disease, Non-alcoholic fatty liver disease, Cardio-metabolic risk factor, Fatty liver.

Introduction:

Nonalcoholic fatty liver disease (NAFLD) has emerged as a prominent cause of chronic liver disease globally.[1] Since June 2023, NAFLD nomenclature has been changed to metabolic dysfunction-associated steatotic liver disease (MASLD). It is commonly associated with cardiometabolic risk factors. Due to worldwide epidemics of metabolic syndrome, obesity, and type 2 diabetes, the prevalence of MASLD has been increasing over time. Recent meta-analysis estimating that more than one-third of the adult population are afflicted by MASLD.[2] About 75% of people with obesity[2] and 69% of people with type 2 diabetes[3] have concomitant MASLD.

MASLD consists of two clinical entities: metabolic dysfunction- associated steatotic liver (MASL, previously NAFL) and metabolic dysfunction-associated steatohepatitis (MASH, previously called NASH). While simple steatosis does not often progress, patients with MASH are at risk of progressive liver injury that can advance to cirrhosis and the development of hepatocellular carcinoma. Progressive MASH is characterized histologically by steatosis, lobular inflammation, and hepatocyte ballooning with varying degrees of fibrosis.[4,5]

MASLD is mostly asymptomatic, although a proportion of patients report fatigue and right upper quadrant abdominal pain. MASLD is suspected by identifying commonly associated risk factors, finding steatosis on imaging and excluding other causes of chronic liver disease. Early diagnosis and management of MASLD are essential in preventing the progression to severe forms of liver diseases.[6]

This review will focus on the most recent data on MASLD epidemiology, pathogenesis, risk prediction, diagnostic strategies, and current and emerging management approaches.

Epidemiology of MASLD:

Eestimated global incidence of NAFLD is 47 cases per 1,000 populations.[7] The global prevalence of MASLD has increased over time, from 26.0% in 1990-2006 to 38.0% in 2016-2019.[8,9] The prevalence varies by race and ethnicity.

The highest prevalence observed in Latin America 44.37%, then Middle East and North Africa 36.53%, South Asia

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33.83%, South-East Asia 33.07%, North America 31.20%, East Asia 29.71%, Asia Pacific 28.02%, and Western Europe 25.10%.[10] These trends are expected to grow as the global prevalence of MASLD is forecasted to reach 55.4% by 2040.[11] The prevalence is higher among males (40%) compared to females (26%).[8] Although there is limited data on the prevalence of MASLD in Bangladesh, it is also experiencing an increasing trend of MASLD due to changing dietary patterns and sedentary lifestyles. A study conducted in 2018, showed that the overall prevalence of MASLD was 33.86% in Bangladesh.[12]

Background of new nomenclature:

Since the introduction of non-alcoholic fatty liver disease (NAFLD) in 1980, numerous efforts have been made to redefine the disease terminology.[13] In 2020, an international panel of experts proposed the term metabolic dysfunction–associated fatty liver disease (MAFLD) to better reflect its metabolic origins.[14] Following a four-round Delphi process, in June 2023, an international panel of 225 participants, led by three multinational liver associations, officially renamed NAFLD as MASLD.[14,15] This change also introduced steatotic liver disease (SLD) as an umbrella term, encompassing MASLD, MASLD with moderate alcohol intake (MetALD), alcohol-related liver disease (ALD), specific etiologies (e.g., drug-induced or monogenic causes), and cryptogenic SLD.

The new nomenclature emphasizes the role of metabolic dysfunction in fatty liver disease and aligns more closely with its underlying pathophysiology. MASLD is defined as hepatic steatosis in the presence of one or more cardiometabolic risk factor(s) (Table 1) and the absence of harmful alcohol intake.

Table 1. Cardiometabolic risk factors and cut off values

Metabolic risk factor	Adult criteria
Overweight or obesity	Body Mass Index ≥25 Kg/m^2 (≥23 Kg/m^2 for Asians) Waist circumference • ≥94 cm in men and ≥80 cm in women (Europeans) • ≥90 cm in men and ≥80 cm in women (South Asians and Chinese) • ≥85 cm in men and ≥90 cm in women (Japanese)
Dysglycaemia or type 2 diabetes	Prediabetes: HbA1c 39-47 mmol/mol (5.7-6.4%) or fasting plasma glucose 5.6-6.9 mmol/L (100-125 mg/dl) or 2-h plasma glucose during OGTT 7.8-11 mmol/L (140-199 mg/dl) or Type 2 diabetes: HbA1c ≥48 mmol/mol (≥6.5%) or fasting plasma glucose ≥7.0 mmol/L (≥126 mg/dl) or 2-h plasma glucose during OGTT ≥11 mmol/L (≥200 mg/dl) or On treatment for type 2 diabetes
Plasma triglycerides	Plasma triglycerides ≥150 mg/dl (≥1.70 mmol/L) or lipid- lowering treatment
HDL-cholesterol	Plasma HDL-cholesterol <40 mg/dl (<1.0 mmol/L) for men and <50 mg/dl (<1.3 mmol/L) for women or lipid- lowering treatment
Blood pressure	Blood pressure ≥130/85 mmHg or on treatment for hypertension

HbA1c (glycated hemoglobin); HDL (high-density lipoprotein); OGTT (oral glucose tolerance test).

The updated terms MASLD and MASH (metabolic dysfunction–associated steatohepatitis) replace the previous NAFLD and NASH terminology, addressing key limitations such as exclusionary definitions and potentially stigmatizing language. The previous nomenclature also failed to reflect the disease's multisystem involvement and metabolic origins.[15]

Additionally, the new classification introduces MetALD, a category for individuals with hepatic steatosis, cardiometabolic risk factors, and moderate alcohol consumption (20–50 g/day for females and 30-60 g/day for males), differentiating them from MASLD and heavy alcohol-related liver disease. Individuals with steatosis but no identified cardiometabolic risk factors fall under "possible MASLD", requiring further evaluation for insulin resistance or cryptogenic SLD.[15]

The pathogenesis of MASLD:

The two-hit hypothesis was initially proposed to explain the pathogenesis of MASLD. In this model, hepatic fat accumulation promotes insulin resistance (the "first hit"), which subsequently triggers inflammatory mechanisms and fibrosis (the "second hit"). However, this hypothesis is being reconsidered as new theories emerge. Research suggests that MASLD results from a complex interplay of genetic, dietary, metabolic, and environmental factors.[16]

Certain genetic polymorphisms have been linked to advanced liver disease and the development of hepatocellular carcinoma (HCC) in MASH.[17] The most

significant genetic variants influencing MASLD susceptibility and progression include: PNPLA3 (patatin-like phospholipase domain-containing protein 3)[18] and TM6SF2 (transmembrane 6 superfamily member 2).[19] PNPLA3– mutations in this gene are associated with increased hepatic lipid content. TM6SF2– mutations lead to loss of function in hepatic VLDL secretion, increasing susceptibility to liver damage.[20]

Accumulating evidence suggests that mitochondrial dysfunction plays a significant role in steatosis and steatohepatitis. Impaired β-oxidation of lipids leads to the overproduction of reactive oxygen species (ROS) in hepatocytes.[21] Excessive ROS triggers lipid peroxidation, protein damage, and DNA injury, creating a pro-inflammatory environment.[22,23] The resulting oxidative stress sensitizes hepatocytes to injury and apoptosis, exacerbating liver inflammation.[24]

Emerging evidence suggests that changes in the gut dysbiosis might play a vital role in the development and progression of MASLD from simple steatosis to steatohepatitis and even HCC. Changes in the gut microbiome can alter the production of short-chain fatty acids and increase intestinal permeability, allowing bacterial endotoxins to enter the portal circulation.[25,26] These endotoxins activate hepatic Toll-like receptors (TLRs), particularly TLR4, triggering inflammation and fibrosis.[27]

Chronic liver injury and inflammation activate hepatic stellate cells (HSCs), which produce extracellular matrix proteins, leading to scar tissue formation (fibrosis). As fibrosis progresses, it causes architectural distortion of the liver, significantly increasing the risk of cirrhosis and HCC.[28]

Diagnosis and Diagnostic Modalities:

The diagnosis is made by combining clinical assessment, along with laboratory tests and imaging. Most of the patients are asymptomatic, and the disease is often overlooked. Some patients may present with non-specific symptoms, such as fatigue, right upper quadrant discomfort, or epigastric fullness. In early disease stages, hepatomegaly may be the only physical finding. In advanced disease (cirrhosis), signs such as splenomegaly, spider angiomata, palmar erythema, or ascites may be present. MASLD is often suspected with the finding of hepatomegaly on physical examination, or incidentally on abnormal liver function tests (elevated liver enzymes) or on abdominal imaging.[17,29]

Elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are common but not specific for MASLD. Fasting glucose, HbA1c, lipid profile, and serum triglycerides aid in identifying metabolic dysfunction. Assessment of insulin resistance (e.g., using the Homeostasis model assessment of insulin resistance [HOMA-IR] or estimates derived from the oral glucose tolerance test) may be considered to clarify metabolic dysfunction in adults with suspected MASLD and without an established diagnosis of type 2 diabetes mellitus (T2DM). Additional tests (e.g., autoimmune markers, viral hepatitis panels, serum ceruloplasmin) may be required to exclude other liver diseases.[17,30]

