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Editorial

Aedes and Dengue- The Vicious Couple

Alam MT

The mosquito *Aedes aegypti* is the primary species responsible for transmitting viruses of Zika, dengue, yellow fever and chikungunya between people. In some communities, other mosquitoes may also contribute to transmission, but their contribution is minor.

Aedes aegypti originates from Africa but is now distributed globally in tropical and subtropical regions. The first registered epidemic of dengue fever happened in 1779-1780 across Asia, Africa, and North America. A global pandemic of dengue fever began in Southeast Asia after World War II.

Rapid population growth and increased urbanization have led to substandard housing, inadequate water supply and waste management systems which consequently flared up abundance of mosquito breeding sites. Importantly, mosquitoes do not naturally carry viruses. Mosquitoes must acquire them from an infected person before they can transmit them to another person. Only female mosquitoes bite, requiring the blood for egg production, and therefore only female mosquitoes transmit viruses. They feed during daylight hours. Biting activity is higher in the two hours after sunrise or before sunset.

Among the four viruses that aedes aegypti transmits, dengue has been ravaging our country. Incidence of dengue has grown dramatically around the world in recent decades. An estimated 3.9 billion people in 128 countries are at the risk of dengue infection according to the World Health Organization. Globally, the number of cases reported increased from 2.2 million in 2010 to 3.2 millions in 2015.

Since the first detected cases of dengue in Bangladesh in 2000, around 49,000 people suffered from the fever, with at least 316 deaths reported. Most of the patients were from Dhaka or its neighboring districts. The number of cases crossed the 6000 mark only three times in the last 18 years- 6,232 in 2002 and 6,060 in 2016. This year, the number already stands at 6,479. Aedes aegypti spreads all four strains of dengue virus. Once detected in a country, the virus never really goes away according to the experts.

Symptoms of dengue, which usually begin four to six days after infection and last for up to 10 days, may include: sudden, high fever, severe headaches, pain behind the eyes, severe joint and muscle pain, fatigue, nausea, vomiting, skin rash, which appears two to five days after the onset of fever, mild bleeding (such a nose bleed, bleeding gums, or easy bruising), swollen glands etc. Sometimes, symptoms are mild and can be mistaken for those of the flu or another viral infection. Younger children and people who have never had the infection before tend to have milder cases than older children and adults. But this year in 2018, children are suffering with more severity and mostly affected by type 3 serotype of dengue virus. These are mostly dengue hemorrhagic fever, a rare complication characterized by high fever, damage to lymph and blood vessels, bleeding from the nose and gums, enlargement of the liver, and failure of the circulatory system. The symptoms may progress to massive bleeding, shock, and death. This is called dengue shock syndrome (DSS). People with weakened immune systems as well as those with a second or subsequent dengue infection are believed to be at greater risk for developing dengue hemorrhagic fever, which is the case for this year as a new strain has emerged this year.

Doctors can diagnose dengue infection with a blood test to check for the virus or antibodies to it. There is no specific medicine to treat dengue infection. Patients should be treated with plenty of fluid, paracetamol, sponging and avoid drugs containing aspirin, non-steroidal anti-inflammatory like diclofenac, ibuprofen. which could worsen bleeding and see a doctor.

Efforts to develop a vaccine against dengue have been ongoing for decades. The first such vaccine Dengvaxia is a live, attenuated tetravalent product developed by Sanofi Pasteur. Following two phase 3 clinical trials published in 2014, Dengvaxia was licenced in December, 2015, and approved in 19 countries. However, after reassessment of data from the clinical trials, Sanofi warned on Nov 29, 2017, that the vaccine can increase the risk of severe dengue in particular circumstances. How any vaccine against dengue is used is complicated by the fact that virus occurs in four serotypes, and immunity against any one serotype does not generate lasting immunity against the other three, hence the need for a tetravalent vaccine. Furthermore, being infected with one and developing immunity to that one viral serotype seems to be the trigger that can lead to a patient having more severe disease manifestations when subsequently infected with a different serotype, a phenomenon known as antibody- dependent enhancement. At this moment a few other trials are going on for newer vaccines.

Newer concept of Aedes aegypti management:

Wolbachia are natural bacteria present in up to 60% of insect species, including some mosquitoes. However, *Wolbachia* is not usually found in the *Aedes aegypti* mosquito. For many years, scientists have been studying *Wolbachia*, looking for ways to use it to potentially control the mosquitoes that transmit human viruses. The World Mosquito Program's (WMP) research has shown that when introduced into the *Aedes aegypti* mosquito, *Wolbachia* can help to reduce the transmission of these viruses to people. This important discovery has the potential to transform the fight against life-threatening mosquito-borne diseases. *Wolbachia* is safe for humans, animals and the environment.

The WMP's *Wolbachia* method helps to protect communities from mosquito-borne diseases and does so without posing a risk to natural ecosystems or human health. This method has some unique features. Unlike most other initiatives, it is natural and self-sustaining. It does not suppress mosquito populations or involve genetic modification (GM) as the genetic material of the mosquito has not been altered.

The best way to prevent the disease is to prevent bites by infected mosquitoes by using mosquito repellents, wearing long sleeved shirts and long pants, using mosquito nets and making efforts to keep the mosquito population down. This is where we should be working with the various government agencies as it is everybody's job. People should keep their homes and surroundings free from unnecessary containers to prevent mosquito breeding. Also, their eggs remain viable in dry place for around two years so solid waste should also be taken care of.

It can be concluded that Aedes aegypti should be studied as a long-term national, regional, and world problem rather than as a temporary local threat to the communities suffering at any given moment from yellow fever, dengue or other aegypti-borne disease. No one can foresee the extent of the future threat of Aedes aegypti to mankind as a vector of known virus diseases, and none can foretell what other virus diseases may yet affect regions where A. aegypti is permitted to remain.

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Original Article

Variations of Frontal Air Sinus Dimension among Adult Bangladeshi People

Maqsood F[a], Ara S[b], Rashid MA[c], Shahana N[d], Hossain F[e], Morium U[f], Asha MT[g]

Abstract

Background: Observation and measurement of frontal air sinus dimension is important to the forensic experts, anatomist, anthropologist, radiologist, otolaryngologist and neurosurgeons in their respective field. The values of the frontal air sinus study can be used for personal identification, sex differentiation and age determination in forensic science due to its unique morphological value in every individual.

Objective: This study was done to analyze and compare the variation of the frontal air sinus dimension among the adult Bangladeshi people.

Methods: The cross sectional analytical study was conducted in the Department of Anatomy, Dhaka Medical College, Dhaka, from January 2016 to December 2016. The study was performed on 100 radiographs of caldwell view of skull of adult Bangladeshis. Out of 100 radiographs, 50 were of male and 50 were of female. The Caldwell radiograph of the frontal air sinus of selected individual were collected and viewed on the x-ray view box, traced on transparent graph paper sheet placed on each radiograph film and different dimension of the frontal air sinus was measured. Unpaired Student's t-test, paired Student's t-test were done for statistical analysis of the results.

Results: The mean height the left frontal air sinus was significantly higher than right frontal air sinus both in male (p= 0.027) and female (p= 0.040). Both right (p= 0.001) and left (p= 0.005) frontal air sinus height was significantly higher in male than female. The mean width of the left frontal air sinus was significantly higher than right frontal air sinus width both in male (p=0.048) and female (p= 0.024). Both right (p= 0.001) and left (p= 0.042) frontal air sinus width was significantly higher in male than female.

Conclusion: The result of the present study demonstrates that the height and width of left frontal air sinus was significantly higher than right frontal air sinus both in male and female. The frontal air sinus height and width was higher in male than female which were highly significant.

Keywords: Frontal air sinus height, Frontal air sinus width.

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Introduction:

Every part of human body is unique in itself as every part of the body is different in its own way from a similar part in another body. The frontal sinuses are bilateral anatomical structures anterior to the ethmoid notch and are unique to every individual, even in monozygotic twin similar to the role of fingerprint and DNA.[1] Right and left frontal air sinus is separated by a septum which may be deviated from the median plane. The frontal air sinus is absent at birth but begin to develop embryologically during 2nd year of life. The frontal sinus grows larger by the size in the late adolescence reaching its maximum size at the age of twenty and remaining stable through the rest of the life.[2] The initiation and process of pneumatization differ from person to person. Pneumatization of the skull is strictly connected with the morphology and degree of development of paranasal sinuses.[3] Occasionally one or both sinuses may be absent associated with the presence of metopic suture. By the sixth year, the frontal air sinus can be first demonstrated radiologically.[3] The frontal sinus area, size, shape differs from person to person and race to race.[4] In forensic medicine the frontal sinus morphology helps as primary tool for personal identification and age determination by comparing ante mortem and postmortem radiograph as in

every individual even in monozygotic twin the frontal sinuses provide a unique morphological value very much similar to the role of fingerprint and DNA test.[5] As morphologically the cranium differs between male and female, the frontal sinus in male is larger than female.[6] The morphology of frontal sinus also shows that the frontal sinus is smaller in female than male and this dimorphic characteristic is used for sex determination.[2] Frontal sinus anatomy helps otolaryngologist during surgical procedures involving frontal sinus and skull base.[7] The bilateral or unilateral agenesis of the frontal sinus differs in most ethnic population and the frontal sinus size is highest in Eskimo population for adaptation in the cold climate condition. Some factor can also modify the normal anatomy of frontal air sinus such as craniofacial configuration, hormonal levels, even thickness of the frontal bone[6], genetic and environmental factors, neoplasia, fractures, severe or past infections, trauma or fracture, mucocele.[8] Infection of frontal sinus causing frontal sinusitis is thought as more symptomatic sinus than other paranasal sinus infection due to the difficulties[9] and frontal sinusitis can give rise to abcess, cellulitis and meningitis. Apart from sinusitis other diseases may also arises in the frontal sinus like mucocele, osteoma, frontonasal duct stenosis or atresia. Frontal sinus fracture also may occur from the trauma to the part of the frontal bone that overlies the sinus.[10] Moreover, the plain radiographs analyzed by the clinicians are important to know the anatomy, size and variants of the frontal sinus for specific populations. It is important for surgeons to be aware of the variations that may predispose patients to increase the risk of intra operative complications and help to avoid possible complications.[9] The evaluation of measures of frontal sinus frequently involved in cranial base surgeries and supraorbital craniotomies in order to surgical approaches that cross this anatomical route.[11] Aim of this study is to analyze and compare the variation of the frontal air sinus dimension among the adult Bangladeshi people.

Materials & Methods:

The cross sectional analytical study was carried out in the Department of Anatomy, Dhaka Medical College, Dhaka, from January 2016 to December 2016. The study was performed on 100 radiographs of Caldwell view of skull of adult Bangladeshis. Out of 100 radiographs, 50 were of male and 50 were of female age ranging from 20 to 50 years. Individual with history or X-ray showing any feature of sinusitis, repeated common cold, chronic headache, trauma or surgery of the skull or face including eye, ear and nose, any clinical characteristics of endocrine disturbances or systemic disorder or any sort of cranial asymmetry or facial asymmetry were excluded from the study. X-ray machine was used for doing skull radiograph Caldwell view. The person was in erect posture in posterior-anterior position in front of a part of the x-ray machine facing stand greed. Cassette of stand greed machine had imaging plate which was kept towards head position. Vertical axis was in the parallel to the film in Caldwell projection and no space between frontal bone and cassette. Center of the forehead and nose was adjusted in a position so that the mid sagittal plane is perpendicular to the plane of film and the orbitomeatal line forms an angle 15 degree from the plane of the film. Then head was immobilized in that position and shoulder was adjusted in the same horizontal plane. The person was advised to suspend the respiration for the exposure. Using a well calumniated beam the horizontal central ray is centered in the midline to the occipital region at the level of the lower orbital margin. Radiation was projected on imaging plate and it was transformed in the monitor for editing, zooming of the radiograph. It was zoomed at 100%. AGFA printer was used for printing the x-ray film.

The radiographs collected from every selected individual and viewed on the x-ray view box and transparent graph paper sheet was placed on the view box containing radiograph and frontal air sinus was traced on transparent graph paper sheet placed on each radiograph film and then different dimension of the frontal air sinus was measured. All the measurment of frontal sinus was taken by drawing a tangential line above the superior border of the two orbits because the inferior limit of the frontal air sinus is not so clear.[4]

The measurement was done according to sequence below by Camargo, et al.[12]:

- At first the radiography was placed on a view box and the transparent graphpaper sheet was placed on the radiograph.
- The baseline (A) was drawn directly on the transparent graphpaper at the level of superior border or upper limit of two orbit (inferior border of frontal sinus) as illustrated in figure.
- The separation between the right and left frontal sinus was based on the frontal intersinus septum in order to permit quantifying one width on each side.
- The height of each side (B and C) was determined by marking and drawing a straight line between baseline and upper limit of the frontal sinus.
- The largest width (D and E) of the frontal sinus was determined by marking and drawing a straight lines from the maximum distance between the medial and lateral lines of the right and left side of the frontal sinus.
- Linear measurements of height and width of frontal air sinus was obtained from the each radiograph by counting the boxes of graphpaper, height and width were expressed in centimeters(cm).
- Square box was counted within the sinus and area covering more than 50% of the square was considered.
- All these measurements were obtained by the portion projecting above the baseline .The separation of the right and left side of the frontal sinus was based on the intersinus septum which denotes the margin between the two main sinus cavities.

Paired Student's t-test were done to compare the differences between height of the right and left frontal air sinus and width of the right and left frontal air sinus. Unpaired Student's t-test were done to analyze the differences between right and left frontal air sinus dimension between male and female. Statistical significance was accepted at ($p<0.05$).

Procedure measurement from radiograph of frontal air sinus height and width is shown in fig: 1, 2 (I), 2 (ii)

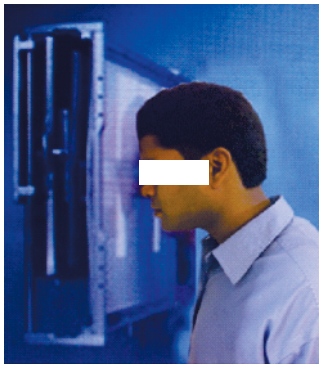

Figure 1: Procedure of taking Caldwell view radiograph of skull

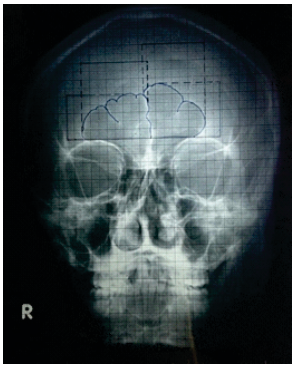

Figure 2 (i): Procedure of tracing Caldwell view radiograph

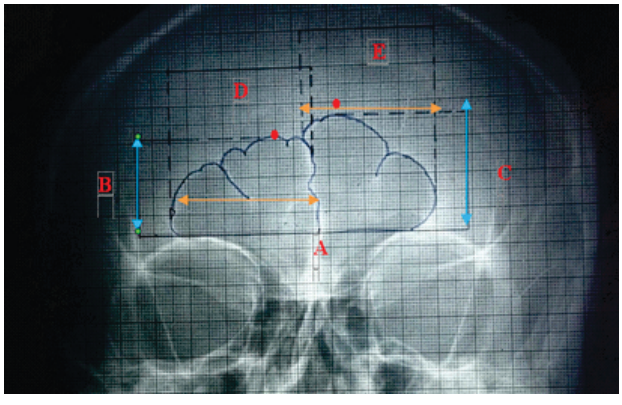

Figure 2 (ii): Procedure of estimating of height, width of right and left frontal sinus.

- Red dot indicate highest point of right and left frontal air sinus.
- ↔ Arrow indicate height of right (line B) and left (line C)frontal air sinus
- Green dot indicate widest point of right and left frontal air sinus.

Arrow indicate width of the right (line D) and left (line E) frontal air sinus.

Results:

Table 1: Comparison of height of right and left frontal air sinus in male and female

Group	Height in cm		
	Right (Mean±SD)	Left (Mean±SD)	
Male (n=50)	1.66 ± 0.69	1.93 ± 0.94	p value
Female (n=50)	1.15 ± 0.73	1.38 ± 0.95	0.027*
P value	0.001*	0.005*	0.040*

Table 1 showing comparison between male and female was done by Unpaired Student's't' test. Comparison between values of right and left frontal sinus of same group was done by Paired Student's't' test. ns= not significant, *= significant, SD= Standard Deviation.

Table 2: Comparison of width of right and left frontal air sinus in male and female

Group	Width in cm		
	Right (Mean±SD)	Left (Mean±SD)	
Male (n=50)	3.22 ± 0.93	3.55 ± 1.07	p value
Female (n=50)	2.60 ± 0.97	3.04 ± 1.37	0.048*
P value	0.001*	0.042*	0.024*

Comparison between male and female was done by Unpaired Student's't' test. Comparison between values of right and left frontal sinus of same group was done by Paired Student's't' test. ns= not significant, *= significant, SD= Standard Deviation.

Discussion:

In present study the left frontal air sinus height was significantly higher than right frontal air sinus both in male (p= 0.027) and female (p= 0.040). Both right (p= 0.001) and left (p= 0.005) frontal air sinus height was significantly higher in male than female (Table 1 & 2). Similar kind of study was conducted by Verma, Mahima and Patil[13], Belaldavar, et al[14], Verma, et al[15], where they observed that the height of the left frontal sinus significantly higher than the right frontal air sinus height in both male and female. The width of the left frontal air sinus was significantly higher than right frontal air sinus width both in male (p= 0.048) and female (p= 0.024). Both right (p= 0.001) and left (p= 0.042) frontal air sinus width was significantly higher in male than female. Our result correlates with the

study of Camargo, et al[12], Verma, Mahima and Patil[13], Belaldavar, et al[14], where the researcher also found the similar result. Camargo, et al[12] and Belaldavar, et al[14], found that width of the left frontal air sinus is significantly higher than the width of the right frontal air sinus both in male and female.

Conclusion:

The result of the present study demonstrates that the height and width of left frontal air sinus was higher than right frontal air sinus both in male and female. The height and width of the frontal air sinus was higher in male than female. This study not only helps as a guideline for the anatomist, radiologist and anthropologist but also clinicians for diagnosis of diseases.

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Original Article

Evaluation of Feto-maternal Outcome in Preterm Pre-eclamptic Mothers in a Selected Tertiary Care Hospital in Dhaka

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Abstract

Background: Perinatal and maternal outcome in eclampsia cases is still poor due to inadequate antenatal care, financial restraints, non-availability of transportation facilities and social taboos causing delay in management resulting in poor maternal and neonatal outcome.

Objective: To evaluate the feto-maternal outcome in preterm preeclampsia in a selected tertiary care hospital in Dhaka city.

Methods: The prospective cross-sectional study was carried out in the department of Obstetrics & Gynaecology, Dhaka Medical College Hospital, Dhaka from 1/07/2010 to 31/12/2010. Diagnosed cases of preeclampsia before completion of 37 weeks of pregnancy, irrespective of their parity who were admitted in labor and antenatal ward were included in this study. Total 64 cases were selected by purposive sampling. Data were collected in a preset questionnaire through interviewing and physical examinations. After delivery, birth weight of newborn was measured by NNC weighing scale within 24 hours of birth. APGAR score at 1-minute and 5-minute were noted. Careful examination was made to find out fetal and maternal outcome. Data were analyzed by Statistical Package for Social Science (SPSS™ version-23). Statistical significance was set at 0.05 level and confidence interval at 95 percent level.

Results: Among 64 cases of preterm (gestational age < 37 weeks) preeclampsia mother, 15%, 10% & 15% fetus were reported as early perinatal death, intra uterine death & fresh still birth respectively. Of all the live births of preterm preeclampsia mothers, 73.68% cases were referred to Special Care Baby Unit (SCABU) due to low birth weight and 26.32% for birth asphyxia. Sixty-six percent mothers had no complications in preterm (< 37 weeks) preeclampsia. PPH, abruption-placenta and eclampsia developed in 25%, 6% & 3% cases respectively

Conclusion: To avoid or reduce fetal, neonatal and maternal morbidity and mortality among preterm pre-eclamptic women, careful examination and investigations are required for early detection and proper timely management.

Keywords: Feto-maternal outcome, Preterm Pre-eclamptic mothers.

Introduction:

Perinatal and maternal outcome in eclampsia cases is still poor due to inadequate antenatal care, financial restraints and non-availability of transportation facilities, and social taboos causing delay in management resulting in poor maternal and neonatal outcome.[1]

Preterm birth is defined as birth before 37 weeks of gestation, when preterm pregnancy is associated with preeclamsia it is called preterm preeclampsia. Preeclampsia usually occurs after 20 weeks' gestation and is a multi-system disorder. The incidence of preeclampsia at DMCH in the year 2010 was 3.76%.[2] As estimated by WHO worldwide approximately 60,000 women die each year from preeclampsia. The incidence in primigravidae is about 10% and multigravidas 5%.[2]

Numerous risk factors are associated with the preterm preeclampsia. Black races are at increased risk. Risk factors for developing preeclampsia are- primigravidae, multiple pregnancy, family history of preeclampsia, chronic hypertension, diabetes, increase insulin resistance, increase body mass index, inherited thrombophilia, renal disease even without significant impairment, low socio economic status, previous preeclampsia, anti-phospholipid syndrome, hydatidiform mole etc.[3] Overall preeclampsia complicates 5 to 6% of pregnancies, but this figure increases to up to 25%

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in woman with preexisting hypertension.[3] Various complications occur in both fetus & mother. Maternal complications include eclampsia, antepartum hemorrhage, acute renal failure, dimness of vision and even blindness, preterm labour, HELLP Syndrome (Hemolysis elevated liver enzymes & low platelet count), postpartum hemorrhage, puerperal sepsis, residual hypertension, recurrent pre-eclampsia, chronic nephritis. Fetal complications include intrauterine death, Intrauterine growth restriction, asphyxia, prematurity etc. Preeclampsia and unexplained IUGR appear to be independent entities. Women who develop preeclamsia are at increased risk for cardiovascular diseases later in life. Woman with a history of preeclampsia compared with women without such a history, have an increased risk for cardiovascular disease, including a fourfold increased risk for hypertension, two fold increased risk for ischemic heart disease, stroke and deep vein Thrombosis.[4] Preeclampsia increases risk for severe perinatal outcomes, mostly by reducing birth weight.[5] Women with preeclampsia undergoing labour induction had higher caesarean delivery rates compared with non-preeclampsia regardless of parity or gestational age.[5] Induction of labour and vaginal delivery could be attempted in woman with mild preeclampsia at 36 weeks. Decision of termination of pregnancy is difficult to attempt in preterm pre-eclamsia due to the prematurity, IUGR & its related complications of fetus. When deterioration of maternal condition is suspected with conservative management in preterm preeclampsia, termination is indicated. Once the termination is decided, then comes the question of mode of termination. Induction of labor and vaginal delivery may cause birth asphyxia due to prolonged induction delivery interval in the already compromised preterm baby. On the other hand, cesarean section may end up with a foetal loss due to prematurity adding risk of operative delivery in next pregnancy. Continuation of pregnancy is justified just to facilitate the foetal growth without compromising the maternal parameters. However, termination in preeclampsia is the only definitive management. So, judicial judgment has to be made between continuation of pregnancy or termination and the mode of termination. Dhaka Medical College Hospital is a tertiary referral center. A considerable number of patients with preterm preeclampsia are admitted in this hospital. This study was done to evaluate the feto-maternal outcome in preterm preeclampsia.

Materials and Methods

The prospective cross-sectional study was carried out in the department of Obstetrics & Gynaecology, Dhaka Medical College Hospital, Dhaka from 1/07/2010 to 31/12/2010. Diagnosed cases of preeclampsia before completion of 37 weeks of pregnancy, irrespective of their parity who were admitted in labor and antenatal ward during the study period were included in this study. Among the exclusion criteria were- Patients who were unable to give accurate history of their LMP or showed early USG or any antenatal care records to determine the gestational age; patients who had history of same irregularities of menstruation prior to existing pregnancy; patients suffering from other diseases like heart diseases, renal diseases, DM, jaundice, eclampsia, systemic hypertension; pregnancy with venereal diseases, patients with multiple pregnancies, congenital anomalies, Rh-incompatible pregnancy, pregnancy induced hypertension. Total 64 cases were selected by purposive sampling. After admission in the hospital, data were collected in a questionnaire through interviewing and physical examinations. Necessary investigations like urine analysis, hemoglobin level, creatinine level, USG were done. Gestational age was calculated from the first day of last menstrual period (LMP). After delivery, birth weight of newborn was measured by NNC weighing scale within 24 hours of birth. APGAR score at 1-minute and 5-minute were noted. Careful examination was made to find out fetal complication and if needed, was planned to refer to Neonatal Intensive Care Unit (NICU). Data were compiled and statistical analysis was done using computer based software, Statistical Package for Social Science (SPSS™ version-23). Statistical significance was set at 0.05 level and confidence interval at 95 percent level.

Results:

Table 1: Age Distribution of the study cases *(*n=64*)*

Age group in years	Number	Percent
15-20	10	15.63
21-25	36	56.25
26-30	09	14.06
31-35	07	10.94
>35	02	3.13
Total	64	100

Table 1 showed that 56.25 % patients were within 21-25 years, 15.6% were within 15-20 years and 14.0 % were within 26-30 years of age and rest 3.12% patients were above 35 years.

Table 2: Mode of delivery of preterm preeclampsia mothers *(*n=64*)*

Mode of delivery	Frequency	Percent
LUCS	41	64
Vaginal(Spontaneous/ Induced delivery)	23	36
Total	64	100

Of the 64 cases majority 41(64%) were delivered by LUCS whereas 23(36%) had either spontaneous/Induced vaginal delivery among preterm pre-eclamptic mothers (Table 2).

Table 3: Indication of LUCS among preterm pre-eclampsia patients *(*n=41*)*

LUCS Indication	Frequency	Percent
Abruption placenta (with live fetus)	3	7.7
Fetal distress	3	7.7
Severe preeclampsia	16	38.5
IUGR	6	15.4
Uncontrolled Hypertension	13	23.1
Total	**41**	**100.0**

Among the LUCS delivery 16(38.5%) cases were done due to severe preeclampsia not responding to conservative management, 13(23.1%) & 6(15.4%) were due to uncontrolled hypertension & IUGR respectively (Table-3).

Table 4: Maternal outcome of Preterm Preeclampsia *(*n=64*)*

Maternal Complications	Number	Percent
No Complication	42	66
PPHE	16	25
Elampsia	2	3
Abruptio-placenta	4	6
Total	**64**	**100.0**

Sixty-six percent mothers had no complications in preterm (< 37 weeks) preeclampsia. PPH, abruption-placenta and eclampsia developed in 25%, 6% & 3% cases respectively (Table 4).

Table 5: Final fetal outcome of preterm preeclampsia cases

Final outcome	Number	Percent
Alive	38	60,0
Early perinatal death	10	10.0
Intra Uterine Death	06	10.00
Fresh still Birth	10	15.00
Total	**64**	**100.0**

Table 5 reveals that 60% babies were discharged alive from the hospital. Among 64 cases of preterm (gestational age < 37 weeks) preeclampsia mother, 15%, 10% & 15% fetus were reported as Early perinatal death, IUD & Fresh Still Birth respectively.

Table 6: SCABU referral for fetal complication among all the births

SCABU referral for fetal complication	Frequency	Percent
LBW	28	73,68
Asphyxia	10	26.32
Total	**38**	**100.0**

Among 38 live births neonates of preterm pre-eclampsia mothers 28(73.68%) cases were referred to Special Care Baby Unit (SCABU) due to Low birth weight and 26.32% for birth asphyxia (Table-6).

Discussion:

Pre-eclampsia complicates about 5-6% of all pregnancies.[6] Pre-eclampsia remains a major cause of maternal and prenatal mortality and morbidity and is particularly devastating in developing countries. Preeclampsia is a multiorgan, heterogeneous disorder of pregnancy associated with significant maternal and neonatal morbidity and mortality. Because preeclampsia is a progressive disorder, in some circumstances, delivery is needed to halt the progression to the benefit of the mother and fetus. However, the need for premature delivery has adverse effects on important neonatal outcomes not limited to the most premature infants.[7]

Out of 64 preterm pre-eclamptic mothers, 36% mother terminated their pregnancy by vaginal delivery and 64% required Caesarean section (Table 2). Among the LUCS delivery 38.5% cases were done due to severe preeclampsia not responding to conservative management, 23.1% & 15.4% were due to uncontrolled hypertension & IUGR respectively (Table 3). In a study by Rahman[8], out of 100 pre-eclamptic mothers, 14% women delivered vaginally and 86% required caesarean section. Our finding was consistent with other studies. Decision in favor of caesarian section is time-honored method for the safety of both the mother and fetus.

In our study 66% mother had no complications and 25%, 6% & 3% cases developed PPH, abruptio placentae and eclampsia respectively (Table 4). A systematic review with a meta-analysis of clinical studies conducted up to 2014 was published in 2017.[9] It showed that the maternal outcomes did not worsen, despite a significant difference in the occurrence of placental abruption in women who underwent expectant management after the diagnosis of early-onset preeclampsia.[10] In a study by Rahman[8], 50% mother had no complications where 40% developed PPH. As it was observed that 66% women had no maternal complications it is assumed that, it may be due to timely and appropriate management-related to the mother and response of the mother positively to the given treatment.