In clinical practice, ultrasonography (USG) is the first-line imaging exam used for the diagnosis of hepatic steatosis because of its wide availability and lower cost. The sensitivity and specificity of the USG are 89% & 93%, respectively. The grading of liver steatosis is usually obtained using some USG features that include liver brightness, contrast between the liver and the kidney, USG appearance of the intrahepatic vessels, liver parenchyma and diaphragm. However, accuracy is reduced in morbid obesity.[31] Vibration-controlled transient elastography (VCTE), commonly known as FibroScan, is a noninvasive technique with higher sensitivity and specificity. It measures controlled attenuation parameter (CAP) and liver stiffness to assess liver steatosis and fibrosis, respectively.[31] Magnetic resonance proton density fat fraction (MRI-PDFF) is considered the gold standard for quantifying hepatic fat, but it is not yet widely available, and it is also very costly.[32] CT scan is not considered the best modality for MASLD due to higher cost, potential radiation exposure, and limited sensitivity in detecting mild steatosis.[17] Liver biopsy is the gold standard for assessing fibrosis stage and distinguishing MASH from simple steatosis. However, due to its invasive nature and risk of sampling error, it is reserved for unclear cases or suspected advanced disease.[33]

Risk Assessment of Patients with MASLD:

Liver enzymes may be a first-step to assess and monitor patients with liver diseases. However, serum liver enzyme concentrations can be normal in more than half of patients with MASLD, and correlate poorly with the histological severity. Traditionally, liver biopsy was used to characterize and quantify histological features of steatosis, inflammation, hepatocyte ballooning, and fibrosis but it has several limitations. Several noninvasive tests composed of demographic, clinical, and routine laboratory parameters have been developed and can be applied easily in the community. Available tools are NAFLD fibrosis score (NFS), Fibrosis-4 (FIB-4) index and AST-to-platelet ratio index (APRI), Enhanced Liver Fibrosis (ELF) panel, Fibrometer, FibroTest.[34] Among these tools, FIB-4 is the most well validated and recognized by most guidelines as a useful tool in identifying patients with higher likelihood of advanced liver fibrosis (F3 or F4).[17,30]

Although the overall sensitivity of FIB-4 is assumed not to be high, it has high negative predictive values to exclude advanced liver fibrosis. Patients with low FIB-4 score are also at a low risk of developing liver-related complications. Patients with high FIB-4 score should undergo a second noninvasive test of higher sensitivity.[35] Vibration-controlled transient elastography (VCTE) or MR elastography (MRE)

are commonly used due to its availability and can estimate both hepatic steatosis and liver stiffness more accurately.[36] The noninvasive tests are used to identify individuals at high risk of progressive fibrosis who may benefit from more comprehensive management. Patients with T2DM or abdominal obesity and ≥1 additional metabolic risk factor(s) or persistently elevated liver enzymes should be screened for advanced fibrosis. Usually, a multi-step process is used. First, an inexpensive simple fibrosis score (e.g., FIB-4) is used to categorize patients according to their risks. Then individuals with a relevant risk profile should follow different pathways depending on the result of this test.[17,30]

Patients with FIB-4 less than 1.3 can be followed in the primary care setting and reassessed every 1-3 years. Patients with FIB-4 >2.67 (or >2.0 in individuals aged >65) are categorized as high-risk group and in these patients assess with alternative noninvasive test (e.g., VCTE) to clarify the stage of fibrosis. Patients with FIB-4 between 1.3 to 2.67 are categorized as intermediate risk group and can proceed to elastography or undergo a 1-year intervention of lifestyle change and intensified management of cardiometabolic risk factors. If the re-tested FIB-4 level is still elevated after 1 year, VCTE is recommended as the second step to clarify the stage of fibrosis.[30]

Management of MASLD:

The cornerstone of MASLD management are lifestyle modifications, including weight loss, a healthy diet, and regular exercise. In addition to lifestyle changes, the management of MASLD also focuses on addressing underlying metabolic risk factors, preventing disease progression, and mitigating complications.

- **Lifestyle Modification**
  Lifestyle intervention should be emphasized at all levels of patient care with the goal of improvements in both MASLD and the cardiometabolic and overall health of the individual patient. Evidence suggests that comprehensive changes in diet and physical activity can significantly improve liver histology, reduce hepatic fat, and address associated metabolic conditions such as obesity and T2DM.[37] Lifestyle interventions are effective even in advanced stages of MASLD, including non-alcoholic steatohepatitis (MASH) and fibrosis.

Diet

The Mediterranean diet (MD) is widely recognized as a leading dietary approach for managing MASLD. Several studies consistently demonstrated that the Mediterranean diet offers significant benefits for liver and cardiovascular health.[38,39] This dietary pattern has been shown to reduce liver fat accumulation and improve insulin sensitivity in individuals with MASLD and insulin resistance, even without weight loss.[38] This diet emphasizes whole grains, fish, monounsaturated fats, antioxidants, polyphenols, vitamins, fiber and olive oil, while maintaining a low intake of sugars and refined carbohydrates, saturated fat, ultra-processed foods, and red and processed meat.[39]

Regular coffee consumption has been found to offer protective effects against MASLD and liver fibrosis. It is also significantly associated with decreased risk of liver fibrosis development in already diagnosed MASLD patients. A meta-analysis encompassing diverse observational studies revealed that individuals who consumed coffee had a lower risk of MASLD and fibrosis compared to non-coffee drinkers.[30,40] The findings indicated a more robust and consistent protective effect against fibrosis than steatosis. Similarly, an earlier meta-analysis showed that intake of ≥3 cups of coffee per day (vs. <2 per day) was related to reduced risk of MASLD. In a nationally representative cross-sectional study, >3 cups of coffee daily were independently associated with lower liver stiffness but not steatosis as measured by CAP among US adult.[41]

A comprehensive meta-analysis performed in 2023 investigated the impact of intermittent fasting (IF) on cardiometabolic and hepatic indicators in individuals with MASLD. Although IF regimens can improve some markers of cardiometabolic and liver function, the available evidence to support the benefits of IF regimens is limited and derived from a small number of studies. Thus, further research is needed to clarify the impact of IF on the cardiometabolic health of MASLD patients.[39]

Physical exercise

Exercise, independent of weight loss, has hepatic and cardiometabolic benefits and should be routinely recommended and tailored to the patient's preferences and physical abilities. Aerobic and/or resistance training (30 mins/day, 5 days/week, 150min/week) with the goal of 400 kcl loss per day can prevent or improve MASLD.[17,42-45]

Weight loss

In all adults with MASLD and overweight or obesity, dietary and behavioral therapy-induced weight loss should aim at a sustained reduction of ≥5% to reduce liver lipid content, 7-10% to improve liver inflammation, and ≥10% to improve fibrosis.[17,27,38] In people with lean MASLD, weight loss is still beneficial, the recommended targeted weight loss is 3-5%. A study on patients with biopsy-proven MASH demonstrated that this could result in the resolution of MASH and improvement in liver fibrosis in up to 90% and 45% of patients, respectively.[38] A 5-10% bodyweight loss could be a challenging goal for many patients if they only do exercise or follow a diet. Therefore, bariatric surgery is an option for some patients who cannot achieve the goal of losing 0.5-1 kg/week.[46]

- **Pharmacological therapy**
  In some cases, diet and lifestyle measures cannot be successfully or sustainably implemented. Pharmacological treatment is an option when non-pharmacological treatment fails, or when the patients already have advanced disease. The majority of drugs are

used to control CV risk factors and to help people to lose weight. Liver-targeted therapy, especially for NAFLD, is limited.

MASLD/MASH directed therapy

Thyroid Hormone Receptor Beta (TR-β) Agonists

Resmetirom is a thyroid hormone receptor beta (THR-β) agonist. Activation of THR-β in the liver improves liver enzymes, LDL-C, triglyceride, and lipoproteins by modulating hepatic lipid metabolism and this signaling pathway has been identified as a promising target to treat MASLD and hypercholesterolaemia.[47] Individuals receiving resmetirom should be monitored for gastrointestinal side effects and thyroid hormone function.

Vitamin E

Vitamin E has antioxidant, anti-inflammatory, and anti-apoptotic properties. It reduces liver lipid content by inhibiting de novo lipogenesis. Broad population-level analyses indicate that higher consumption of dietary vitamin E may lower the risk of metabolic dysfunction-associated steatotic liver disease (MASLD), based on both clinical assessments and imaging findings, especially in people with T2DM.[48] In the PIVENS randomized controlled trial, treatment with 800 IU of natural vitamin E (α-tocopherol) daily for 96 weeks led to significant histological improvement—defined as a reduction of at least 2 points in the NAFLD Activity Score-compared to placebo in nondiabetic individuals with NASH.[49] Case-control studies suggest that prolonged use of vitamin E may lower the chances of mortality, liver transplantation, and liver function decline in people with MASH who also have bridging fibrosis or cirrhosis.[50] Although some smaller studies have indicated that vitamin E may help lower liver enzyme levels, there is still no conclusive evidence that it improves fibrosis, and a large-scale phase III clinical trial has yet to be carried out.[30]

Targeting MASH through glycemic and body weight control

Patients with MASLD have 2-3-fold increased risk of subsequent diabetes.[51] There is a bidirectional relationship between NAFLD and T2DM i.e. the possible involvement of fatty liver disease in patients with T2DM, and, in turn, the possibility of these T2DM in patients with MASLD.[52] Patients with MASH should be screened for T2DM and vice versa. The insulin resistance may result in development of both MASLD and T2DM, thus, the same management principle can be applied for both. Several antidiabetic medications have a role not only treating hyperglycemia per se but also reduce hepatic steatosis, liver enzyme levels, and inflammation, with some potential role in fibrosis.

GLP-1 receptor agonists (GLP1 RAs)

Glucagon-like peptide 1 (GLP-1) is an endogenous gut hormone (incretin) that promotes insulin production and release, inhibits glucagon secretion indirectly. In addition to glycemic control, GLP-1 RAs also reduce appetite and have a weight loss effect.[53] A number of clinical studies documented beneficial effects of GLP- 1RAs in patients with MASLD, mainly in those with concomitant T2DM.[54-60] Given these robust benefits, GLP1 RAs have emerged as the key pillars for managing people with T2DM and/or obesity.[61] Although, several studies have reported positive effects of GLP-1 analogs on liver health, particularly in reducing hepatic steatosis and improving MASH, the impact on hepatic fibrosis appears to be less significant.[62]

GLP-1 and GIP receptor co-agonists (GLP1-GIP RAs)

Tirzepatide is the first dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon- like peptide-1(GLP-1) receptor agonist that has been approved for the treatment of T2DM and obesity.[63] Tirzepatide was found superior to GLP-1 receptor agonists to decrease bodyweight, both in people with and without T2DM.[64] Tirzepatide, together with a marked improvement in glucose metabolism and cardiometabolic risk factors, is becoming an attractive therapeutic option for MASLD/MASH patients with coexisting T2D and obesity.[65]

Sodium-glucose cotransporter type 2 (SGLT2) inhibitors

SGLT2 inhibitors induce renal glucosuria and weight loss which could be beneficial in patients with MASLD. These agents have shown great advantages in patients with heart and kidney failure patients, even in patients with no diabetes. SGLT2i seem to be promising agents for MASLD treatment, since they could inhibit liver steatosis via a variety of mechanisms.[66,67,68,69] A recent study on the outcomes of various classes of oral antidiabetic drugs (OADs) on MASLD demonstrated that SGLT2 inhibitors might be preferred over other OADs (thiazolidinediones, DPP-4 inhibitors, and sulfonylureas) with respect to MASLD regression and composite liver-related outcomes using a well-established Korean nationwide cohort.[70] These findings also demonstrated more favorable outcomes associated with the use of SGLT2 inhibitors, including MASLD improvement, compared with thiazolidinediones.[71]

Peroxisome proliferator-activator receptor (PPAR) agonists

In several RCTs, pioglitazone, a thiazolidinedione which mainly activates PPAR- γ, has been shown to improve histological features of steatohepatitis,[72,73,74] without a clear effect on fibrosis regression even after prolonged (3-year) therapy with pioglitazone.[73] The clinical use of pioglitazone is limited due to availability of newer antidiabetic agents and side effects, such as the risk of fluid retention, heart failure, weight gain, risk of bladder cancer and a potential increase in bone fractures.[75,76,77]

Saroglitazar, a dual PPAR α/γ agonist, enhances β-oxidation of fatty acids, reduces triglyceride levels, improves insulin sensitivity and glucose uptake.[78,79] Saroglitazar was first approved in in 2013 for the management of diabetic dyslipidemia and in 2020, it received approval for MASLD and non-cirrhotic MASH by DCGI in India.[80] In patients with MASLD, saroglitazar has been shown positive benefits in glycemic control and lipid profile, along with reduction of liver fat, fibrosis and elevated liver enzymes in various studies.[81-84]

Metformin, dipeptidyl peptidase-4 inhibitors

Clinical studies demonstrated that metformin reduces liver enzymes and improves insulin sensitivity.[85-89] Currently, there is no actual evidence shows that the use of metformin shows that the use of metformin alone can improve histology in MASH.[30,89] Observational and case-control studies showed that, in people with T2D and MASLD-related advanced fibrosis or cirrhosis, metformin may improve transplant-free survival, and reduce the risk of primary liver and extrahepatic cancer.[90,91] Metformin contributes to intestinal barrier integrity and prevents bacteria translocation from the gut to the bloodstream, it may have a role in preventing hepatic encephalopathy.[89] Metformin can be used in adults with compensated cirrhosis and preserved renal function but should not be used in adults with decompensated cirrhosis, especially when there is concomitant renal impairment, because of the risk of lactic acidosis.[30]

Given the lack of efficacy data, dipeptidyl peptidase-4 inhibitors,[92] alpha-glucosidase inhibitors, and insulin are not recommended for the treatment of MASLD. However, these anti-hyperglycemic drugs can be tailored for hyperglycemia among people with T2DM and MASLD.[93]

Therapy targeting cardiovascular disease risk reduction:

Currently, CVD remains the leading cause of death in patients with MASLD. Due to the close association between MASLD and cardiovascular disease, regular evaluation of CVD risk factors is crucial. CVD risk factors should be screened for regularly and treated aggressively, focusing on lipid-lowering agents, smoking cessation, treatment of hypertension, glycemic control in patients with diabetes and weight loss through diet changes and exercise. SGLT2 is and GLP-1 RAs both offer cardiovascular benefits and support weight reduction, positioning them as encouraging therapeutic options for individuals with N2DM and cardiovascular disease risk.[94]

Lipid-lowering Drugs

Dyslipidemia affects approximately 60-70% of those with MASLD.[95] Moderate-intensity to high-intensity statins are the first line treatment of dyslipidemia in MASLD based on lipid risk levels and atherosclerotic CVD risk scores. Statins seem to be under-prescribed and several studies have shown a favorable safety profile for the use of statins in MASLD.[96-98] Moderately elevated transaminases should not preclude initiation of statins in MASLD. Except for decompensated cirrhosis, patients with all other stages of MASLD, including transplant, should be optimally treated. In the context of hypertriglyceridemia, the use of TG-lowering medications, such as fibrates, is advisable in cases of severe hypertriglyceridemia (fasting TG level ≥ 500 mg/dL).[99] There is a large but nevertheless inconsistent body of evidence supporting the use of omega-3 fatty acids in MASLD.[100] Non-statin medications are to be used as adjuncts when treatment goals are not achieved with statins, or in cases of statin intolerance.[101]

Anti-hypertensive drugs

A high prevalence of MASLD was observed among patients with hypertension and vice versa.[102-104] Lowering blood pressure is also vital to reduce CVD risk. The preferred agents with proven cardiovascular benefits are drugs that block the renin–angiotensin–aldosterone system (RAAS).[102]Angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) have been shown to exert protective effects against liver fibrosis.[105] Although several studies indicated the beneficial effects of these drugs, the current evidence is insufficient to support the efficacy of RAAS blockers in managing fibrosis in MASLD patients.

Management of MASLD: Bariatric surgery

Bariatric surgery has long-term beneficial effects on the liver and is associated with remission of T2DM and improvement of cardiometabolic risk factors. In the setting of obesity class III (BMI ≥40 kg/m2) or obesity class II (BMI ≥35 kg/m2) and comorbidities (T2DM or pre-DM, uncontrolled hypertension, osteoarthritis, polycystic ovarian syndrome, or sleep apnea), bariatric surgery should be considered, especially for patients at high or very high risk of CVD.[106]

Management of MASH-related cirrhosis

Once the patients develop cirrhosis, treatment should focus on preventing hepatic decompensation and managing complications of cirrhosis, including management of portal hypertension, sarcopenia and monitoring for hepatocellular carcinoma (HCC). Liver transplantation is the definitive treatment for end-stage MASH-related decompensated cirrhosis and/or HCC.[46] In adults with compensated advanced chronic liver disease but LSM ≥20 kPa and/or platelet count <150 × 109/L, an upper gastrointestinal endoscopy should be performed to screen for varices. may be considered for HCC surveillance. Surveillance with 6-monthly ultrasound examination of the liver with or without serum alpha-fetoprotein level is recommended in patients with advanced fibrosis or ≥F3 fibrosis with LSM-VCTE ≥16.1 kPa.[30]

Conclusion:

MASLD is one of the common liver diseases and is the leading cause of liver-related morbidity and mortality. It is commonly associated with metabolic syndrome such as obesity, dyslipidemia, and insulin resistance. Early diagnosis and management of comorbidities are essential to prevent disease progression. A multidisciplinary approach involving hepatologists, endocrinologists, cardiologists, and nutritionists is essential for better patient outcomes. The management of the underlying metabolic disorders by lifestyle modification and weight loss is the cornerstone of the treatment of MASLD/MASH. Despite the limited number of MASH-specific drugs, some drugs used to treat the comorbidities have some potential efficacy in slowing down disease progression. Several MASH-specific drugs are on the horizon and will likely change our management in the near future.

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Severe plaque psoriasis with systemic lupus erythematosus: A case report with comorbidities

Afrin S

Abstract

This is a case report of a 45-year-old female patient who was diagnosed with severe plaque psoriasis and systemic lupus erythematosus (SLE) with comorbidities, including hypertension, rheumatoid arthritis, diabetes mellitus with diabetic nephropathy (grade II), and lupus nephritis (Class I). The patient had been experiencing severe itching and burning sensation for a year and a half and had been diagnosed with SLE in July 2021. The case discusses the patient's symptoms, diagnosis, and treatment. Patient examination revealed multiple joint tenderness, swan neck deformity, boutonniere deformity of the thumb and erythematous medium to large-sized plaques on various body parts. The case also includes investigation results, such as CBC, creatinine, SGPT, electrolyte levels, Hbs Ag, anti-ds DNA, ANA/ANF, and skin biopsy for histopathology. The diagnosis was severe plaque psoriasis with SLE, rheumatoid arthritis, diabetes mellitus with diabetic nephropathy (grade II), and hypertension. The treatment included various medications and topical ointments. This case report highlights the challenges in managing patients with multiple comorbidities and the importance of a comprehensive approach to patient care.

Keywords: Severe plaque psoriasis, Systemic lupus erythematosus (SLE), Comorbidities.