In this current study, fetal outcome were 60% live births, 15% fresh still births, 15% early neonatal deaths and 10% IUD (Table 5). In another study[8], the fetal outcome was 51%

live births, 2% IUDs and no fresh stillbirth among 100 pre-eclamptic patients. Severe preeclampsia represents significant risk factor for intrauterine fetal demise, with estimated stillbirth rate of 21 per 1000.[11] Fetal outcome is poor than maternal outcome in case of preterm pre-eclampsia. Iatrogenic prematurity may be responsible for fetal mortality and morbidity. Preterm babies are at risk of complications like asphyxia, hypothermia, RDS, Infection etc. due to immaturity of various organs and also for the causes of preterm birth.

Among 38 live births neonates of preterm pre-eclampsia mothers around 74% cases were referred to Special Care Baby Unit (SCABU) due to Low birth weight and 26.32% for birth asphyxia (Table 6). In other study, among 100 patients 18% babies were low birth weight and 10% baby developed birth asphyxia[8]. Ødega ̊rd et al.[12] showed pregnancies complicated by severe preeclampsia had infant birth weights 12% lower than expected, while pregnancies with mild preeclampsia showed no difference in weight gain from expected norms. In a population- based study of neonatal morbidity in the United States, the incidence of RDS was 7.4% at 34 weeks, 4.5% at 35 weeks, 2.3% at 36 weeks, and 1.2% at 37 weeks[13]. The findings of this current study were consisted with other studies as both condition, preterm and preeclampsia were responsible for the low birth weight baby.

Conclusion:

It is assumed that maternal outcome is better than fetal outcome. To avoid and/or reduce fetal and neonatal morbidity and mortality among preterm pre-eclamptic women, careful examination and investigations are required for early detection and management. Preeclampsia is not a totally preventable disease. However, several factors known to increase the risk of pre-eclampsia can be addressed before pregnancy and are therefore at least potentially amenable to intervention before attempts at conception

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Original Article

Maternal and Fetal Outcome in Postdated Pregnancies in a Tertiary Care Hospital in Dhaka: An Experience of 120 Cases

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Abstract

Background: Postdated pregnancy has always been regarded as high risk condition because perinatal mortality and morbidity is known to rise.

Objective: To determine the maternal and fetal outcome in women with postdated pregnancies in a tertiary care hospital of Dhaka city.

Methodology: This cross- sectional study was carried out on 120 patients at Department of Obstetrics and Gynecology in Combined Military Hospital, Dhaka. All singletons uncomplicated pregnancies with vertex presentation above 40 weeks 1 day and above, admitted in the ward were included in the study. Exclusion criteria were strictly followed. Gestational age was calculated by last menstrual period or from first trimester scan. Induction of labour was given in the selected group. All the information including neonatal notes were collected in a structured questionnaire. The data were analyzed by statistical software SPSS version 16.

Results: Among 120 patients with postdated pregnancy 108 (90%) delivered between 40 weeks 1 day-41 weeks and 12(10%) delivered > 41 weeks. About 81% patients had regular antenatal checkup about 16% had irregular checkup. Majority 74 (61.7%) delivered by vaginal route and 46 (38.3%) underwent cesarean section. Out of 74 patients 28(37.8%) delivered spontaneously and 46(62.2%) delivered after induction with injection oxytocin, prostaglandin tablet or gel and surgical induction. About 94% mothers had no complications and about 81% fetuses were healthy.

Conclusion: It can be concluded that timely intervention can improve the maternal and fetal outcome in postdated pregnancy.

Keywords: Postdated, Bishop Score, Induction of Labor, Fetal Distress.

Introduction:

Postdated pregnancy has been defined as the pregnancy that exceeds 280 days or 40 weeks of gestation. Expected date of delivery (EDD) is most reliably determined early in the pregnancy and may be based on the last menstrual period (LMP) in woman with normal regular menstrual cycle. If the estimated gestational age by a patient's LMP is difficult to determine, then EDD is obtained by ultrasonography especially in the early weeks of pregnancy.[1] Postdate, post term, post-maturity and prolonged pregnancy is accepted term by WHO and the International Federation of Gynecology and Obstetrics to describe pregnancy beyond dates (expected date of delivery).[2] It has been reported that in a pregnancy which has crossed the expected date of delivery, there is an increased risk of oligohydramnios, meconium stained amniotic fluid, macrosomia, fetal post-maturity syndrome and caesarean delivery, all of which jeopardize the baby as well as the mother. The interest in postdatism (just beyond the expected date of delivery) has been recent and the management is controversial.[3] Induction of labour is an obstetric procedure in which termination of pregnancy is done at or after the viable age of gestation by the use of some interventional methods (Medical, Surgical or Combined) with the purpose of initiation of labour and vaginal delivery and such intervention is done to serve the interest of either the mother or baby or both.[4] Meta-analysis of randomized controlled trials demonstrates that a policy of induction of labour for pregnancies at or beyond 41 weeks as compared to expectant management of gestation is associated with fewer perinatal deaths.[5] Induction of labour is increasingly becoming a practice but caesarean section is also comparatively more for complications in case of postdated pregnancy. The aim of this study is to find out the mode of delivery and outcome of labor which crossed the expected date of delivery and also to evaluate the maternal and fetal outcome.ut this figure increases to up to 25%.

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Materials and Methods:

The study was carried out in the department of Obstetrics and Gynecology of Combined Military Hospital, Dhaka from October 2016 to May 2017. A total of 120 patients

beyond 40 weeks of gestation admitted in this Hospital had been taken in the study groups. Inclusion criteria were antenatal cases beyond 40 weeks of gestation with regular menstrual cycles and known LMP or with first trimester scan and with singleton pregnancy with vertex presentation. Exclusion criteria of this study were congenital anomalies of fetus, chronic hypertension, pregnancy induced hypertension (PIH), preeclampsia and eclampsia, preexisting or gestational diabetes, heart disease, antepartum hemorrhage, placenta previa, intrauterine death and previous cesarean section.

The data were collected using a prepared questionnaire in a structured form from history, examination and investigation records by means of personal interview with the patients after taking written consent from the pregnant women as per inclusion criteria and 120 cases were selected. Pregnancy was dated according to last normal missed period and early ultrasound scan. Amniotic fluid index (AFI) in centimeters was assessed. Bishop score was assessed and induction of labour was started. Tablet Misoprostol was given in patients with AFI >8. Details of labour were noted down Induction delivery interval was calculated, mode of delivery and fetal outcome were recorded. Limitation of the study was lack of facilities of internal CTG, fetal scalp blood pH and no availability of automatic oxytocin infusion pump.

The data were analyzed by statistical software SPSS version 16.

Results:

Table 1: Distribution of patients according to age group (n=120)

Age group (years)	No. of Patients	Percentage
18-20	34	28.33
21-30	76	63.33
>30	10	8.33

Table 1 shows majority about 63% were in age group between 21-30 years, about 28% cases were in age group 18-20 years, and above 30 years were about 8%.

Table 2: Distribution of parity & duration of pregnancy in weeks (n=120)

A. Gravida	No. of Patients	Percentage
Primigravida	48	40
Multigravida	72	60
B. Duration of Pregnancy	**No. of Patients**	**Percentage**
40 weeks 1 day - 40 weeks	108	90
41 weeks - 42 weeks	12	10

Table 2 shows primigravida patients were 40% and multigravida were 60%. Maximum 90% cases were of gestational age between 40 weeks 1 day and 41 weeks and only 10% cases were beyond 41 weeks.

Table 3: Distribution of Antenatal checkup (n=120)

ANC	No. of Patients	Percentage
Regular Check Up	98	81.7
Irregular Check Up	19	15.8
No Check Up	3	2.5

Table 3 shows about 81% percent were booked cases and 2.5% cases did not do any checkup during pregnancy. About 16 % patients did irregular antenatal checkup.

Table 4: Distribution of Mode of Delivery (n=120)

Mode of Delivery	No. of Patients	Percentage
Spontaneous VD	28	37.8
Induced VD	46	62.2
Total VD	74	61.7
LSCS	46	38.3

Table 4 shows vaginal deliveries were conducted in about 62% cases and about 38% were delivered by lower uterine caesarean section. Out of 74 cases of normal vaginal deliveries, 28(37.8%) were delivered spontaneously and 46(62.2%) were induced.

Table 5: Comparison of patients with Gestational age and onset of labour (n=74)

Gestation age (week)	No. of Patients	Spontaneous labor No.(%)	Induced labor No.(%)
40 weeks 1 day - 41 weeks	66	23 (31.08)	43 (58.1)
>41 weeks	8	5 (6.7)	3 (4.05)

Table 5 shows among 74 cases of vaginal deliveries within 41 weeks about 31% delivered spontaneously and about 58% were induced. Beyond 41 weeks about 5% delivered spontaneously and 3% were induced.

Table 6: Distribution of type of induction of labour (n=46)

Types of Induction	No. of Patients	Percentage
Oxytocin	24	52.2
Tab Misoprostol	12	26.1
ARM	08	17.4
Prostaglandin Gel	02	4.3

Table 6 shows among 46 patients who were delivered by vaginal route after induction of labour, about 52% were by injection oxytocin, about 26% by tab misoprostol and about 4% delivered after insertion of prostaglandin gel and about 17% needed artificial rupture of membranes.

Table 7: Indications for lower segment cesarean section (n=46)

Indication of LSCS	No. of Cases	Percentage
Failed Induction	10	21.7
Non-reassuring CTG	11	23.9
Meconium stained liquor and fetal distress	12	26.9
Cervical dystocia	2	4.3
CPD	6	13.0
Non-progress of labour	5	10.9

Table 7 shows among 120 cases of postdated pregnancies total number of cesarean section were 46. Majority were for meconium stained liquor and fetal distress 12(26.9%), followed by Non-reassuring CTG 11(23.9%) and failed induction 10(21.7%) cases etc.

Table 8: Distribution of maternal complications (n=120)

Complications	No. of Cases	Percentage
Atonic PPH	3	2.5
Cervical tear	2	1.65
Perineal tear	1	0.83
Shoulder dystocia	1	0.83
No complications	113	94

Table 8 shows out of 120 cases of postdated pregnant mothers about 113(94%) had no complications, only 3(2.5%) mothers developed atonic PPH, about 1% cases of cervical tear and others had very few complications like shoulder dystocia.

Table 9: Distribution of fetal complications (n=120)

Fetal Complications	Number	Percentage
Fetal distress	10	8.3
Meconium aspiration syndrom	3	2.5
Birth asphyxia	8	6.6
Macrosomia	1	0.83
Jaundice	1	0.83
No complications	97	80.8

Table 9 shows in this study about 81% fetuses had no complications. Fetal distress was found in about 10(8%) cases, birth asphyxia in about 6.6% of cases, meconium aspiration syndrome in about 3(2.5%) cases, and other complications were very less.

Discussion:

This study was conducted in eight months' duration and included both primigravida and multigravida patients beyond 40 weeks of gestation who were admitted in Department of Obstetrics and Gynecology in CMH Dhaka. Nineteen trials randomizing women with uncomplicated pregnancies at 41 or more week's gestation to induction or expectant management with surveillance were identified.[6] In this study about 28% cases were between age group of 18-20 years, 63% were of 21-30 years and about 8% were above 30 years. Rithika et al found in her study 79% were under 20-25 years, 19% were under 26-30 years and 2% cases were under 31-35 years.[2]

Akhter SN et al in their study in ICMH, Dhaka found about 55% of the postdated pregnancy were multigravida and about 44% were primigravida[1] but in this study 40% cases were primigravida and 60% cases were multigravida. The study is comparable to other study in abroad and in other hospital of Bangladesh. Chhabra S et al found 68.66% were primigravida and 31.34% were multigravida.[3] Accurate dating of pregnancy is essential in order to prevent prematurity or postdatism. Studies have shown that most pregnancies undergoing post term induction are not post term when assessed by ultrasound date.[7] We found in our study that 90% cases were gestational age within 40 weeks 1 day -41 weeks and 10% cases were> 41 weeks. Chhabra S et al in Mahatma Gandhi Institute of Medical Science, India found 62% were between 40-41 weeks and 26.07% cases between 41-42 weeks and 12% beyond 42 weeks.[3]

Contact with the medical personnel during the gestational period is a significant contributor to the better pregnancy outcome. In this study about 81% cases visited regularly for antenatal checkup, about 16% cases made their visit irregularly and about 2.5% had no antenatal checkup. The high incidence of antenatal checkup is possibly due to free medical services in Defense services. But Ritika B et al found in her study that 82% were booked cases and 18% were not booked.[2]

In this study about 61% cases delivered by vaginal route and 38% were delivered by lower uterine cesarean section. This is contradictory with the findings of Akhter SN et al that normal vaginal delivery was conducted in about 27% and majority 73% delivered by LUCS.[1] Sultana R et al showed in her study 60% were delivered by vaginal route and 40% were delivered by cesarean section.[4] Aisha Arif et al found in their study that most of the cases had spontaneous vaginal delivery **i.e.** 76**%**, 15**%** had cesarean section and 9**%** had vacuum vaginal delivery**.**[8] Same results were found in study done by Aaron B in which spontaneous vaginal delivery was found in 80**%** cases, cesarean section in 12**%** and vacuum vaginal delivery in 8**%** cases.[9] The primary objective of treatment should be to identify the fetus at risk and thereby to plan an appropriate management. The decision regarding the expectant versus active management of postdate pregnancy should depend on balancing the effectiveness of induction against the effectiveness of increased fetal surveillance.[3] In this study among 61% of patients who were delivered by vaginal route about 37% were spontaneous and about 62% were delivered with induction with injection oxytocin, tablet

misoprostol, prostaglandin gel and ARM. A similar study was done by Jahan,1990, showed that 63% patients with unfavorable cervix required cesarean section after induction of labour and their study no prostaglandins was used for cervical ripening.[10] This study showed better outcome in patients with unfavorable cervix than the previous study. Ambreen Navid found that the higher number of vaginal deliveries in 41weeks of gestation was independent of association between the induction agent, parity and mode of delivery.[5] Another feature of this data is the use of mean vertical pool of liquor at term as induction criteria. In this study tablet misoprostol was given in patients with AFI up to 8cm. Although, an Indian study showed that an AFI of 5cm or less was a better predictor of fetal distress than a mean vertical pocket of 3cm or less.[11]

In these study indications for cesarean section were meconium stained liquor, fetal distress, no reassuring CTG, cervical dystocia, cephlalopelvic disproportion, non-progress of labour and failed induction. In another study indication for cesarean section were fetal distress, failed induction, chorioamnionitis and failure to progress in labour. Only 3 cesarean sections were performed for failed induction which shows that induction of labour is not associated with increased cesarean rate as evidence by RCOG guidelines.[12]

Cunninghum FG found that post term pregnancy associated with an increased risk of postnatal mortality and morbidity including meconium aspiration syndrome, oligohydramnios, macrosomia, fetal birth injuries, septicemia, rate of non-reassuring fetal heart rate, fetal distress in labour and maternal complications increased caesarean section, cephalopelvic disproportion, cervical tear, dystocia, postpartum hemorrhage.[13] In this study about 94% mother had no complication, atonic PPH was in 2.5% cases which were managed by conservative treatment, cervical tear in 1.65% cases and perineal tear and shoulder dystocia was less than 1%. Kana R Odedara and Pooja A Kamaria found in their study that 6% mother developed atonic PPH, 5% had cervical tear, 1% had perineal tear, 1% had shoulder dystocia and 87% mother had no complications.[14] Post term pregnancy (pregnancy beyond 294 days) is associated with an increased risk of fetal and neonatal mortality as well as an increased maternal morbidity.[15]

Fetal wellbeing was monitored in this study by assessing FHR and color of the liquor during labour. We used only external CTG but there was no scope for fetal scalp blood PH. As there is high false positive result in CTG, so there might be some error in fetal monitoring clinically. To avoid fetal risk, cesarean section rate might be higher in this study. We found in our study about 8% cases were fetal distress, 2.5% cases had meconium aspiration syndrome, macrosomia in 0.8% cases and about 82% had no complications. Sultana R et al found in their study 80% were healthy baby, 16% had asphyxiated baby and only 4% had other complications.[4]

Conclusion:

Management of postdated pregnancy is a challenge to obstetrician and proper planning and monitoring is mandatory to reduce unwanted complications. The fact is that postdated pregnancy does not necessitate a hasty line of management towards operative delivery. The women should be offered induction of labor before 42 weeks to avoid adverse neonatal consequences. It can be stated that the patients at 41 weeks have a good fetal outcome who delivered either by spontaneous or induced. If the fetal monitoring and the uterine activity of the patients with labor would be possible to monitor by intra-partum CTG, fetal scalp blood PH and automatic infusion Oxytocin pump then there is more chance of reduction of rate of caesarean section.