Introduction:

Immune-mediated inflammatory diseases (IMIDs) are a set of chronic diseases and the manifestation of the pathological immune response and constant inflammation or tissue injury.[1] Despite the common features in terms of genetic susceptibility and disease pathways, the presence of multiple IMIDs in a patient may have diagnostic and management implications that are distinct from those in patients with solitary IMIDs.[2] This case report describes the successful therapeutic approach initially in a 45-year-old with severe chronic plaque psoriasis, systemic lupus erythematosus, and other diseases, complicating the process. Psoriasis is a chronic skin disorder that, like most skin conditions, shows frequent relapses and is estimated to affect 2-3% of the world's population. This is through increasing the rate of division and making the keratinocytes change dimensions and create what appears as erythematous scaly plates. Psoriasis as a skin disorder has genetic predisposition factors, environmental factors, and immunological changes, and the main attention is paid to the pro-inflammatory cytokines IL-23 and Th-17.[3] Psoriasis is a chronic inflammatory skin disease and is divided into five types, but plaque psoriasis is the most common type of the disease that can dramatically affect the patient's quality of life and can be related to cardiovascular disease and metabolic syndrome. On the other hand, systemic lupus erythematosus is a chronic multisystem autoimmune disorder due to the presence of autoantibodies directed against nuclear antigens that produce inflammation and tissue damage of various internal organs. SLE has been described to affect skin, joints, kidneys, the body's central nervous system, and other organs and present in different clinical variations. SLE has a multifactorial, polygenic heritage, and there are genetic, environmental, as well as hormonal influences that impact the immune system and self-tolerance.[3,4] It is quite unusual to have both psoriasis and lupus in the same patient; however, it has been found in approximately 0. 8% among patients with eczema, but mortality during the therapy was 100%. For example, it was reported to be 1% among the SLE patients. Despite these differences, their low incidence is linked to the differences in genetic susceptibility and immune response alterations that characterize these two diseases. While psoriasis is mostly T cell mediated inflammation involving Th17 cells, SLE is associated with B cell synchronization and autoantibody synthesis. The above-mentioned diseases can be present at the same time making it challenging in diagnosing, treating and coming up with management plans.[5]

Case Presentation:

A 45-year-old female housemaid from Mohammadpur, Dhaka, Bangladesh, attended the dermatology and venereology department of Bangladesh Medical College Hospital with erythematous multiple medium- to large-size plaques containing silvery white scale. The lesions affected the head and neck, chest, abdomen, back, forearms, arms, and lower limbs. These lesions had started developing one and a half years before the beginning of intense itching and burning. The patient was diagnosed with SLE and lupus nephritis (Class I) in July 2021. She also had hypertension, rheumatoid arthritis, and diabetes mellitus with grade 2 diabetic nephropathy since February 2009. There was no family history of psoriasis or SLE. The patient was able to ambulate and was oriented to place, person, and time.

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She had a blood pressure of 150/100 mmHg, a pulse of 88/min, and a respiratory rate of 16b/min, with no fever. She had multiple joint pain and displayed both swan neck deformity and boutonniere deformity. The skin examination revealed that the patient had erythematous plaques with silvery scales in the chest, back, abdomen, limbs, scalp, and face. Auspitz sign was positive. PASI score represented a case of severe psoriasis that is erythematous, scaling, and indurated severer than third, involving more than a quarter of body surface area.

Reports of Investigations
• ANA/ANF-Reactive
• Anti ds DNA-Reactive
• Anti-CCP-77.40 U/ml
• RA Test-Positive
• iPTH-285.80 pg/ml
• Phosphate-3.30 mg/dl
• Calcium-9.01 mg/dl
• C3-1.28 gm/L
• C4-0.31 gm/L

Skin biopsy revealed features suggestive of psoriasis, including hyperkeratosis, parakeratosis, acanthosis, and Munro microabscesses. The diagnosis was severe plaque psoriasis with SLE, lupus nephritis (class I), rheumatoid arthritis, diabetes mellitus with diabetic nephropathy (grade II), and hypertension.

Treatment
Multidisciplinary Treatment approach was initiated:
• Deucravacitinib: 6mg daily
• Antihistamine
• Topical Potent Steroid
• Liquid Paraffin
• Anti-Hypertensive Medication
• Anti-Diabetic Medication

Discussion:

The patient presented with multiple medium- to large-size plaques in different body parts such as the scalp, chest, abdomen, back, forearms, arms, and lower limbs. She complained of itching and burning sensations that lasted for about one and a half years before she decided to consult a doctor. The patient was diagnosed with SLE in July 2021 and has lupus nephritis (Class I), hypertension, and rheumatoid arthritis. Moreover, she had a past medical history of diabetes mellitus with grade 2 diabetic nephropathy since February 2009.

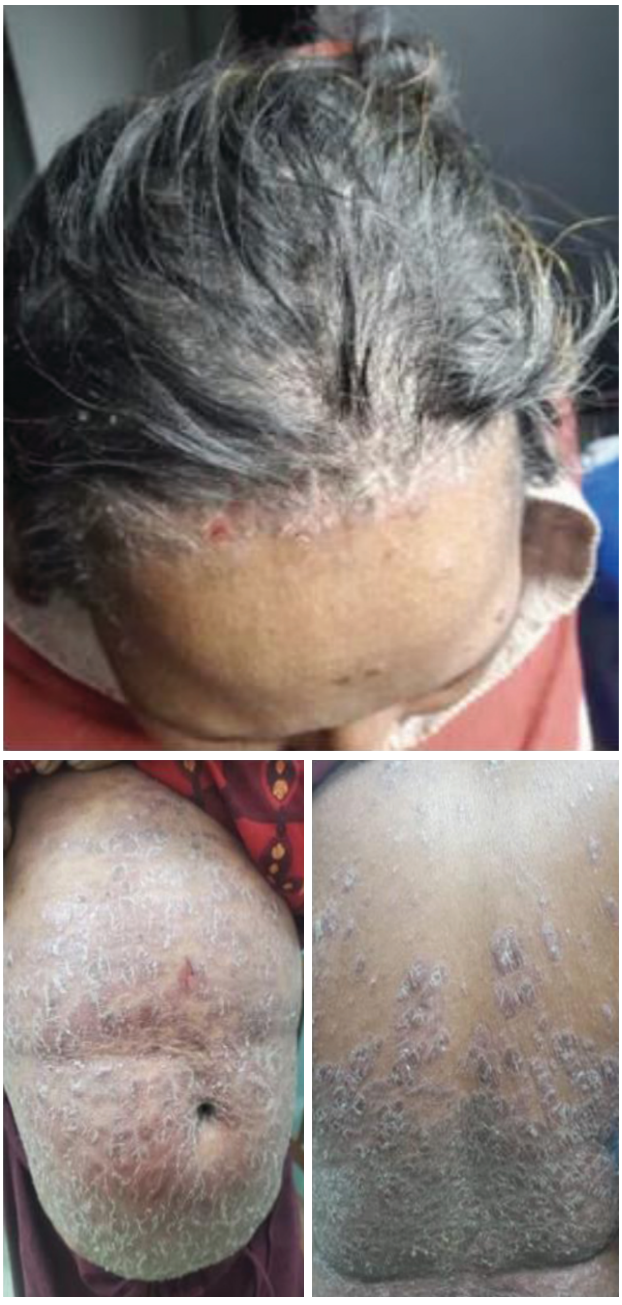

Figure 1: First visit, Plaque psoriasis on scalp, abdomen, back

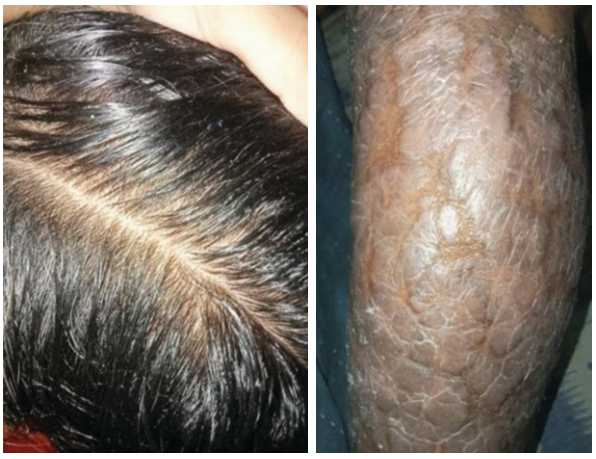

Figure 2: After 2 months' treatment, Plaque psoriasis with Systemic Lupus Erythematosus on scalp and arms

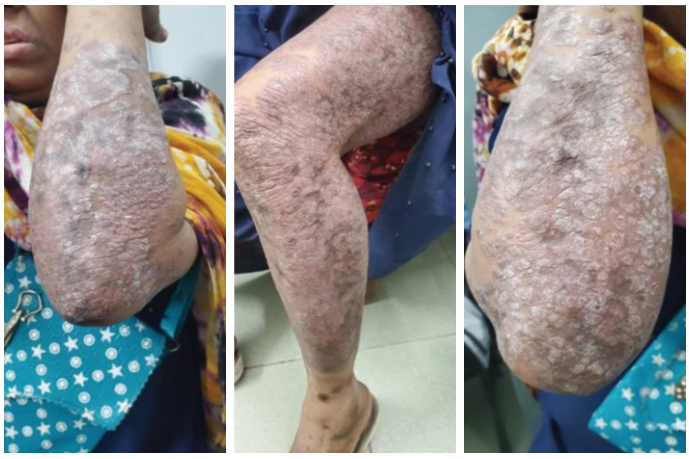

Figure 3: After 4 months of treatment, Severe plaque psoriasis with Systemic Lupus Erythematosus on arms and knees