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Original Article

Correlation between Serum Uric Acid and Dyslipidemia in Hypertension

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Abstract

Background: Hypertension (HTN) is an important medical and public health issue because it increases the risks of cardiovascular and kidney disease. Serum uric acid (SUA) is thought to have an association with hypertension and plays a role in the pathogenesis of hypertension. Assessment of association between dyslipidaemia, hyperuricaemia and hypertension, may enable use of Serum lipid profile and serum uric acid level as good screening tools to aid the risk stratification of individuals at risk of developing hypertension.

Objective: To evaluate the correlation between serum lipid profile and serum uric acid in hypertension.

Methods: This case-control study was conducted in the Department of Biochemistry, Sir Salimullah Medical College (SSMC, Dhaka during the period of July 2012 to May 2014.A total of 100 individuals were selected as study subjects. Among them 50 diagnosed cases of hypertension were selected as cases and 50 normotensive healthy individuals as controls. Serum uric acid and serum lipid profile were measured in all study subjects. Collected data were analysed by Statistical Package for Social Science (SPSS) version 12.0.

Results: The study population mostly belonged to 36 to 45 years of age group. The mean ±SD) levels of serum TG (205.24 ± 56.74mg/dl), TC (225.44 ± 60.67mg/dl), LDL-C (134.34 ± 49.58mg/dl) and Serum uric acid (7.05 ±1.31 mg/dl) in hypertensive patients were significantly higher than those in controls whereas mean level of HDL-C (42.10± 12.27 mg/dl) in hypertensive patients were lower than those of controls. The study showed a positive linear correlation of Serum uric acid with TG (r=0.455 p=0.001), TC (r=0.504 p=0.001), LDL-C (r=0.425 p=0.002) and negative correlation with HDL-C (r= -0.158 p=0.274) in the hypertensive.

Conclusion: From the study it is evident that dyslipidaemia is correlated to hyperuricemia in hypertension.

Keywords: Hypertension, Serum lipid profile, Serum uric acid

List of Abbreviations Used

Hypertension (HTN), Serum uric acid (SUA), Cardiovascular disease (CVD), Total cholesterol (TC), Triglycerides (TG), Low Density Lipoprotein (LDL-C), High Density Lipoprotein (HDL-C) Very Low Density Lipoprotein(VLDL), Congestive cardiac failure(CCF).

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Introduction:

Hypertension (HTN) is an important medical and public health issue because of its high prevalence and also because it increases the risks of cardiovascular and kidney disease. Among individuals aged 40 to 90 years, each 20/10 mmHg rise in blood pressure doubles the risk of fatal coronary events.[1] HTN accounts for an estimated 54 percent of all strokes and 47 percent of all ischemic heart diseases globally.[2] The prevalence of HTN is increasing overtime. More than a quarter of the global adult population is currently hypertensive and this figure is projected to rise to 60% by 2025. It is a matter of great concern that almost three quarters of the global population with HTN live in developing countries, which contributes to widening of the existing global health disparities. [3,4] Bangladesh Non-communicable Disease Risk Factor Survey 2010 carried out among adults aged above 25 years, reported prevalence of HTN to be 17.9%.[5] Reports of national surveys conducted in various countries in North America, Asia, and Africa indicate that although the prevalence of HTN is high, it is poorly treated and/or controlled.[6]

It is plausible that serum uric acid (SUA) has an association with hypertension and plays a role in the pathogenesis of hypertension. Much evidence suggests that increased SUA may be a significant modifiable risk factor for development of hypertension. A study conducted in Australia, showed that the prevalence of hyperuricaemia is 31% among untreated hypertensive subjects[7], as compared to that of 10% among healthy general population.[8]

Abnormalities in serum lipid and lipoprotein levels (dyslipidemia) are recognized as major modifiable risk factors for cardiovascular disease (CVD)[9] and have been identified as independent risk factors for essential hypertension giving rise to the term dyslipidemic hypertension. [10,11]

Dyslipidemia is more common in untreated hypertensives than normotensives.[12,13] Many studies have shown that total cholesterol (TC), triglycerides(TG) and virtually all fractions of lipoproteins tend to be more frequently abnormal among hypertensive patients than in the general population.[14]

Hypertension is known to be associated with alterations of lipid metabolism in which there is rise of serum lipid and lipoprotein levels. It has also been documented that presence of hyperlipidaemia substantially worsens the prognosis in hypertensive patients.[15] It is recognized that early detection of modifiable risk factors along with drug treatment can prevent and delay hypertension. Although hypertension is one of the leading causes of death and disability in Bangladesh, it still lags behind in detecting, treating and controlling blood pressure.[16]

Hyperuricaemia and dyslipidaemia may be modifiable risk factors for HTN. It may be possible to reduce the incidence of hypertension by curbing down hyperuricaemia and dyslipidaemia.

Materials and Methods:

This case-control study was conducted in the Department of Biochemistry, Sir Salimullah Medical College (SSMC), Dhaka during the period of July 2012 to May 2014. The study included 50 cases hypertensive patients of both sexes above 30 years old attending the Medicine OPD of Bangabandhu Sheikh Mujib Medical University (BSMMU) and SSMC. Fifty controls with age and sex matched non-hypertensive subjects were also taken. They were taken from the medical or paramedical staff, attendants of patients and outdoor patients with minor illness. The exclusion criteria were:patients with diabetes mellitus, thyroid disorder, heart disease, renal impairment and liver disease and patients taking certain medication like thiazide and loop diuretics, cytotoxic drug, antitubercular drug, aspirin, and medications used to reduce uric acid and lipid level. Smokers and alcohol abusers were also excluded.A thorough physical and relevant clinical examination was performed. Blood pressure(BP) was measured in a sitting position. Laboratory investigation of Serum lipid profile and uric acid wre done. Collected data were checked, edited, processed and statistical analysis were performed using Statistical Package for Social Science (SPSS) version 12.0.

Results:

Table 1: Distribution of study subject according to age

Age (years)	Group		p value
	Case (n=50)	Control subjects (n=50)	
<35	6 (12.0)	8 (16.0)#	
36 – 40	18 (36.0)	21 (42.0)	
41 – 45	19 (38.0)	15 (30.0)	
46 – 50	7 (14.0)	6 (12.0)	
Mean ± SD	40.78 ± 4.00	39.58 ± 4.12	0.142*

The mean± SD age of hypertensive patients was 40.78 ± 4.00 years and that in controls was 39.58± 4.12 years. Age of most of the study subjects were between 36 to 45 years (74% in hypertensive and 72% in control subjects). There were no statistically significant differences observed the groups in respect of mean age (p=0.142) as shown in Table 1.

* Data were represented as Mean ± SD and Unpaired 't' test was done to measure the level of significance.

#Figure within parentheses indicates percentage.

Table 2: Comparison of lipid profile and serum uric acid between cases and controls

Biochemical findings	Group		p value
	Hypertensive patients	Controls	
TG (mg/dl)	205.24 ± 56.74	136.84 ± 52.81	0.001
TC (mg/dl)	225.44 ± 60.67	175.72 ± 45.54	0.001
HDL-C (mg/dl)	42.10 ± 12.27	48.10 ± 12.43	0.017
LDL-C (mg/dl)	134.34 ± 49.58	101.74 ± 38.83	0.001
Serum uric acid (mg/dl)	7.05 ± 1.31	4.84 ± 1.27	0.001

In Table 2, biochemical parameters revealed that the mean (± SD) of Serum TG, TC, LDL-C and SUA in the hypertensive patients were 205.24 ± 56.74 mg/dl, 225.44 ± 60.67 mg/dl, 134.34 ± 49.58 mg/dl and 7.05 ± 1.31 mg/dl respectively which were significantly higher when compared with those of controls (p=0.001). Mean(±SD) of serum HDL-C in hypertensive and controls were 42.10 ± 12.27 mg/dl and 48.10 ± 12.43 mg/dl respectively showing significantly lower levels in the hypertensive subjects ($p<0.05$).

Data were represented as Mean ± SD and Unpaired 't' test was done to measure the level of significance.

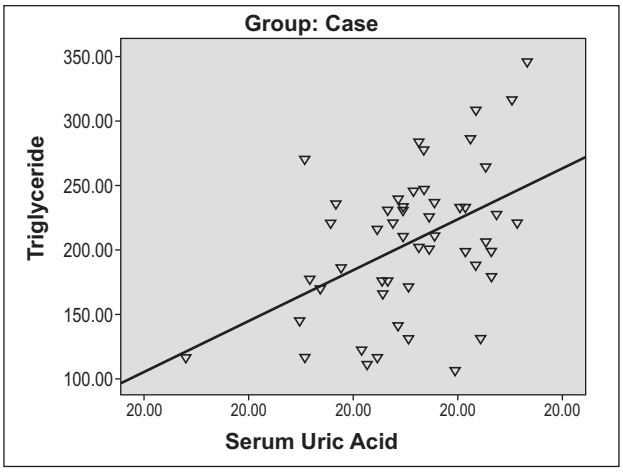

Figure 1:Correlation between serum uric acid and triglyceride in cases. Pearson correlation, r is 0.455 with a p value of 0.001

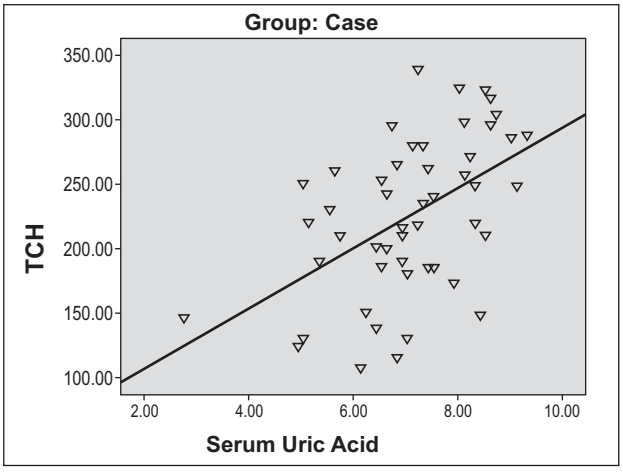

Figure 2: Correlation between serum uric acid and TC in cases. Pearson correlation, r is 0.504 with a p value of 0.001

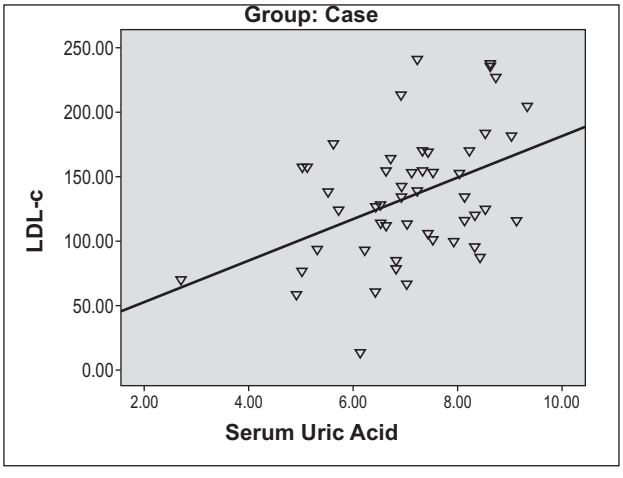

Figure 3: Correlation between serum uric acid and LDL-C in case. Pearson correlation, r is 0. 425 with a p value of 0.002

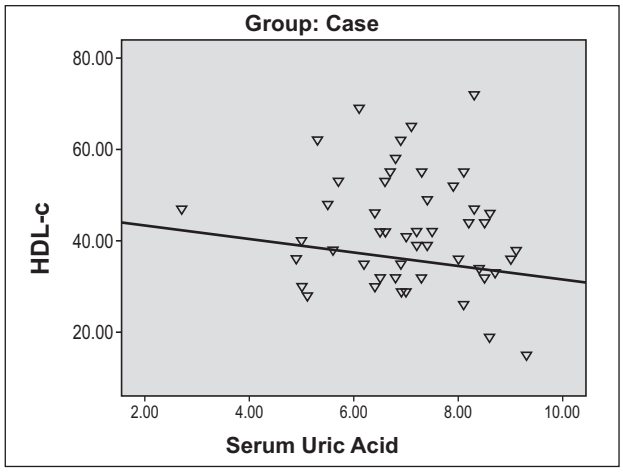

Figure 4: Correlation between serum uric acid and HDL-C in cases. Pearson correlation, r is -0.158 with a p value of 0.274

Discussion:

The present study shows that serum TG, TC, LDL-C were significantly higher in hypertensive patients than those of normotensive controls.Saha et al[17] revealed similar findings of elevated serum TG, TC and LDL-C in the hypertensive subjects when compared to that of controls. Present study showed that serum HDL-C was significantly lower (p=0.017) in cases than controls and this finding agree with the finding of the study done by Baldawi-Al and Ali.[18]

Dyslipidemia in hypertension is due to lipid deposition in lumen of arterial wall, causing atherosclerosis. This increases the resistance to flow of blood in blood vessel, causing hypertension.HDL-C is a protective factor decreased on hypertensives, suggesting more risk of developing complication of hypercholesterolemia. High cholesterol influence adrenergic stimulation and therefore, damage to organs like Heart, Kidney and Liver–called Target Organ Damage-is more in hypertensive.[19] Epidemiological evidence suggest that low HDL-C is an independent risk factor for CVD and they suggested that interventions to increase HDL–C will yield clinically significant benefits against the development of coronary heart disease (CHD).[20-21] Increased release of free fatty acids from adipose tissue, particularly from visceral fat into the portal veins, stimulates the production of triglyceride-rich lipoproteins in the liver with the release of more and larger VLDL particles. This in turn has effects the other lipoprotein particles, resulting in reduced levels of HDL-C.[22] LDL-C is vasoconstrictor, mitogenic, proinflammatory and thrombogenic. So it's rise in hypertensive is a risk for developing complications.[23]

In the study, the mean serum uric acid level in cases was 7.05±1.31 mg/dl which was close to that found by Feig et al[24] who found mean uric acid level to be 6.9 mg/dl in their study patients. Uric acid has been implicated in

hypertension through its probable role in mediating hypertension via mechanisms like inflammation, vascular smooth muscle cell proliferation in renal microcirculation, endothelial dysfunction and activation of the 'renin–angiotensin– aldosterone' system.[25]

Our study shows a positive linear correlation between SUA and TG, TC, LDL-C. This finding is consistent with studies of Zhang W et al, Nakanishi N et al and Bonora E et al.[26-28] Present study showed a negative correlation of SUA with HDL-C in hypertensive patients. This finding also agrees with the findings of Ishizaka et al.[29]

In hypertension, there is enhanced proximal tubular reabsorbtion and depressed tubular secretion of uric acid causing hyperuricemia.[30] Hypertension complications like CCF, Heart failure has more endothelial dysfunction due to dyslipidaemia and raised uric acid.[31]

Conclusion:

The mean TG,TC, LDL-C and SUA level were significantly higher in hypertensive cases, as compared to those of healthy normotensive control.We also found that a positive correlation of SUA and TG,TC, LDL-C.Therefore, it can be concluded that dyslipidaemia is correlated to hyperuricemia in hypertension.Further larger scale study is needed for assessment of association with strength of association and risk relationship between dyslipidaemia, hyperuricaemia and hypertension.Serum lipid profile and serum uric acid level could be a good screening tool to aid the risk stratification of individuals at risk of developing hypertension, if these findings are confirmed by larger scale studies.

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Original Article

Knowledge and Practice of Self-Breast Examination among Medical Personnel of a Selected Tertiary Care Hospital

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Abstract

Background: Breast cancer appears to be a disease of both the developing and developed worlds. Among Bangladeshi women, breast cancer is the leading cause of cancer-related deaths.

Objective: To determine knowledge and practice of self-breast examination and to evaluate health beliefs concerning the model that promotes breast self- examination (BSE) in a group of women aged 20– 65 years.

Methods: This descriptive study was conducted on 571 women of aged 20-65 years, who were staffs and female students of Bangladesh Medical College Hospital. Informed consent was taken from the participants. Sampling technique was purposive. They filled up the "Self-Breast Examination form" and a self-administered questionnaire. The questionnaire consisted of sociodemographic variables and variables about knowledge, practice and believes of self-breast examination. Collected data were analyzed by SPSS.

Results: A total of 571 participated in this study. Majority (70%) were in the age group of 20-29 years. Most of the participants (43.8%) were nurse followed by 23.5% female medical students. Only 5% responder had previous breast problems. 7.5% (43) had history of breast cancer in family. Near 58% (332) respondents had knowledge of SBE and 42% (239) had no knowledge of SBE. About 48.2% (275) respondent performed SBE, among them 10.2% did SBE regularly, 89.8% did SBE irregularly and 51.8% (296) respondents never performed SBE. About 100% of the participants believed that BSE is important to detect a breast lump in an early stage and can help to reduce morbidity and mortality

Conclusion: The breast self-examination rates were insufficient in the study group. In addition to training women breast self-examination for early diagnosis, the breast cancer risk questionnaire - an easy, simple and cost-effective tool –may be recommended to administer in the primary health care centers.

Keywords: Knowledge, Practice, Self-Breast Examination, Medical Staff, Tertiary Care Hospital.

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Introduction:

Breast cancer is the most frequent type of cancer and cause of death in women all over the world.[1,2] Breast cancer (BC) composes about 23.0% of all cases of cancer in women with 1.38 million new cases in the world every year, so it is a major public health concern.[3] According to the World Cancer Report published in 2008 by the World Health Organization (WHO), breast cancer is one of the most frequent types of cancers in women globally.[4,5] In 2013, 1,8 million breast cancer cases and 464 million deaths were reported in the world.[6,7]

Early diagnosis is the most effective way for health protection and disease prevention and to reduce the morbidity and mortality in breast cancer. Clinical breast examination and mammography are the primary methods recommended for the early diagnosis of breast cancer.[8] Although there are various views and studies about the effectiveness of self-breast examination (SBE), it is recommended to detect the palpable breast tumors and it is stated to be effective for increasing awareness of breast

health in women especially in developing and under-developed countries.[9]

Many women miss early detection and treatment opportunities due to lack of knowledge, information, and awareness of BC, as well as cancer screening practices. Hopefully, the outcome of this study will help to raise awareness among females and improve future research in BC. The purpose of this study was therefore to determine the knowledge, perception and practice on BC screening and to find out sociodemographic data, knowledge and perception of BC and practice of BSE among female staff and students of Bangladesh Medical College and Bangladesh Medical College Hospital.

Materials and Methods:

We conducted a cross-sectional, descriptive study to identify knowledge and practice about BC screening among the students and female staff of Bangladesh Medical College &Hospital. It was conducted on both the Campuses of Bangladesh Medical College and Bangladesh Medical College Hospital, and the study period was from October 2016 to March 2017. The sample size was 571. The questionnaire comprised of sociodemographic variables, variables on knowledge, practice and believes on SBE. All the female students and staff who agreed to participate and were available during the study period were included. The respondents were informed about the study and the consent was taken through the informed consent form. Study participants were distributed a questionnaire. Participants' responses and information were kept strictly confidential and the raw data were then collected and entered into the Microsoft Excel and was analyzed by using the SPSS.

Results:

Table 1: Sociodemographic features of the study group (n-571)

A) Age group	Number	Percent
20-29	400	70
30-39	101	17.7
40-49	53	9.3
50-59	14	2.4
60-69	3	0.6
B) Marital status	**Number**	**Percent**
Unmarried	312	54.6
Married	259	45.4
C) Profession of participant	**Number**	**Percent**
Nurse	250	43.8
Doctor	92	16.1
Medical Student	134	23.5
Attendant/ Cleaner	95	16.6

Table 1 shows a total of 571 medical staff of different level and female students participated in this study. The participants' age range was 18–64 years; majority (70%) were between 20-24 years. About 54.6% were unmarried and 45.4% were married. Most of the participants (43.8%) were nurse followed by 23.5% female medical students.

Table 2: Distribution of previous history of breast problem (n=571)

Previous history of Breast Problem	Number	Percent
Present	28	4.9
Absent	543	95.1

Table 2 shows majority 95.1 % had no history of previous breast problem and 4.9% had previous history of breast problem.

Table 3: Distribution of family history of breast cancer (n= 571)

Family History of BC	Number	Percent
Present	43	7.5
Absent	528	92.5

Out of 571 participants 92.5% had no family history and 7.5% had family history of breast cancer.

Table 4: Status of knowledge about Self Breast Examination (n=571)

Knowledge about SBE	Number	Percent
Present	332	58.1
Absent	239	41.9

About 58.1% had knowledge about SBE and 41.9% had no knowledge about SBE.

Table 5: Source of knowledge about SBE (n=571)

Source of knowledge of SBE	Number	Percent
Institute	474	83
Digital/Print media	76	13.3
Family/Friend	21	3.7

Of the 571 participants 83% participants' source of knowledge was institute, 13.3% was digital/print media and 3.7% was from family and friend.

Table 6: Status of Practice of SBE

A) Ever Performed SBE	Number	Percent
No	296	51.8
Yes	275	48.2
Total	571	100
B) Performed SBE	**Number**	**Percent**
Regular	28	10.2
Irregular	247	89.8
Total	275	100
C) Frequency of performing SBE in last year	**Number**	**Percent**
1-2 times	85	30.9
5-6 times	162	58.9
12 times	28	10.2
Total	275	100

Table 6A & B shows 51.8% participants never performed SBE and 48.2% performed SBE of which only 10.2% performed SBE regularly. Again table 6C shows out of 275 majorities (58.9%) performed SBE for 5-6 times and only 10.2% performed SBE regularly.

About 100% of the participants believed that BSE is important to detect a breast lump in an early stage and can help to reduce morbidity and mortality.

Discussion:

The literature supports the argument that regular practice of SBE influences treatment, prognosis and survival rates.[10,11] In this study only 10.2% of the participants reported practicing SBE on a regular monthly basis while 89.8% stated that they examined themselves irregularly. Similarly, some studies have reported that less than half of their study groups actually practice BSE monthly.[12, 13] In contrast, some studies have found that the majority of older women performed breast screening activities on a regular basis.[14,15,16]

Although 58% of the women in this study reported having heard or read about breast cancer, 83% of whom had acquired the information from institute. Nearly 13% of the study group reported their main source of information on SBE was obtained from the TV/radio or internet. In Hyun's study also reveals that women who are taught to perform SBE have a better level of knowledge about breast cancer.[17] Similarly, in Jirojwong's study, it was found that sociodemographic variables were not effective on SBE practice.[18]

In this study seriousness, susceptibility and motivation were not significant in explaining BSE performance on a regular basis, but increased confidence, BSE-benefit and reduced SBE-barriers were significantly associated with it. Benefits were a significant variable predicting BSE. It was supported by the findings of American studies that have reported where women who perceived more benefits from BSE behavior were more likely to perform BSE.[19,20]

Conclusion:

Our results indicate that an increase in BSE practice may be achieved through enhancement of breast cancer awareness and possibly by reducing barriers. About 83% of the participants that reported ever hearing or reading about breast cancer from institute, 13.3% of participant mentioned Television/radio and other digital media as their main source of information. So, the role of institutional health care workers as an information source in breast cancer should be increased. Further research is recommended using a larger sample size with women in rural and urban areas, including the cost-effectiveness of designing and implementing preventive care.

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Review Article

Confronting a Rising Tide of Adolescent Obesity: Treatment vs. Prevention and Policy

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Abstract

Adolescent obesity has become an increasingly urgent issue in low and middle income countries. Recent relevant advances include the application of the neurobiology of addiction to food addiction and obesity. The aetiology of obesity indicates the need for multilevel interventions that go beyond simple behavioural approaches. Additional research on food addiction and adolescent obesity in low and middle income countries, as well as program evaluations that examine the effects of complex interventions, is urgently needed.

Keywords: Adolescent, obesity, prevention, multilevel interventions, healthy setting.

Introduction:

The prevalence of overweight and obesity among adolescents has increased in many low and middle income countries.[1] These countries are now faced with the challenge of addressing this growing obesity epidemic while also contending with issues of food scarcity and under nutrition.[2] Overconsumption of high-calorie low-nutrient foods is due, in part, to changes in global food production. To save money and time, many consumers in have shifted from consuming meals prepared at home to consuming mass produced processed foods that are high in sugar, salt, and artificial ingredients.[2-5] The addictive nature of these ingredients has led to patterns of overeating.[6] This change in diet along with increasingly sedentary lifestyles is contributing to increase in obesity. Obesity outcomes are influenced by genetics[7] and epigenetic mechanisms[8] biological bases for food preferences[9] and biological mechanism for exercise.[10] American obesity rates are the highest in the world. Sixty-eight percent of adults are categorized as overweight, one-third of them are diagnosed as clinically obese. There are nearly 7 million overweight and obese children in the U.S. today. Childhood obesity statistics show that obese children have a 70% chance of becoming obese adults. The percentage increases to 80% if either one or both parents are obese as well.[11] Some 300,000 deaths are attributed to obesity-related diseases each year, making it the second-leading cause of preventable death. In addition, the estimated annual medical cost of treating obesity-related diseases in the United States is staggering: $147 billion.[12] According to the CDC, this rate is on the rise in the U.S. and has almost tripled in the last three decades.[11] The World Health Organization projects that, by 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese.[13] The trend has become obvious: As the Western diet of highly refined carbohydrate junk foods reaches developing countries, obesity rates increase.[14]

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Impact of food habit on obesity and other non communicable diseases

The excess fat, carbohydrates, and processed sugar found in junk food contributes to an increased risk of cardiovascular disease, diabetes, weight gain, hypertension, dyslipidemia, impaired glucose tolerance[15] and many other chronic health conditions.[16] Also consumers tend to eat too much at one sitting and consumers who have satisfied their appetite with junk food are less likely to eat healthy foods like fruit, vegetables or dairy produce.[17]

One of the major causes of food cravings, overeating, addiction, and delayed food intolerance in newborns, infants and toddlers is the dangerous addictive chemicals used by food manufacturers. These chemicals are used by food manufacturers in even baby food to cause the compulsive/obsessive eating that leads to obesity. Food colorings, MSG, pesticides, factory farmed meats, genetically modified fruits and vegetables and other additives have been linked to a range of health problems in children including allergic reactions, food intolerance, inflammation, headaches, asthma, growth retardation, hyperactivity, weight gain and other illness.[11]

Fast foods have high level of fat and sugars that are not only unhealthy but addictive and that creates a vicious cycle making it hard for children to choose healthy food. High content of trans fat in commercially available fast foods predispose children to risk of future heart diseases.[18] Energy density of fast food is more than twice the recommended daily allowance for children.[19] Fast food intake leads to higher proportion of calories being derived from total and saturated fat.[20] Moreover, the micronutrient content (carotene, vitamin A, vitamin C) of the fast food is also low.[21] Low levels of calcium and magnesium in the diet can contribute to osteoporosis. Diets rich in free sugars can lead to increased risk of dental caries. Fast food consumption and globalization of diet has lead to loss of traditional healthy food practices. One of the consequences of ready availability of cheap food outside the home is devaluation of cooking skills.