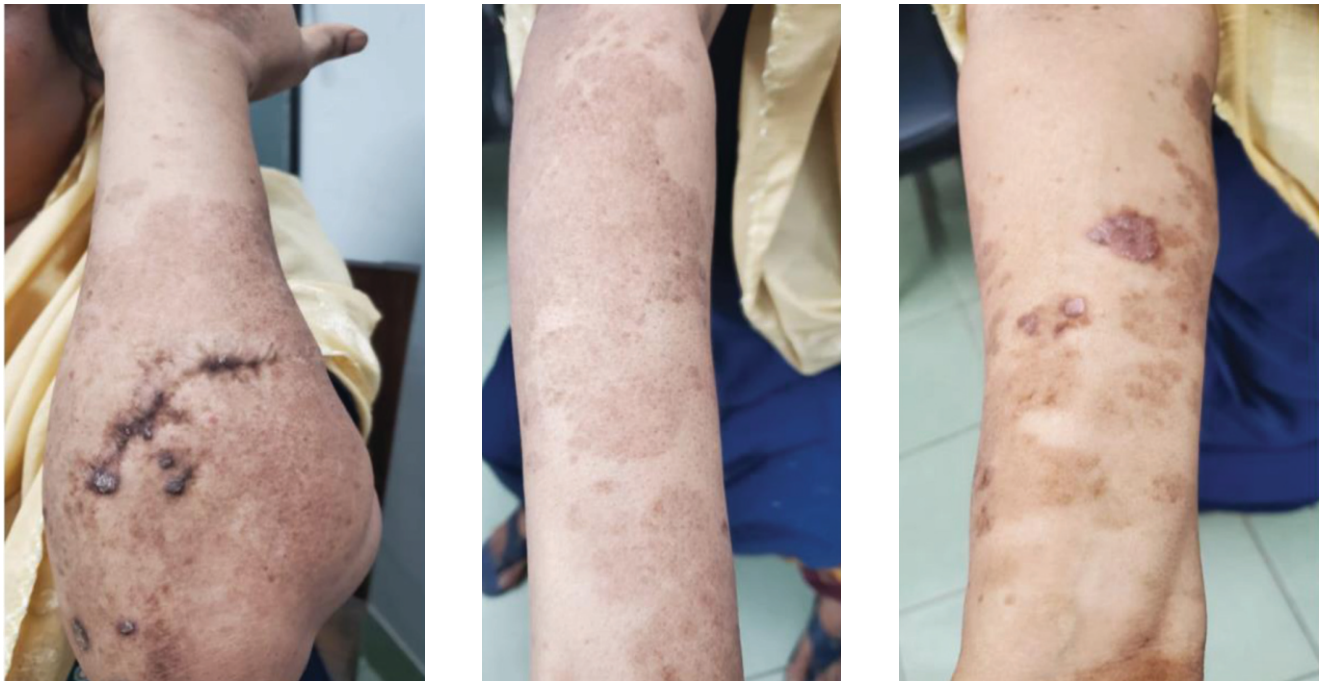

Figure 4: After 6 months of treatment, Severe plaque psoriasis with Systemic Lupus Erythematosus on arms

The patient had not only moderate to severe plaque psoriasis and SLE but also hypertension, rheumatoid arthritis, diabetes mellitus with diabetic nephropathy of the second stage, and lupus nephritis class I. This intricate scenario suggests that there is a need to consider multiple issues and the correlation between different diseases and drug treatment.[6] This made the case even more complicated due to the patient's immunosuppressive medications, which included systemic steroids (Tab. Cortan 20 mg) and methotrexate, among other medications due to other illnesses.[7] Thus, the main challenge in managing such patients is finding the right balance between the need to control the disease and the potential for episodes of immunosuppression or drug interactions.

The physical assessment observed the involvement of multiple joints and tenderness, swan neck deformity, and boutonniere deformity of the thumb pointed towards rheumatoid arthritis. Based on the findings of the integumentary system, the patient developed erythematous, medium- to large-sized plaques over various parts of the body with a silvery-white scaly surface, and a positive Auspitz sign indicates plaque psoriasis. Psoriasis severity was assessed using the PASI, which measures severe erythema, scaling and induration, and BSA involvement >25%. Investigations done included simple count, renal function test, liver function test, ANA/ANF and anti-DNA, and skin biopsy for histology.[8] The skin biopsies also confirmed positive for psoriasis with the clinical characteristics of hyperkeratosis, parakeratosis, acanthosis, and Munro micro abscess. Several management issues are involved in this case, including poly-autoimmunity,

comorbidity, and the drug interactions that multiple medications might cause. The treatment plan also took care of other diseases associated with psoriasis and SLE, some of which are hypertension and diabetes, among others.[9]

This case presents the scenario of multiple autoimmune disorders and raises questions on how to deal with patients who have Ps and SLE. Such pairing is rare due to the genetic susceptibility and etiological processes related to these diseases. This depicts the importance of studying possible interaction effects between diseases, treatment sensitivity and effects, and toxicity profiles, which are central in developing solid treatment modalities for complicated conditions.[10] While Ps has been linked to HLA-Cw6, ERAP1, and IL23R, SLE has been discussed in relation to STAT4, IRF5, and TNFAIP3 polymorphisms. These genetic differences explain why the overlap of Ps-SLE is rare and it is believed to affect approximately 0.69 percent in psoriasis patients and 1.1% of those with SLE.[7] The main mechanism in the pathogenesis of Ps involves the activation of Th1 and Th17 cells and the release of pro-inflammatory cytokines such as TNF, IL-17, and IL-23. These cytokines stimulate excessive growth of keratinocytes and maintain persistent inflammation in the dermis. SLE, on the other hand, is defined by B cell activation, production of autoantibodies against nuclear antigens, and immune complex deposition that leads to inflammation and tissue injury.[11,12] Despite these differences, both conditions have overlapping elements in the context of inflammation, especially Th17 cells and increased concentrations of IL-17 and IL-23 cytokines. There are significant therapeutic complexities in the case of concurrent Ps and SLE since therapies for each may worsen the other.[13] For instance, ultraviolet radiation [UV] commonly used in the treatment of Ps is known to induce or precipitate SLE flare due to DNA damage and apoptotic cell death, which results in leakage of nuclear antigens and the formation of autoantibodies. In contrast, certain drugs used in the treatment of SLE—antimalarial agents including hydroxychloroquine—have been identified to worsen psoriasis. These treatment conflicts suggest that it is wise to approach intervention cautiously.[14,15] Ju HJ et al. conducted a nationwide cross-sectional study using Korean NHI data, finding significant associations between psoriasis, systemic lupus erythematosus (SLE), and autoimmune rheumatic diseases.[16] In this case, the presented work proposes the use Deucravacitinib, an oral selective TYK2 inhibitor, is the key systemic treatment. Deucravacitinib focuses more on TYK2, which could reduce systemic side effects associated with the treatment of both psoriasis and SLE. This selectivity may be particularly helpful since the patient has a number of chronic conditions, including diabetes and hypertension. The presence of associated diseases such as rheumatoid arthritis, diabetes mellitus with diabetic nephropathy, and hypertension alters the clinical scenario and makes management challenging, thus requiring skillful management. These conditions have their particular risks, prognoses, and therapeutic implications that should be addressed within the comprehensive treatment strategy.[17,18]

Table 1: Prevalence rates for each autoimmune rheumatic disease in patients with and without psoriasis

			Univariable Analyses		Multivariable Analyses	
Patient diseases	Prevalence rate*		Crude OR (95% CI)	p value	Adjusted OR (95% CI)[†]	p value
Ankylosing spondylitis						
Controls	100.3	(268/267,230)	Reference		Reference	
Patients with psoriasis	253.0	(676/267,230)	2.526 (2.193-2.910)	<.001	2.418 (2.097-2.789)	<.001
Rheumatoid arthritis						
Controls	101.0	(270/267,230)	Reference		Reference	
Patients with psoriasis	203.6	(544/267,230)	2.017 (1.743-2.334)	<.001	1.947 (1.680-2.256)	<.001
Behçet disease						
Controls	64.4	(172/267,230)	Reference		Reference	
Patients with psoriasis	80.5	(215/267,230)	1.250 (1.023-1.528)	.029	1.212 (1.016-1.474)	.036
Systemic lupus erythematosus						
Controls	48.6	(130/267,230)	Reference		Reference	
Patients with psoriasis	73.7	(197/267,230)	1.516 (1.214-1.891)	<.001	1.448 (1.158-1.810)	<.001
Sjögren syndrome						
Controls	44.9	(120/267,230)	Reference		Reference	
Patients with psoriasis	51.6	(138/267,230)	1.150 (0.900-1.469)	.263	1.115 (0.871-1.428)	.387
Systemic sclerosis						
Controls	10.5	(28/267,230)	Reference		Reference	
Patients with psoriasis	25.8	(69/267,230)	2.465 (1.589-3.824)	<.001	2.410 (1.550-3.749)	<.001
Dermatomyositis/polymyositis						
Controls	9.4	(25/267,230)	Reference		Reference	
Patients with psoriasis	22.1	(59/267,230)	2.360 (1.478-3.768)	<.001	2.303 (1.439-3.686)	<.001

CI, Confidence interval; OR, odds ratio.
*Per 100,000 population.
[†]Adjusted for age, sex, and insurance type.

The use of antihistamines and topical potent steroids can further manage the cutaneous manifestations of psoriasis in a manner which was not effectively covered by the systemic strategy. Together this can act locally that is reducing itching and inflammation, perhaps enhancing the patient's quality of life quicker. The employment of liquid paraffin as an emollient also enhances skin barrier, an element that plays a significant role in handling of psoriasis.[19] Such a case clearly shows that the treatment of a patient should involve several specialists who possess different kinds of knowledge. However, such a treatment plan appears to have enhanced the status of the patient; it should be remembered that further evaluation and possible modification may still be necessary owing to its treatment connotations. As deucravacitinib is a relatively novel therapy, the identification of other related autoimmune disorders and comorbidities may require careful monitoring to determine the efficacy and safety of the treatment.[20] Therefore, this case underscores the difficulties and interventions needed in the treatment of patients with multimorbid autoimmune diseases. It increased awareness towards the requirement of personalized therapeutic management, the risks of drug interactions and side effects, and interprofessional collaboration.