Role of Families

Families influence children's dietary choices and risk of obesity in a number of ways, and children develop food preferences at home that can last well into adulthood.[22] The food that families keep at home and how family members share meals influences what and how much they eat. Eating meals as a family has also been linked with increased child and adolescent intake of fruit and vegetables and other healthy foods.[23] Increased frequency of family meals has been linked with lower BMI in some studies[24, 25] but not in others.[26] Low-income families face additional barriers to healthy eating that may contribute to the higher rates of obesity seen in lower-income groups.[27] One roadblock is that healthy foods, such as vegetables, fruits, and whole grains, are more expensive than less healthful foods, such as refined grains and sweets, and may be too expensive for low-income families.[28] It also takes longer time to prepare healthful meals than to buy convenience foods or fast food. But now- a- days working parents may have less time for meal preparation and other household responsibilities.[28,29]

Role of Schools

Researchers have found that participating in the School Breakfast Program is associated with lower BMI in children, while participating in the lunch program did not affect obesity. Students participating in the School Breakfast Program were also less likely to skip breakfast, which may reduce risk of overweight by spreading food intake more evenly across the day.[30] Most schools sell foods to students outside of the school meal programs. These so-called "competitive foods" are widely available in the cafeteria, vending machines, and school stores[31] Eating competitive foods has been linked with poorer quality diets and increased risk of obesity in several studies.[32] In addition to unhealthy foods, schools have long provided a ready supply of sugar-sweetened beverages,[33] which are linked to increased risk of obesity and diabetes.[34] Although recent agreements between Alliance for a Healthier Generation and the American Beverage Association have substantially reduced the supply of sugary beverages, schools continue to offer students unhealthy sugar-sweetened beverages.[35]

Role of Worksites

Worksites often provide easy access to unhealthy foods in vending machines and limited access to healthier options, such as fruits and vegetables.[36] A number of studies have shown that making changes to the workplace food environment, such as offering more healthy foods in company cafeterias, results in improved diet quality.[37] Work environments can also increase the risk of obesity arising from job stress and work-related fatigue, which are linked to poor diets and reduced physical activity. Time at work also plays a role: Shift workers and employees working longer-than-usual hours every week have a higher risk of obesity.[38]

Pharmacological Treatments

Medications that are effective in reducing substance use are also effective for reducing food intake. Topiramate is thought to inhibit dopamine release in the mesocorticolimbic system, thus dampening the rewarding effects of alcohol.[39] Topiramate similarly appears to be effective in producing weight loss in obese individuals.[40] Rimonabant, a drug that blocks the cannabanoid receptors, has been tested as a treatment for both substance use disorders and obesity[41]. Preliminary findings suggested it was effective as a treatment for nicotine and alcohol dependence, as well as reducing food intake and improving lipid and blood sugar levels in obese patients.[42] However, rimonabant was associated with a high incidence of serious psychiatric side effects, leading the U.S. Food and Drug Administration to deny its approval.[43]

Strategies to Reduce the Trend of Fast Food

Considering the burden of non communicable diseases among adults and its origin to childhood dietary habits, fast food consumption needs to be curtailed among children and adolescents. Strategies for healthy food intake include availability of healthy standard foods, information campaigns and surveillance of diets and disease burden.

Increased consumption of fruit and vegetables

One of the most obvious unions between agriculture, health, nutrition and education is via the promotion of fresh fruit and vegetables. Most national and international dietary guidelines are in agreement that consumption of fresh fruit and vegetables is a healthy food choice and generally needs to be increased.[44] The benefits of fruit and vegetable consumption span the spectrum of nutritional disorders. Fruit and vegetables are rich sources of micronutrients, needed by children for optimal growth and development. Consumption of fruit and vegetables also decreases risks of obesity, cardiovascular disease and some cancers,[45] perhaps in part through their contribution of bioactive substances. Schools and child care facilities are an ideal place to promote fresh fruit and vegetables. Here children can be taught the health benefits of consuming them in adequate amounts. Appreciation of local produce can also be cultivated through exposing children to indigenous crops and teaching them about their nutritional properties.[46]

Regulation of Marketing

In 2008, the Federal Trade Commission (FTC) reported that the food industry spends almost $10 billion per year marketing food and beverages in the U.S. that appeal to children and adolescents, including $1.6 billion to target children and adolescents directly with soft drinks, fast-food, and cereal promotions.[47] Despite this widespread recognition of the negative impact of marketing unhealthy foods, the practice continues unabated. One recent study found that, from 2006 to 2008, food companies increased the use of licensed cartoon and other entertainment characters targeting young children, and that most foods

marketed with such characters failed to meet IOM standards for snack food suitable for school children.[48] Food marketers are increasingly using sophisticated digital marketing techniques to target youth across a host of platforms, including cell phones, video games, social media, and immersive "virtual worlds," prompting public health advocates to call for stronger government regulation and industry self-regulation.[49] Union of European beverages association (UNESDA), in addition has laid down guidelines to restrict marketing communication to be placed in printed media, web sites, or programs aimed at children. It directs not to directly appeal to children to persuade parents to buy their products, nor should the promotional activities encourage children to consume larger quantities for participation.[50]

Nutritional Labelling

Nutritional labeling refers to disclosure of nutritional content (calories, added sugar, total fat, trans fat, saturated fat, sodium and protein content) in product labels. Nutritional value should be provided in menu, menu boards, food wrappers and containers in fast food restaurant. This might restrict the quantity and choice of food among children of educated parents. In a recent study conducted on parents of children aged 3-6 years, it was observed that parents who were offered the nutritional value menu card ordered food of lesser calorie.[51] However in a study by Yamamota et al., it was observed that provision of nutritional value did not modify the food ordering behavior among the enrolled adolescents.[52] It has been often debated that labeling might result in financial loss to fast food industry, but it has been shown that restaurants which project lower fat menu have a better customer satisfaction.[53]

Conclusions:

Changes in lifestyle and loss of the family tradition of eating together trigger the popularity of fast foods among young people. The rise of the fast-food industry has influenced the social conditions of life in developed and developing countries in ways that can contribute to childhood obesity. Solutions to address obesity among adolescents in LMICs must approach this public health priority on a variety of levels from national policy to individual-level interventions and should take into account what is known about the neurobiology of food addiction. Policies must be made to discourage consumption of high-calorie low-nutrient foods and encourage consumption of healthy foods. Physical exercise must be encouraged throughout childhood and adolescence in the home, school, and community settings.

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Case Report

Acute Respiratory Failure in a Patient with Duchenne Muscular Dystrophy

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Abstract

Muscular dystrophies are a clinically and heterogeneous group of disorders that all share clinical characteristics of progressive muscular weakness. Duchenne muscular dystrophy (DMD) is the most common X-linked disorder muscular dystrophy in children, presenting in early childhood and characterized by proximal muscle weakness and calf hypertrophy in affected boys. There is usually delay in motor development and eventually wheelchair confinement followed by premature death from cardiac or respiratory complications. Treatment modalities such as corticosteroid therapy and use of intermittent positive pressure ventilation have provided improvements in function, ambulation, quality of life, and life expectancy, although novel therapies still aim to provide a cure for this devastating disorder. Here, we present a case of DMD in an 18-years old male with remarkable severe respiratory and skeletal muscle involvement.

Keywords: Acute respiratory failure, Duchenne Muscular Dystrophy, Calf hypertrophy, Creatine kinase, Gower's sign, Muscle weakness.

Introduction:

Muscular dystrophies are a group of progressive, genetically determined, primary degenerative myopathies characterized by different degrees and distributions of muscle wasting and weakness.[1] Duchenne muscular dystrophy (DMD) is the most common and severe form of muscular dystrophy, beginning at 3–5 years of age and characterized by proximal muscle weakness and calf hypertrophy in affected boys.[2, 3] DMD has a very high mutation rate with distinctive and relentless clinical presentation. Some carriers also have symptoms. New mutations are common in DMD; this means that female relatives of a child with DMD are not necessarily carriers of the gene. Patients usually become wheelchair-bound by the age of 12 and die in their late teens to early twenties.[4, 5] Respiratory muscle involvement is common among Duchenne muscular dystrophy and myotonic dystrophy. In others such as Becker, limb girdle, and facioscapulo-humoral dystrophies, respiratory muscle involvement is infrequent and generally occurs in the more severe cases. Respiratory failure is an important contributor to morbidity and mortality in progressive neuromuscular disorders,[6] and these patients often experience acute respiratory failure requiring endotracheal intubation or tracheostomy and mechanical ventilation.[7]

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Case Presentation:

In December 2017, an 18 years' young male was admitted to our institution Bangladesh Medical College Hospital because of dyspnea on exertion, weakness, and easy fatigability of the muscles of the shoulders and pelvic girdles. His recent medical history included 2 admissions of hypercapnic acute respiratory failure. His parents gave medical history of repeated falls, fatigue, muscle weakness, difficulty in standing, walking, getting up from sitting position and inability to climb stairs. There was no history of muscle pain and cranial nerve involvement. His intelligent quotient was claimed to be in the normal range. Patient's family history revealed that one of his maternal uncle and a cousin on his maternal side died of the same illness at a young age.

Physical examination found symmetrical atrophy in the shoulder and pelvic girdle muscles, hyperlordosis, marked kyphosis, proximal weakness, calf hypertrophy and hamstring muscle contracture. He was hindered when raising his arms, running and climbing stairs. Gower's sign was positive. Manual muscle testing revealed 3/5 and 2/5 in the left and right triceps respectively and 2/5 and 3/5 in the left and right iliopsoas, respectively. His nutritional status, as assessed by body mass index (kg/m^2), was 21. In supine position his abdominal wall retracted paradoxically during the inspiratory phase.

The patient was subjected to radiological and laboratory investigations. Serological analysis showed creatine kinase (CK) level to be elevated to 5674 U/L, serum aldolase level 350 U/L, lactate dehydrogenase to 456 µg/dl, alanine transaminase level 120 U/L, C-reactive

protein 3.5 mg/dl and fibrinogen level 444 mg/dl. His CK and serum aldolase levels are 50 fold higher than normal. Elevated creatine kinase/aldolase levels strongly suggested a case for myopathy. Serum aldolase test indicates muscle degeneration and a definite aid in distinguishing primary myopathy from secondary or neurogenic muscle atrophy.

A previous investigation performed in another hospital showed throughout low amplitude compound muscle action potentials in motor nerve conduction studies, with normal conduction velocities. Muscle biopsy showed muscular fibers ranging from 9 μm to 43 μm in diameter, and was conclusive for a non-evolutive myopathy. Electromyography showed a myopathic pattern. A radiograph showed elevation of both diaphragms during maximal inspiration. There were no abnormal electrocardiographic findings. Spirometry was performed according to the American Thoracic Society recommendations. Respiratory muscle strength was assessed via multiple measurements of the maximum inspiratory pressure and maximum expiratory pressure at the mouth, generated against an occluded airway. PaO2, PaCO2, and pH were measured with the patient sitting and breathing room air for at least 30 min. Static lung volumes were determined via the helium dilution method. His vital capacity was 2,140 mL (73% of predicted) in the sitting position, and decreased to1,850mL (64% of predicted) in the supine position. A nocturnal polysomnogram showed an apnea/hypopnea index of 26.2 episodes per hour (with apnea defined as absence of air flow for > 10 s, and hypopnea defined as a > 30% decrease in air flow associated with a > 4% decrease in SpO2).

To relieve airway obstruction, domiciliary inhaled bronchodilator was integrated with a 3 week course of oral prednisone (25 mg/d), after which we instituted nocturnal intermittent noninvasive ventilation (NIV) to prevent nocturnal hypoventilation and desaturations. The prednisone substantially improved both spirometry and blood gases. Nocturnal oxyhemoglobin saturation was monitored with a pulse oximeter with a finger probe. On NIV his mean nocturnal SpO2 increased from 91% to 95%, and the percent of sleep time with SpO2 < 90% fell from 24% to 2%. After 2 weeks of nocturnal NIV his blood gases further improved, to pH 7.42, PaCO2 45 mm Hg, and PaO2 86 mm Hg. At a follow-up 6 weeks later he had satisfactory ventilation, his clinical condition was stable, he reported no further episodes of acute respiratory failure.

The patient was counseled to undergo daily physiotherapy, steroid therapy, and regular assessment for progressive muscle and cardiac/respiratory damage.

Discussion:

DMD is the most common muscle dystrophy in the world, caused by mutations in dystrophin gene as a result of which the body is unable to synthesize the protein dystrophin required for muscle contraction. Every time the muscle contracts, muscle damage occurs which is repaired but with deficient protein resulting in repaired muscle which is also a damaged one. This continuous succession of damage and repair and eventually replacement of muscle with fibro fatty tissue is responsible for the clinical signs of progressive muscle wasting and degeneration that is usually evident by 3–4 years.[2,5]

DMD is caused by mutations in the DMD gene encoding a protein called dystrophin, which localizes to the cytoplasm of the sarcolemma of the skeletal muscle, forming one component of a large glycoprotein complex (dystrophin-associated glycoprotein complex). Dystrophin consists of an N-terminal actin-binding domain, 24 spectrin-like repeat units interspersed by four hinge regions, followed by a cysteine-rich domain and a C-terminal domain. The cysteine-rich domain binds to laminin-2 through alpha and beta-dystroglycan, and therefore acts as mechanical link between actin in the cytoskeleton and the extracellular matrix. The DMD gene contains 79 exons but accounts for only 0.6% of the gene; the rest made of large introns. The large size of the DMD gene makes it susceptible to mutations, leading to loss of function of dystrophin, resulting in a prematurely truncated and unstable dystrophin protein. The majority of mutations are intragenic deletions, which account for 65–72% of all DMD patients. The precise mechanism of how dystrophin deficiency leads to degeneration of muscle fibers remains unclear. The absence of dystrophin at the plasma membrane leads to delocalization of dystrophin-associated proteins from the membrane, disruption of the cytoskeleton with resultant membrane instability and increased susceptibility to mechanical stress. In addition, altered membrane permeability and abnormal calcium homeostasis are thought to play a role, with increased cytosolic calcium concentration leading to activation of proteases such as calpains.[4]

Pulmonary involvement is frequent in neuromuscular diseases. The severity of pulmonary impairment and the disease stage at which it develops differ in the different types of neuromuscular disease.[7]

In a study that included 60 patients with limb-girdle muscular dystrophies, there were only minimal changes in respiratory function and no evidence of respiratory hypoventilation, even in the patients who were not more ambulatory.[8] In several other patients with limb girdle muscular dystrophies, mild pulmonary abnormalities were reported, but there were substantial blood-gas abnormalities in only 3 patients, who were already wheelchair-bound.[6] Differently from the above reports, our patient had severe diaphragmatic impairment, respiratory hypoventilation, and substantial blood-gas abnormalities while he was still ambulatory. To the best of our knowledge, among the limb girdle muscular dystrophies, no respiratory dysfunction in dysferlinopathies have been reported.

Our patient had severe weakness of both skeletal (shoulder and pelvic girdles) and respiratory muscles, but no treatment, nutritional state, or electrolyte abnormalities explained that muscle impairment. Patients with respiratory muscle weakness are at risk of respiratory failure during sleep, especially rapid-eye movement sleep.[9] In patients with bilateral diaphragm paralysis the intercostal and accessory muscles become the principal muscles of inspiration. Inhibition of those muscles' activities during rapid-eye-movement sleep causes severe hypoventilation, apneas, and hypoxia.[9] Later, nocturnal hypoxemia and hypercapnia may persist even during the daytime.

Our patient's arterial blood gases substantially improved after relieving the airway obstruction, but they normalized only after NIV treatment. In previous reports, satisfactory ventilation was maintained for more than 2 years, with NIV administered only during sleep.

Current management of DMD involves physiotherapy and corticosteroid therapy which delays loss of ambulation 1–3 years but does not cure the disease which was provided in our case. However, corticosteroids are associated with significant side effects, including weight gain, decreased bone mineralization, Cushing syndrome, and behavioral disturbances. Alternate regimens have been tried, although the efficacy of these regimens in comparison to daily dosing is incompletely studied.[10,11] A growing number of reports suggest that treatment before the age of 5 years is especially beneficial, though the data to support early use are limited.[10] Recent treatment modalities include gene therapy and stem cell therapy which appear very promising and suggest that an up regulation of dystrophin-like protein has beneficial effects. Prenatal counseling and genetic tests like multiplex ligation-dependent probe amplification are being used to offer hope in this progressive and eventually fatal muscular dystrophy to prolong and improve the quality of patient's life.[12,13]

Conclusion:

Current management of DMD involves physiotherapy and corticosteroid therapy which delays progression but does not cure the disease. Prenatal counseling and various other genetic modalities are being tested to offer hope in this progressive and eventually fatal dystrophy to prolong and improve the quality of life in such patients.

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Case Report

Isoniazid Resistant Tubercular Lymphadenitis with Superimposed *Acinetobacter* Infection: a Clinical Case Report

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Abstract

A 17-years old girl hospitalized with high fever, gradual weight loss and headache for 2 months. After 10 days of admission, multiple lymph nodes were palpable in anterior chain of right cervical region. Excisional lymph node biopsy showed frank pus and pus was found AFB (++). Tissue for Gene Xpert detected MTB and RIF resistance was not detected. Tissue from lymph node was also sent for AFB culture and sensitivity. The patient was diagnosed as Tubercular lymphadenitis and given treatment with 6 months' regimen of ATT (4 drugs with HRZE for 2 months, then 2 drugs with HR for another 4 months) initially. The temperature decreased after commencing anti-tubercular therapy, but after 1½ months she again developed high grade fever and re-admitted to hospital. Investigation of blood culture revealed growth of *Acinetobacter spp* with Carbapenem sensitivity and TB culture report showed growth of MTB with INH resistance. Based on new evidence, she was diagnosed as a case of INH-resistant Tubercular lymphadenitis with superimposed *Acinetobacter* infection. Then, her ATT was revised with 9 months' therapy with Rifampicin, Pyrazinamide and Ethambutol with added Levofloxacin. In addition, she was treated with injectable Meropenem and oral Ciprofloxacin. On completion of therapy, patient showed good clinical response. This case highlights the observation that even extra-pulmonary primary mono-drug-resistant tuberculosis can be successfully treated with revised dose of currently available ATT.

Keywords: Tubercular lymphadenitis, Isoniazid-resistant TB (Hr-TB), Gene Xpert detected MTB, *Acinetobacter* infection.

Introduction:

The most common presentation of extra-pulmonary TB in both HIV-seronegative and HIV-infected patients, lymph node disease is particularly frequent among HIV-infected patients and among children.[1,2] Once caused mainly by *Mycobacterium bovis,* tuberculous lymphadenitis today is largely due to *Mycobacterium tuberculosis*. Lymph node TB presents as painless swelling of the lymph nodes, most commonly affecting posterior cervical and supraclavicular sites (a condition historically referred to as scrofula). Lymph nodes are usually discrete in early disease but develop into a matted nontender mass over time and may result in a fistulous tract draining caseous material but characteristically there is no erythema over the skin (cold abscess formation).[2,3] Associated pulmonary disease is present in fewer than 50% of cases, and systemic symptoms are uncommon except in HIV-infected patients. The diagnosis is established by fine-needle aspiration biopsy (with a yield of up to 80%) or surgical excision biopsy.[1,2,3] Bacteriological confirmations are achieved in the vast majority of cases, granulomatous lesions with or without visible AFBs are typically seen, and cultures are positive in 70-80% of cases.

Compounding the challenges of an already lengthy and complicated treatment course, the World Health Organization (WHO) in 2017 reported an estimated 6,00,000 incident cases of MDR-TB in 2016. Isoniazid (H) is one of the most important first-line medicines for the treatment of active tuberculosis (TB) and latent TB infection (LTBI), with high bactericidal activity and a good safety profile. The emergence of TB strains resistant to Isoniazid threatens to reduce the effectiveness of TB treatment. About 8% of TB patients worldwide are estimated to have Rifampicin-susceptible, Isoniazid-resistant TB (Hr-TB).[3]

In 2018, following an assessment of available evidence for the treatment of Hr-TB, including the evaluation of results from an analysis of individual patient data (IPD), and advice from members of the Guideline Development Group (GDG), WHO made the following recommendations[3]:

In patients with confirmed Rifampicin-susceptible and Isoniazid-resistant tuberculosis, treatment with Rifampicin, Ethambutol, Pyrazinamide and Levofloxacin (Lfx) is recommended for a duration of 6 months.

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As there is no approved REZ FDC available, the 4-drug "HREZ" fixed-dose combination (FDC) with I*soniazid (H), Rifampicin (R), Ethambutol (E) and Pyrazinamide (Z)* – may be used to limit the need for using single drugs. Before starting the treatment, drug susceptibility to fluoroquinolones should preferably be confirmed.

In patients with confirmed Rifampicin-susceptible and Isoniazid-resistant tuberculosis, it is not recommended to add Streptomycin or other injectable agents to the treatment regimen.

Acinetobacter species are oxidase-negative, non-fermenting, short, gram-negative bacilli. The bacteria grow well at 37° C in aerobic condition. *Acinetobacter* species can be found in water, in soil, and on vegetables. *Acinetobacter* is a component of human skin flora and is sometimes identified as a contaminant in blood samples collected for culture. The vast majority of infections occur in hospitalized patients and other patients with significant health-care contact. Outbreaks of Carbapenem-resistant *A. baumannii* are particularly problematic.[1,4]

A.baumannii colonizes patients exposed to heavily contaminated hospital environments or the hands of health care workers. Emerging data suggest that the organism can be found in the air in rooms of patients infected with *Acinetobacter*. Colonization of the upper airways in mechanically ventilated patients may lead to nosocomial pneumonia. Colonization of the skin may lead to central line-associated bloodstream infection, catheter-associated urinary tract infection (UTI), wound infection, or postneurosurgical meningitis.[1,4]

Here, we present a case report of patient with Isoniazid resistant tubercular lymphadenitis, who was on standard anti-TB chemotherapy, infected with superimposed *Acinetobacter spp* after 1½ months of starting ATT.

Case Presentation:

A 17-years old girl presented on May 30, 2017, with complaints of fever for 2 months, loss of weight of 5-7 kgs in last 2 months and headache for same duration. Fever was continuous in nature and was associated with chills and rigor. Maximum temperature was recorded about 102°-103° F. There was no productive cough, nausea or vomiting, altered bowel habit, joint pain or abdominal pain. On examination, she was ill-looking, body temperature was 103° F, pulse was 104 beats/min, BP was 100/70 mm of Hg, respiratory rate was 16 breaths/min. Neck stiffness and Kernig's sign were absent. Chest was clear on auscultation and no organomegaly was found on abdominal palpation. Initially, lymph nodes were not palpable, but after 10 days of admission, multiple lymph nodes were palpable in anterior chain of right cervical region. All other systemic examinations revealed no abnormality.

On investigation, Haemoglobin was 08.72 g/dl, MCV 57 fl, WBC count 10,000/mm^3, platelet 3,50,000/mm^3, ESR 40 mm in 1st hour. ALT 65 U/L, S. Bilirubin 0.38 mg/dl, Alkaline phosphatase 218 U/L, Prothrombin time 18.50 sec, LDH 570 U/L, S. Iron 23 µgm/dl, TIBC 36 µgm/dl, Transferrin Saturation 6.55%, Chest X-ray P/A view-suggestive of right paratracheal mass, blood C/S- no growth, Urine C/S- no growth. Other relevant investigations that might exclude the cause of fever was done, such as- ICT for Malaria, ICT for Kala-azar, ANA, HIV (1+2), but all reports were negative. USG of whole abdomen was done which revealed biliary sludge, prominent ovaries with tiny cyst. On the 10th of June 2017, FNAC of lymph node from anterior chain of right cervical region was done which revealed suppurative lymphadenitis. On the 12th of June, excisional lymph node biopsy showed frank pus. Pus for Gram Stain showed no organism and C/S showed no growth. AFB stain showed Acid Fast Bacilli (++). Tissue from abscess wall of lymph node for histopathology was sent that revealed inflammatory tissue, compatible with abscess and Gene Xpert for detection of MTB was also sent which detected MTB, RIF resistant was absent. At the same time, a piece of tissue from lymph node for TB culture was sent. As because, there was confirmed microbiologic evidence of tuberculosis infection, treatment with fixed dose combination anti-TB chemotherapy was commenced with regular dressing of the operative site.

Iron deficiency anaemia was corrected with 500 mg of inj. Ferric Carboxymaltose. Temperature was gradually coming down but was not remaining in the base line. Temperature was fluctuating to 100°-101°F after a couple of weeks of starting ATT. But the patient was feeling better and her appetite improved. She was discharged from the hospital after 19 days with treatment of ATT for 6 months' regimen (4 drugs with HRZE for 2 months, then 2 drugs with HR for another 4 months) with oral iron supplementation. She was also advised for doing regular dressing at Surgery out-patient department (OPD).

After 1½ months of discharge, she developed high grade fever, recorded about 103°F-105°F, continuous in nature and also had severe menorrhagia as well as irregular per vaginal bleeding. She was hospitalized for second time on 21st July 2017. On examination, she was running fever; maximum temperature was recorded about 105° F, mildly anaemic, nonecteric but very toxic looking. Other systemic examination was unremarkable. Lab reports showed high ESR (102 mm in first hour), total WBC count was 10,000/mm^3, neutrophil 70%, lymphocyte 20%, monocyte 08%, haemoglobin was 08.90 gm/dl, MCV 75.80 fl, platelets 3,00,000 mm^3, Urine C/S- no growth, but 2 samples of Blood C/S revealed growth of *Acinetobacter spp* with Carbapenem sensitivity. Echo-color Doppler was done to exclude bacterial endocarditis. We had also done CT scan of neck and chest to exclude lymphoma.

Along with supportive care and ATT, treatment was started with injectable Meropenem and oral Ciprofloxacin. She also received 1 unit of packed cell transfusion and 500 mg

Discussion:

Globally drug resistant TB has become a major health problem since early 1990 and for failure of anti-TB treatment, drug resistance must be considered.[2,5,6] The four different categories of drug resistant TB are mono-resistance, poly-resistance, multidrug-resistance TB (MDR-TB) and extensive drug-resistance TB (XDR-TB). Extra-pulmonary TB among the immunocompetent individuals constitutes about 15% to 20% of all cases of TB,[6] and, in HIV patients it accounts for more than 50%.[7] In majority of the patients it occurs as a result of lymphohematogeneous dissemination during primary infection.[8,9] To our best of knowledge there is only few literatures available on the prevalence of extra-pulmonary drug resistant TB.[10] Like pulmonary, extra-pulmonary TB should also be confirmed microbiologically and if resistance is suspected, it should be studied for culture and susceptibility. High failure rates are usually seen in the treatment of drug resistant TB compared with drug susceptible ones.[2,11]

Active pulmonary TB occurs infrequently in immunocompetent patients with TB lymphadenitis. However, all patients presenting with adenopathy should have a chest radiograph performed as part of the initial evaluation.[12,13] Fifteen percent of patients on a study conducted by University of Saskatchewan of Canada had concurrent active pulmonary TB, but 41% of patients with abnormal films suggestive of TB had culture-positive pulmonary TB.[13] Here we have done chest X-ray of this patient after getting admission that showed paratracheal and right hilar lymphadenopathy. But there was no evidence of active pulmonary TB.

In case of our patient we have done FNAC initially which revealed suppurative lymphadenitis. As because, there was no conclusive explanation from the FNAC report, we did excisional lymph node biopsy and collected 3 pieces of tissue. Among the pieces, one piece in formalin was sent for histopathology that revealed inflammatory tissue, compatible with abscess; one piece in normal saline for PCR (GeneXpert) showed MTB detected, RIF resistance not detected and another final piece in other container containing normal saline also for TB culture which reported as growth of *Mycobacterium tuberculosis*, resistant to Isoniazid.[14,15] It is noteworthy that patients with granulomas on initial FNA (sample with necrotizing granulomas), subsequently may be diagnosed with lymphoma.

In this context, we also tried to exclude lymphoma because patient was not responding well to ATT after1½ months; fever was not coming down to base line, occasionally fluctuating to 100°-101°F. Here we did CT scan of neck and findings were consistent with multiple cervical lymphadenopathy at level III, IV, V and VI on right side and level IV on left side- most likely lymphoma. CT scan of chest demonstrated multiple mediastinal lymphadenopathy-possibly lymphoma. Excisional lymph node biopsy for histopathology was also done that excluded lymphoma.[11,16,17]

The diagnosis of TB in the absence of a positive culture requires a combination of epidemiologic and histopathologic criteria as well as a trial of anti-tuberculous medication. Granulomas or inflammation and necrosis with a positive AFB stain patient was highly suggestive of TB.[18,19] In patients with pathology consistent with TB and negative stains and cultures, TB was still likely and patients were considered to have TB based on their clinical response to therapy. We had done the same thing in case of our patient. We started ATT before getting TB culture report that was available after 6 weeks of starting ATT.

Apart from multidrug-resistant TB (MDR-TB), about 9.5% (8.1% in new and 14.0% in previously treated) of TB cases worldwide are estimated to have Isoniazid-resistant TB without MDR-TB (HR-TB). MDR/RR-TB is associated with an increased risk of treatment failure in patients who receive first-line regimens.[2,5,6]

Empirical treatment of Hr-TB is not generally advised. In cases where Hr-TB diagnosis is strongly presumed (e.g. close contacts of Hr-TB cases with active TB but without laboratory confirmation of Hr-TB), (H)REZ-Lfx may be introduced pending laboratory confirmation of Isoniazid resistance, so long as Rifampicin resistance has been reliably excluded**.** Drug-susceptibility testing results eventually indicate susceptibility to Isoniazid, Levofloxacin is stopped and the patient completes a 2HRZE/4HR regimen. For other patients, in whom Hr-TB is detected after the start of treatment with the 2HRZE/4HR regimen, the (H)REZ component drugs are continued (or Pyrazinamide and Ethambutol are re-introduced) and Levofloxacin added once Rifampicin resistance has been excluded.