Conclusion:

This case report highlights how Ps and SLE can occur simultaneously and the need to promptly diagnose and treat such conditions. Thus, it stresses proper management of patient care that requires addressing multiple autoimmune diseases and symptoms' interactions. SLE and psoriasis may occur simultaneously, and this calls for the appropriate use of drugs. The compound deucravacitinib has demonstrated effectiveness in both ailments based on the phase trial studies and opens up the possibility of effectively managing multiple autoimmune diseases.

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Dental implant after dredging of ameloblastoma in the mandible: A case report

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Abstract

Ameloblastoma is a benign aggressive tumour of jaw and the most common odontogenic tumour. Instead of complete resection dredging method is being used as a conservative surgical technique. Dredging is the excellent method for the treatment of ameloblastoma and dental implant can be possible on the regenerated bone of the mandible effectively

Keywords: Ameloblastoma, Dredging, Implant.

Introduction:

Ameloblastoma is a locally invasive benign epithelial odontogenic tumour of jaw, mostly occurs in mandible.[1] The relative frequency of the mandible to maxilla is reported as varying from 80-20%. Sign and symptoms of ameloblastoma are slow growing, painless swelling, causing expansion of the cortical bone, perforation of the lingual and/or buccal plates and infiltration of soft tissues. There is often delay in the diagnosis because of its slow growing nature.[2] Radiologically amaeloblastomas typically form rounded, cyst like radiolucent areas with moderately well- defined margins and typically appear multilocular.[3] Ameloblastoma have a high post-surgical recurrence rate. They often recur after simple conservative surgeries, such as enucleation.[4] Resection of the mandible has been the principal treatment of ameloblastoma though it is associated with numbers of complications.[1] The dredging method was thus developed as an alternative, conservative treatment of ameloblastoma. This method leading to lower recurrence rate by removing all tumours and accelerates new bone formation and dental prosthesis can be performed on the reformed bone.[4]

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Case Presentation:

A 19-year-old boy presented with a swelling in the left side of lower jaw which was asymptomatic for six months. However, the time since the lesion started was undetermined. Patient was unable to report the time of evolution of the lesion. There was no history of trauma or toothache or any discharge from the swelling. On extra oral examination there was little facial asymmetry, no palpable lymph nodes were found. There was a solitary well defined swelling over the left lower third of the face measuring about 4cm*3cm extending 2cm from the left angle of the mouth to left lateral border of the mandible. The surface was smooth and the overlying skin was free and normal color. It was non tender and firm in consistency.

On intra oral examination revealed well-defined smooth solitary swelling in the left lower posterior buccal vestibule extending anteroposteriorly from 1st molar to retromolar region and buccolingually 2cm from the buccal surface of the molars to lingual surface. Surface was smooth and colour of mucosa revealed normal overlying the swelling. It was non tender and hard in consistency with buccal and lingual cortical plate expansion. The patient had got developmental anomalies like retained deciduous mandibular central incisors and left maxillary canine and congenital missing of permanent 23,31,41 teeth (Fig 1).

Patient was subjected to radiographic evaluation by OPG and computed tomography. Ameloblastoma was thought to be our provisional diagnosis as it is the most common benign tumour in the molar region of the mandible. Radiology shows a well-defined radiolucent area in the left side of the mandible extending from distal to 2nd premolar to condyle and coronoid process of the same side of the mandible (Fig 2).

Incisional biopsy was performed and histopathological examination confirmed follicular ameloblastoma. Patient was subjected to conservative surgical procedure dredging. Dredging method consists of deflation, enucleation and repeated dredging. Deflation was done under GA to release the intra cystic pressure and facilitate the formation of bone. After 2 months of deflation enucleation was performed and bony was kept open.

Then 2 times dredging were performed here 6 weeks interval. Every time we sent the tissue for histopathological examination and finally we got the tumour free scar tissue. One year after operation we placed 2 implant fixtures on that regenerated bone (Fig-3 and 4). The patient is in regular follow up with us for the last 2 years.

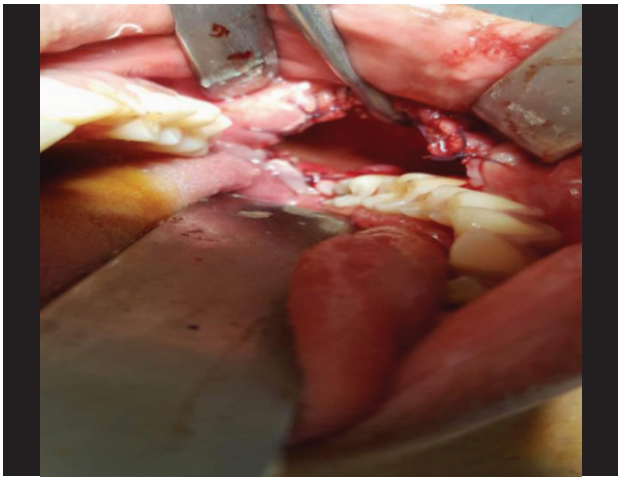

Figure 1: Intraoral finding's image

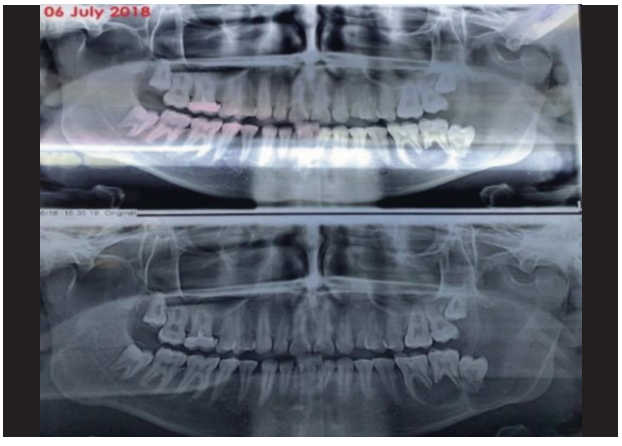

Figure 2: Initial radiograph

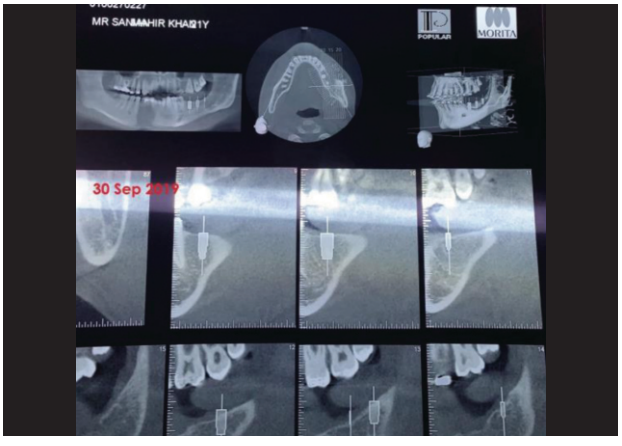

Figure 3: CBCT for evaluation of implant

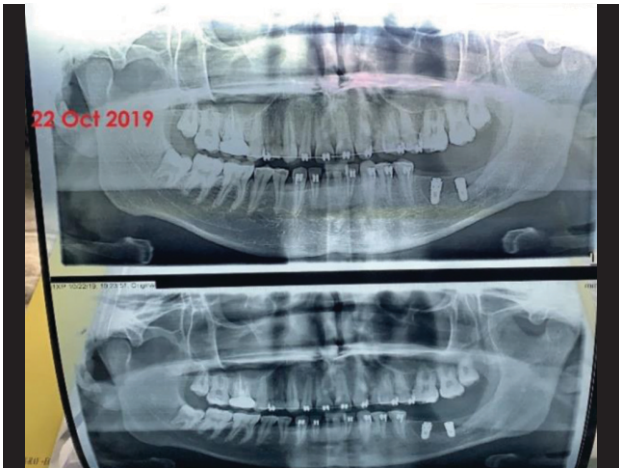

Figure 4: Post-operative radiograph of dental implant

Discussion:

Ameloblastoma originate from epithelial remnants of dental embryogenesis.[5] Although a wide variation in the range of ages can be observed, ameloblastoma primarily affects young adults between the fourth and fifth decades of life. The typical ameloblastoma begins as a slowly destructive asymptomatic and intraosseous expansion, being a lesion that tends to expand and infiltrate rather than perforate the bone. However, diagnosis can also be suggested through a routine radiographic examination.[6] Patient may experience pain or numbness, swelling, malocclusion, tooth mobility or secondary infection. Ameloblastoma can occur at any location in the mandible or maxilla, but the regions of the inferior molars and mandibular ramus are the most prevalent anatomical location.[7]

Larger tumours may rupture the bone cortex and infiltrate adjacent soft tissues on the lingual surface of the mandible. When the tumour occurs in the maxilla, the posterior region is the most affected site. Ameloblastoma occur in distinct clinical and radiographic situations, which need to be considered separately due to therapeutic and prognostic differences.[8]

For reporting ameloblastoma, it seems acceptable to group the treatment regimens into three modalities being conservative that includes enucleation and curettage, marsupialization, dredging and radical surgery. Mandibular reconstruction of large defect may need in case of large ameloblastoma. Dredging method is considered to restore the normal contour and function of jaw with complete removal of tumour tissue. Dental implant can be possible on the regenerated mandible.

Conclusion:

Ameloblastoma is the most commonly occurring tumour in the mandibular body and ramus region. Dredging is the excellent method for the treatment of ameloblastoma and dental implant can be possible on the regenerated bone of the mandible effectively.

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Case Report

Polycystic ovarian syndrome co-existing in a patient of MRKH syndrome: A case report

Habib A[a], Banu NA[b], Akter H[c], Islam S[d]

Abstract

Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome, also referred to as Müllerian aplasia, is a congenital disorder characterized by aplasia of the uterus and upper part of the vagina in females with normal secondary sex characteristics and a normal female karyotype (46,XX).There is a known association between polycystic ovary syndrome and Mullerian developmental defects. A 27 year old unmarried girl presented with primary amenorrhoea due to complete Mullerian agenesis as documented sonographically by absence of the uterus. She presented with features of overt hirsutism, obesity Class 1 and biochemical evidence of hyperandrogenism as manifested by elevated LH to FSH ratio. She was found to be hypertensive and there was evidence of insulin resistance manifested as impaired glucose tolerance along with hypercholesterolemia. On physical examination, her BMI was found to be 32, no kyphoscoliosis, blood pressure was 150 / 95 mm of Hg and Ferriman-Gallwey score (F-G score) of 18. Internal examination revealed presence of a 3 centimetre deep vaginal dimple with complete absence of the cervix. Secondary sexual characteristics were normal in accordance with the age of the patient. However, she was not thoroughly evaluated by an IVU or MRI regarding concomitant urogenital dysplasia. Every case of MRKH syndrome is unique in the sense of management priorities, reproductive and sexual needs, co-morbidities. Addressing each of these issues is quite challenging to the clinician as these have significant long–term impacts on the quality of life in physical, sexual and mental health aspect. The prospect of having genetic and legal motherhood of MRKH patients through uterine transplantation has opened a new arena to be explored.

Keywords: Polycystic ovary symdrom, MRKH syndrome, Mullerian agenesis.

Introduction:

The ovarian development is separate from that of the uterus,cervix, vagina and fallopian tubes. Therefore normally formed and functioning ovaries are present even though there is a concomitant Mullerian defect. The ovaries differentiate from the undifferentiated gonad in thegonadal ridge, medial to the mesonephros induced by the migration of Primordial germ cells from the yolk sac under the influence of WNT4 gene (ovary-determining gene).[1]The oviducts, uterus, cervix, and upper two-thirds of the vagina origin from the paramesonephric (Müllerian) ducts (PMD), whereas the lower part of the vagina origins from the urogenital sinus. Formation of the PMD starts around 5th-6th gestational week as bilateral craniocaudal invaginations of the coelomic epithelium of the urogenital ridges (intermediate mesoderm) growing caudally guided by the mesonephric (Wolffian) ducts to reach the urogenital sinus (endoderm). The caudal part of the two PMDs fuses to form the uterus, cervix and upper vagina, whereas the upper parts of the PMDs form the twooviducts.[1] In MRKH syndrome which has a prevalence of 1 in 5000 live female births, there is aplasia or hypoplasia of the Mullerian ducts resulting in either complete absence or variable uterine remnants. Increasing reports of familial occurrence of MRKH syndrome and its associated anomalies support a monogenic genetic etiology.[2] Most pedigrees suggest autosomal dominant inheritance with incomplete penetrance. In contrast, most cases occurring sporadically, lacking recurrence in outcomes of surrogate pregnancies and several reports of discordant twin pairs support either polygenic/ multifactorial or non-genetic etiologies (e.g. teratogenic exposures in utero). [3,4]

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy among reproductive age women. Although the diagnostic Rotterdam criteria include 2 of 3 features hyperandrogenism (HA), ovulatory dysfunction (OD), polycystic ovarian morphology (PCOM), PCOS women often display notable metabolic co-morbidities. These long-term metabolic consequences of PCOS cannot be denied in women with or without uterus as in Mullerianagensis. In the etiology of PCOS, hyper-androgenism at various developmental periods has been

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proposed as the major driver of associated reproductive and metabolic perturbations with long-term cardiovascular implications.[5]Therapeutic strategies need to be instituted to prevent and or manage the metabolic complications; starting from lifestyle modification, maintenance of an optimal BMI and appropriate medications for hypertension, glucose intolerance, hirsutism etc. However in patients with complete Mullerian agenesis with co-existing PCOS, the effect of unopposed oestrogen on the uterine endometrium is not applicable.

Case Presentation:

A 27 year- old unmarried Muslim working lady (self-employed) presentedas a known case of complete Mullerian agenesis detected at 16 years of age due to non–resumption of menstruation. She was diagnosedby the absence of the uterus and two ovaries on ultrasonography of lower abdomen and pelvis and presence of bilateral rudimentary uterine budson Pelvic MRI. She complained of excessive facial and body hair over the last 3 years and significant weight gain which was 17 kilograms over 9 months. On examination, blood pressure was 150 / 95 mm of Hg, weight: 79 kg, Height; 157 cm, BMI: 32.04. Ferriman - Gallwey score of 18 , Hair distribution was moderate hair under chin (FG score 3), upper abdomen(FG score 3), lower abdomen(FG score 3), upper back (FG score 4), thigh (FG score 3)and arms (FG score 2). Vaginal examination with consent revealed normal external genitalia and urethral orifice, presence of a 3 centimetre deep vaginal depression with absence of the cervix. Breast development was normal for age. Investigations revealed TSH and fT_4within normal limit, Serum FSH: 3.9 IU/l and serum LH 18.4 IU/l (evidence of HA), Fasting Blood sugar was 5.7 mmol/l , 2 hours Post prandial 8.7 mmol/l, HbA1c 6.9% reflecting impaired glucose tolerance , Fasting Lipid profile: Total cholesterol 315 mg/dl, HDL 28 mg/dl , LDL 132 mg/dl (suggesting atherogenic dyslipidemia). Ultrasonography of lower abdomen revealed bilaterally enlarged ovaries with 12-14 peripherally arranged follicles measuring 9-11 mm in diameter and absent uterus. Karyotype revealed 46XX. She was counselled regarding the long-term impact of chronic anovulation related to PCOS. Emphasis was placed on diet and life style modification. In collaboration with cardiologist, she was advised anti-hypertensive medication and lipid-lowering agents along with long-term cardiovascular surveillance. However, at the moment, she expressed no inclination towards sexual/reproductive needs. Moreover, she was not thoroughly evaluated regarding urogenital, cardiacand skeletal system.

Discussion:

The association of polycystic ovarian syndrome with Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome has been studied and published in different articles.The polycystic ovary syndrome, whose etiopathogenesis is not clearly understood, has a wide spectrum of clinical presentations, and may co-exist with other pathologic conditions. In a study conducted by Mustafa Ugur, the prevalence of ultrasound-defined polycystic ovaries (PCO) in patients with müllerian anomalies (n =167, study group), and those without müllerian anomalies (n =3165, control group) from 1990 to 1994, in a population markedly composed of infertility patients was evaluated. PCOS were found in 50 (29.9%) patients in the study group, compared to 637 (20.1%) patients in controls ($P <$ 0.01).[5] Müllerian anomalies were further grouped according to the American Fertility Society (AFS) classification and it was found that patients with the septate uteri and bicornuate uteri malformations had a higher prevalence of PCOS (polycystic ovaries) than the controls ($P < 0.001$, $P < 0.05$, respectively). It was concluded in another study that PCOS was more prevalent in certain müllerian anomalies and an embryogenetic defect may also be involved in the etiopathogenesis of PCOS.[6]

MRKH is one of the most common causes of primary amenorrhea and affects at least 1 out of 4500 women. MRKH syndrome is caused by either complete agenesis or aplasia of the paramesonephric ducts to form the uterus and upper vagina. Its penetrance varies, as does the involvement of other organ systems. MRKH syndrome is classified as type I (isolated uterovaginal aplasia) (56-72%) or type II (associated with extra genital manifestations, 28-44%). Extra genital anomalies typically include renal, skeletal, ear, or cardiac malformations. MRKH syndrome Type II is associated in 40% of cases with kidney abnormalities (15% of these girls/women will be born with only one kidney), 10% will have hearing problems, and 10-12% will have vertebral anomalies, rib anomalies, hypoplastic or absent radii, abnormalities of the carpal bones and hypoplastic phalanges, bilateral femoral hypoplasia.[7,8,9] Duncan et al in 1979, in a review of 30 cases proposed the designation of the entity as "MURCS Association (Müllerian duct aplasia/hypoplasia, renal agenesis/ectopy, and cervicothoracic somite dysplasia (MURCS) association)". The four most common malformations specifically described in the association are: 1. uterine hypoplasia or aplasia, 2. renal agenesis or ectopia, 3. vertebral anomalies **(**e.g. Klippel-Feil syndrome/cervical vertebral fusion syndrome**)**, and 4. adult stature less than 152 cms. [10]

MRKH patients typically (95%) display bilateral uterine rudiments combined with a fibrous band and normally located ovaries on MRI. The uterine rudiments are generally small with only one-layer differentiation, a subset of which might be large and exhibited other atypical presentations, including two- or three-layer differentiation or even hematometra. Functional rudimentary uteri may present with endometriosis and related symptoms explained by retrograde menstruation. In MRKH syndrome, both ovaries are typically present and well-functioning. However, their anatomical position is usually more cranial than the normal position and they are often found lateral, rather than medial, to the external iliac arteries, probably due to the lack of Fallopian tube development. Ovarian anomalies are rare and only found in ~5-10% .[10,11]Inguinal ovary has also been reported as a rare diagnostic sign of Mayer-Rokitansky-Küster-Hauser

syndrome in a girl in attempt to operate on an inguinal hernia in a case of Type I MRKH syndrome.[12] However, in our case both ovaries were intra-abdominally located. Different ovarian anomalies previously reported include unilateral agenesis, ectopic ovaries, polycystic ovaries, streak ovaries, and rarely tumors.[14,15]