The (H)REZ-Lfx regimen is given for as long as it is necessary for the patient to receive Levofloxacin for six months. Thus, in cases where the diagnosis of Hr-TB is made after first-line TB treatment has already been initiated, the patient may receive more than six months of (H)REZ by the end of treatment. When the confirmation of Isoniazid resistance arrives late into treatment with a 2HRZE/4HR regimen (e.g. 5 months after start during the continuation phase), the clinician would need to decide, based on an assessment of patient condition and laboratory tests, whether a 6 months course of (H)REZ-Lfx needs to be started at that point or not.

Patient with *Acinetobacter* infection may present with pneumonia (usually hospital acquired), may acquire the infection within days of arrival in an ICU, usually ventilator- associated pneumonia. Community acquired pneumonia due to *A. baumannii* may occur in tropical regions of Australia and Asia.[1] Sources of bloodstream infection are typically a central line or underlying pneumonia, UTI, or wound infection.[1,4] Meningitis, urinary tract infection and other clinical manifestations like *Acinetobacter* prosthetic-valve endocarditis and endophthalmitis/ keratitis may also present.

Treatment is hampered by the remarkable ability of *A.baumannii* to up-regulate or acquires antibiotic resistance determinants. The most prominent example is that of β-lactamases, including those capable of inactivating Carbapenems, Cephalosporins, and Penicillins.[1] These enzymes, which include the OXA type β-lactamases (e,g; OXA-23), the metallo-β-lactamases (e,g; NDM), and rarely KPC-type carbapenemases, are typically resistant to currently available β-lactamase inhibitors such as Clavulanate or Azobactam.Carbapenems (Imipenem, Meropenem, and Doripenem but not Ertapenem) have long been thought of as the agents of choice for serious *A. baumannii* infections. Sulbactam may be an alternative to Carbapenems.

Conclusion:

Globally, Hr-TB is more prevalent than MDR-TB. Efforts need to be made for universal testing of both Isoniazid and Rifampicin at the start of TB treatment and to ensure the careful selection of patients eligible for the (H)RZE-Lfx regimen. Physicians must ensure completion of treatment for patients taking Isoniazid and other first line drugs as part of their TB, latent TB infection or extra-pulmonary TB therapy. Decisions on treatment of infections with *Acinetobacter* should be made on a case-by-case basis by a healthcare provider. *Acinetobacter spp.* infection typically occurs in ill patients and can either cause or contribute to death in these patients. Careful attention to infection control procedures, such as hand hygiene and environmental cleaning, can reduce the risk of transmission.

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Case Report

The Mermaid Syndrome: an Extreme Example of the Caudal Regression Syndrome

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Abstract

Sirenomelia or the mermaid syndrome, is an extreme example of the caudal regression syndrome**.** A case of sirenomelic baby of 34 weeks' gestation reported to a tertiary care hospital of Dhaka city. The baby was alive at birth and was having fused lower extremities. Anal opening & genitalia couldn't have been recognized due to fusion. But there was no other gross congenital anomaly. Patient's party didn't agree to do further investigations & treatment of the baby as baby was congenitally abnormal. Also patient's party didn't agree to take any measures such as NICU support for the baby and ultimately baby died in the early perinatal period.

Keywords: Sirenomelia, Mermaid baby, Caudal regression syndrome, Potters facies.

Introduction:

An extreme example of the caudal regression syndrome is Sirenomelia, or the mermaid syndrome. Symelia, the fusion of lower limbs, which constitutes the characteristic element of malformation at the very first look, is not the only abnormality in these cases.[1] It is invariably associated with lower limb anomalies in the form of flexion, external rotation, symelia and often atrophy, vertebral anomalies (epistasis or sacral agenesis), anorectal agenesis, urinary tract anomalies in the form of renal, ureteral, vesical and urethral agenesis, Woffian or Mullerian duct agenesis. Gonads are usually spared. Because of the resultant oligohydramnios, these infants most often have Potter's facies and pulmonary hypoplasia. Another important feature is the presence of a single large artery, arising high in the abdominal cavity that assumes the function of the umbilical arteries and diverts nutrients from the caudal end of the embryo to distal to the level of its origin. We wish to report an additional care of sirenomelia. Cases of sirenomelia surviving beyond the perinatal period have been reported by Savader et al[2] for the first time in 1989 and later by Murphy et al[3] in 1992. However, in the majority of cases the visceral anomalies are constant and uniform, incompatible with life.

Case Presentation:

A 24-year-old woman of 2nd gravida with 34 weeks' pregnancy with labour pain with breech presentation and foetal distress delivered a single 2 kg weighted baby by caesarian section on 10th October, 2017 in Jomila General Hospital, Tongi, Dhaka. Mother was from a poor socioeconomic group, had irregular antenatal check up (only two USG at 26 weeks & 32 weeks). The pregnancy was uneventful up to 26 weeks. Thereafter she was diagnosed as a case of severe oligohydramnios due to premature ruptured membrane. Since then pregnancy continued without improvement of liquor volume. There was no family history of congenital anomalies or stillbirths. Attempts were made for resuscitation & the baby cried after resuscitation. There was fusion of lower extremities from thighs to feet. There were 10 toes in two legs. There was no anal opening. Genitalia also couldn't been recognized due to fusion. Both the upper limbs & facial features were normal (Fig-1). We suggested to keep the baby in ICU for necessary supports and management. But baby's guardian refused to proceed for further treatment as they apprehended that the baby with so much congenital malformation might not survive for long time. Ultimately baby died in the early perinatal period.

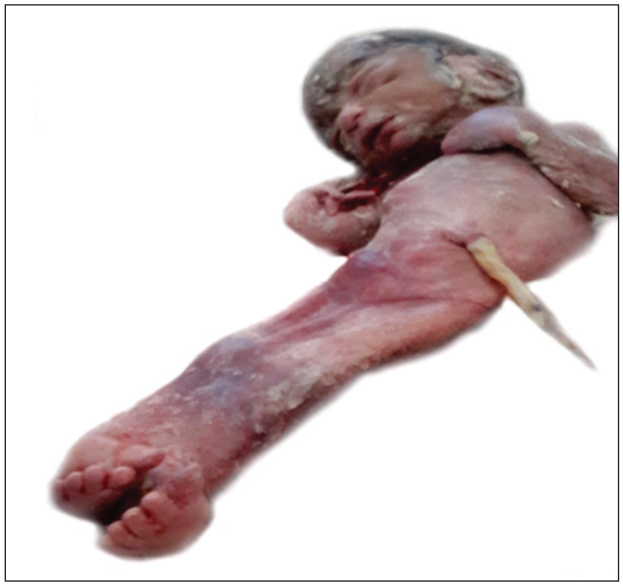

Fig 1: Mermaid Baby

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Discussion:

Various theories have been postulated to explain the etiology of sirenomelia. The first is a "pressure theory"[4] which proposed that some intrauterine force, probably amniotic, acts on the tail of embryo. This leads to defective development of the caudal region including the allantoic structures. Ballantyne[5] supports this view, noting that the allantois fails to develop completely and that the placenta is vitelline in origin. Bolk[6] in 1899 proposed a theory of "primary failure". He suggested that sirenomelia resulted from failure of development of the caudal somites leading to greater or lesser hind part deficiency. Weigert[7] coined the theory of "nutritional deficiency". He observed the vitelline nature of the umbilical artery and hypoplasia of the remaining vasculature. In 1961 Duhamel[8] proposed the theory of "caudal regression" resulting in a spectrum of malformation including anal imperforation and the mermaid syndrome.

In 1970 Davis et al[9] suggested that sirenomelia results from injury to the caudal mesoderm between 28-32 days of foetal development. The primitive streak at the lumbar and sacral areas undergoes a developmental arrest allowing the halves of the hind limb buds to move medially and dorsally fusing along their postaxial surfaces. Midline structures i.e. cloacal and urogenital derivatives are destroyed. This arrest of primitive streak due to unknown etiology initiates formation of a second primitive streak, giving rise to a second normal embryo. This thus explains the association of sirenomelia in monozygotic twins. In 1976 Smith[10] supported his theory.

In 1980, Gardner and Brener[11] proposed a theory of "neural tube over distension". According to them neural tube over distension in the caudal area may lead to a roof plate expansion of the tube leading to lateral rotation of mesoderm by 180°. This results in fusion of the lower limb buds, closing off the midline primitive gut and urethra. Stevenson et al[12] in 1986 postulated that a "vascular steal" accounts for caudal regression. He dissected abdominal vasculature in 11 cases of sirenomelia and observed that in all cases there was a single umbilical artery that entered aorta in the upper abdomen. The vessels distal to this aberrant umbilical artery were under developed and malformed resulting in diversion of blood flow and nutrients from all caudal structures of embryo to the placenta. This decreased delivery of nutrients caused subsequent developmental arrest or malformation. Distal to this point the aorta bifurcated into two hypoplastic iliac vessels. The inferior mesenteric artery was absent. This may explain the atresia. Stevenson's theory, fails to explain the non-caudal anomalies present in conjunction with sirenomelia. The abnormal abdominal vasculature in sirenomelia could either be a cause or an effect of sirenomelia. The pressure theory of Dareste[4] and caudal mass deficiency theory of Bolk[6] provides the two alternative explanations in which the vascular mal-developments are viewed as a consequence of sirenomelia rather than the cause. No teratogenic agent has been postulated in the etiology of sirenomelia until now. Thus the occurrence of sirenomelia is a random event without any proven etiology.

Conclusion:

Sirenomelia, or the mermaid syndrome is an extreme example of the caudal regression syndrome**.** Various theories have been postulated to explain the etiology of sirenomelia but actual cause is not established till now**.** No teratogenic agent has been postulated in the etiology of sirenomelia**.** Thus the occurrence of sirenomelia is a random event without any proven etiology**.** But we should counsel patient about the possibility of sirenomelia if presents with preterm premature rupture of membrane with persistent oligohydramnios because in my case patient gives history of preterm premature rupture of membrane with oligohydramnios**.**

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College News

College Events:

- International Mother Language day was observed in Bangladesh Medical College & Hospital on 21st February, 2018. Teachers, doctors, students and staffs actively participated in that event and paid tributes to the martyred of Bengali language movement.

- A discussion meeting, organized by Bangladesh Medical College and Hospital was held on 24th March, 2018 at BMC lecture hall on account of celebration of the historical recognition of Bangladesh as Developing Country, on the 17th March, 2018 on 98th birthday of Father of Nation- Bangabandhu Sheikh Mujibur Rahman. Among the presenters were- Prof. Sharmeen Yasmeen, Prof (CC). Raihana Begum and Dr.Yasmin Aktar Khan. Honorable EC members of BMSRI and senior professors were among the discussants. Teachers, doctors, staffs and students attended this meeting.

- 47th National Independence day was observed in Bangladesh Medical College and Hospital and Bangladesh Dental College on 26th March,2018. Teachers, doctors, students and staffs participated spontaneously in that event.

- A discussion meeting was held on 28th March, 2018 on account of 47th National Independence Day in Bangladesh Medical College and Hospital. In the meeting Dr. Md. Abdur Razzaq MP, Chairman, EC, BMSRI was present as Chief Guest and Dr. Mustafa Jalal Mohiuddin, President, BMA & Ex-Member of Parliament was present as Special Guest. Engr. Md. Siddique Ullah, Co-chairman, Prof. Selima Khatun, honorable EC Member, Maj. General (Retd.) Md. Rafiqul Islam, Hony. Secretary, Engr. G M Jainal Abedin Bhuiya, Treasurer and Dr.AH Rezwanul Kabir, Co-Hony.Secretary, EC, BMSRI were the Guest of Honor. Prof.Rezaur Rahman Talukdar, President of BMC & H and BDC Doctors and Teachers Welfare Association chaired the meeting.

- World Nurse day was observed on 12th May, 2018 in Bangladesh Medical College Hospital (BMCH). Dr. Md. Abdur Razzaq MP, Chairman, EC, BMSRI graced the occasion as Chief Guest. Officials of EC of BMSRI were present as Special Guests. The event was presided over by the Director of BMCH Birig. General (Retd.) Dr. Md. Abdus Sabur Miah.

- Induction course of new intern doctors of BM-27 and others was held on 8th July, 2018. Dr. Md. Abdur Razzaq MP, Chairman, EC, BMSRI graced the occasion as Chief Guest. Officials of EC of BMSRI were present as Special Guests. The event was presided over by the Director of BMCH Brig. General (Retd.) Dr. Md. Abdus Sabur Miah.

Seminar in BMC:

- Society of Laparoscopic Surgeons of Bangladesh (SLSB) organized a scientific seminar and special lecture on "Metabolic Surgery: a new horizon of treatment by laparoscopic surgery for diabetes mellitus" on 6th March, 2018 at BMC. The speaker was one of the world's pioneer laparoscopic surgeons Prof. Dr. Peter Goh of Singapore.

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

- Dr. Yasmin Aktar Khan, Consultant of Endocrinology, Bangladesh Medical College Hospital (BMCH) attended the EMSS-MAYO advance course of Endocrinology from 1-4 February, 2018 in Singapore.

- Dr. Md. Amir Hossain, Associate Professor of Cardiology, BMC, attended International Conference in Cardiology held in Thailand from 7-9 March, 2018.

- Prof.Sharmeen Yasmeen, Head of the dept. of Community Medicine, BMC attended the International Conference on Epidemiologic Research organized by Nepal Public Health Foundation held in Kathmandu, Nepal from 11-15 March, 2018. She was one of the main speakers of the conference and presented paper on Non-communicable diseases.

- Dr. Syed Khalid Hasan, Associate Professor of Surgery, BMC, attended Apollo International Colorectal Symposium, 2018 from 22- 25 March,2018 in Chennai, India.

- Prof. Dr. Md. Mizanur Rahman, Professor & Head of the Department of Ophthalmology, BMC, attended the Asia Pacific Glaucoma Congress, 2018 from 13-15 April, 2018 in South Korea.

- Dr. Raihana Begum, Professor (CC), Dept. of Community Medicine, BMC, attended a meeting on the Global Taskforce on Cholera held on 13-14 June, 2018 in Annecy, France.

- Prof. Md. Fazlul Kadir, Professor of Medicine, BMC, attended the International conference of American Diabetic Association held in Florida, USA from 22-26 June, 2018.

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