Detection of normal female karyotype (46,XX) on chromosomal analysis by G/Q-banding in this patient excludes the possibility of Complete androgen insensitivity syndrome (CAIS, also referred to as Morris syndrome) which is an X-linked disorder affecting genetically males (46,XY) caused by hemizygous mutations in the androgen receptor gene, AR (OMIM #300068) and 17-hydroxylase/17,20-lyase deficiency in 46,XY females caused by biallelic CYP17A1 mutations. These patients also have normal female appearance, blind-ending vagina and absent uterus and have breast development but sparse pubic hair at puberty.[16]

Relevant laboratory tests include FSH, LH, androgens and estradiol, which are generally considered to be normal in MRKH syndrome.[5,17,18] However, biochemical (non-clinical) hyperandrogenemia was recently reported in ~50% of patients. 85 In this case, the patient experienced excessive facial and body hair (F-G score of 18)over the last 3 years and 17 kilograms weight gain over 9 months with blood pressure 150 / 95 mm of Hg, BMI(Body Mass index): 32.04 .

After confirmation of the diagnosis of MRKH syndrome, management usually relates to the psychological and psychosexual issues, reproductive needs such as vaginoplasty and reconstructive surgery for hypoplastic /rudimentary uterus. Addressing concomitant issues are important in this case such as management of common co-morbidities such as insulin resistance, obesity and hypertension. MRKH syndrome may have profound psychological and/or psychosexual impact once the diagnosis is disclosed.[19] Receiving the diagnosis, many patients experience facing overwhelming issues regarding identity, sexuality and infertility, and the importance of good caring and counselling should not be underestimated. The diagnosis is often made during adolescence; a period of physical/emotional development and vulnerability, which further imposes the provider's caring and awareness towards the patients' emotions, reactions and coping strategies. Furthermore, it is important to be aware of potential cultural aspects and their influence on reactions to the diagnosis in patients and their families and peers.In our patient, mental adjustment was well and she was found to coping well with the condition.

Historically, in MRKH syndrome creation of a functional neovagina has been a hallmark in the treatment for those who are sexually active. Dilation therapy either by Frank's method or d'Alberton's method as first choice is supported by Callens et al.[20], which further suggest laparoscopic Vecchietti vaginoplasty as preferred second-line therapy. Most importantly, thorough counseling regarding expected outcome and possible complications should always precede any attempt for vaginal construction, and it is fundamental to ensure the full maturity and motivation of the patient undergoing such treatment. Importantly, the three latter methods, which are based on dilation of vaginal dimple, will provide the vagina with a normal mucosal lining. This may be advantageous in a uterus transplantation situation (UTx) since this will provide the vagina with a normal vaginal microbiota, which may be of importance for success at embryo transfer as well as for correctly grade rejection by cervical biopsy. Moreover, it is important to recognize the option of no treatment, which for some patients might be the right choice;[2,21] as currently applies to our case.

The current reproductive options for MRKH patients are adoption (non-genetic), gestational or host surrogacy (genetic) and uterine transplantation (UTx).Upto 2020, approximately 75 UTx procedures have been performed and all but two of these have been performed in MRKH patients (Brännström, personal communication). Around 25 babies have been born worldwide, with some of the MRKH patients having delivered healthy babies twice.[22,23]

Conclusion:

Every case of MRKH syndrome is unique in the sense of management priorities, reproductive and sexual needs, co-morbidities. Addressing each of these issues is quite challenging to the clinician as these have significant long –term impacts on the quality of life (QoL) in all aspects (physical, sexual, mental health). Approximately 30% of MRKH syndrome patients have concomitant urological, renal, skeletal or cardiac structural problems. Moreover, ovarian pathology such as Polycystic changes with or without clinical hyperandrogenism, endometriosis issues in case of functional uterine remnants in case of hypoplastic uteri are also to be kept in mind. Moreover, the prospect of having genetic and legal motherhood of MRKH patients through uterine transplantation has opened a new arena to be explored.

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College News

College Events:

- 52nd Victory Day of Bangladesh was celebrated in Bangladesh Medical College and Hospital premises on 16th December 2023. Teachers, doctors, nurses, students of BMC & BMCH and officials & staffs of BMSRI participated in that event.

- Commencement ceremony of newly admitted students of BM-37 was held on 24th July, 2023 at the BMC auditorium. Total 120 students were admitted in the session of 2022-2023. Prof. Paritosh Kumar Ghosh, Principal, BMC chaired the ceremonial event.

Seminar/Workshops:

- Central CME on "Diagnostic Dilemma of Surgical Emergency" was held on 06.07.2023. Speaker was Dr. Mahmudul Hasan Shakil, Assistant Registrar, dept. of Surgery, BMCH

- Central CME on "Obstetric ICU: Necessity and Challenges" was held on 06.08.2023. Speaker was Dr. Asma Habib, Associate Professor, dept. of Gynae & Obstetrics, BMC

- Seminar on "Thyroid dysfunction: Treat or Not to Treat" was held on 29.08.2023. Speaker was Dr. Yasmin Aktar, Associate Professor, dept. of Endocrinology, BMC

- Seminar on "Folic Acid Fortification: Nourishing Today, Building Tomorrow" was held on 01.10.2023. Speaker was Prof. Dr. Rezina Hamid, Professor and Head, dept. of Neurosurgery, BMC

- Seminar on Breast Cancer and Celebration of Breast Cancer Awareness month October 2023 was held on 21.10.2023. Speakers were Dr. Rabab Sultan, Assistant Professor, dept. of Oncology, BMC and Dr. Shahnaz Begum, Assistant Registrar of Oncology, BMCH.

- Seminar on "Counteracting Antimicrobial Resistance" was held on 25.11.2023. Speaker was Dr. Sharmila Huda, Associate Professor, dept. of Pharmacology and Therapeutics, BMC

Participation in the International Conference/Seminar/Workshop/ Congress/Meeting:

- Dr. Muhammed Akhtaruzzaman, Associate Professor, Dept. of Cardiology, BMC attended the Asian Pacific Society of Cardiology (APSC) Congress 2023 held in Singapore from 13th to 15th July 2023.

- Prof. Dr. Zafor Md. Masud, Professor and Head of the Dept. of Oncology, BMC attended the Annual Scientific Meeting on Frontiers in Cancer Care held on 2nd August to 4th August 2023 in Australia.

- Dr. Muhtamim Chowdhury, Assistant Professor, Dept. of Neurosurgery, BMC attended the 2023 CNS Annual Meeting of the Congress of Neurological Surgeons held in Washington D.C, USA from 9th to 13th Sept., 2023.

- Dr. A. T. M Zulfiqur Rahman, Assistant Professor, Dept. of Orthopaedics, BMC attended the 20th Indian Arthroscopy Society (IASCON 2023) held in Lucknow, India from 1st to 3rd September 2023.

- Dr. Mohammad Nazrul Islam Bhuiyan, Associate Professor and Head of the Dept. of Urology, BMC attended the 20th Urological Association of Asia Congress 2023 & 12th Emirates International Urological Conference 2023 held in Dubai from 28th September to 1st October 2023.

- Dr. Md. Saydur Rahman, Professor (Current Charge), Dept. of Orthopaedics, attended the 43rd Orthopaedic World Congress held from 21-23 November 2023 at Cairo, Egypt.

- Prof. Dr. Jamal Uddin, Professor and Head of the Dept. of Dermatology, BMC attend the 32nd EADV Congress 2023 in Berlin, Germany held on 11th to 14th October 2023.

- Prof. Dr. Mushtaque Ahmad Rana, Professor, Dept. of Gastroenterology, BMC attended the EASL Congress held at Denmark from 14-17 October 2023.

- Prof. Dr. Zafor Md. Masud, Professor and Head of the Dept. of Oncology, BMC attended the ESMO Asia Congress 2023 held on 1st to 3rd December 2023 in Singapore.

- Dr. Yasmin Aktar, Associate Professor, Dept. of Endocrinology, BMC attended the IV Advanced Pediatric Endocrinology Symposium and Onsite Course at Kanpur, India on 17th to 20th December 2023.

- Dr. Zannat Nur, Assistant Professor, Dept. of Transfusion Medicine, BMC attended the XVIII Annual Congress of Asian Association of Transfusion Medicine & 4th Joint Meeting of AABB-AATM 2023 held in New Delhi from 1st to 2nd December, 2023.

- Dr. Sonia Mahjabin, Assistant Professor, Dept. of Nephrology, BMC attended the Indian Society of Nephrology Conference 2023 held at Kolkata, India from 14th to 17th November 2023.

New Promotions in BMC:

Dr. Sazia Afrin, Assistant Professor of Dermatology, BMC

New Appointments in BMC:

Dr. Mst. Umma Khair Fatima, Assistant Professor of Forensic Medicine, BMC

Dr. Sumona Islam, Assistant Professor of Gastroenterology, BMC

Dr. Sayeda Shabnam Malik, Assistant Professor of Neuromedicine, BMC

Dr. Nurunnahar Faizun Nesa, Lecturer of Community Medicine, BMC

Dr. Nusrat Tanzil, Lecturer of Forensic Medicine, BMC

Dr. Tasnova Ferdous Prema, Lecturer of Pathology, BMC

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