Osmani Medical Teachers Association Journal

Volume 15: Number 2
July 2016

Original Articles

Teachers Association, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh

Osmani Medical Teachers Association Journal

Teachers Association, Sylhet MAG Osmani Medical College

Executive Committee

President
Dr. Jamil Ahmed

Vice Presidents
Prof. Manajjir Ali
Prof. Zakia Sultana

General Secretary
Dr. Dilip Kumar Bhowmik

Treasurer
Dr. Ishtiaque Ul-Fattah

Joint Secretary
Dr. Md. Enayet Hossain

Organizing Secretary
Dr. Shankar Kumar Roy

Members
Dr. Lutfe Elahi Faruqui
Dr. N.K Paul
Dr. Subir Kumar Das
Dr. Probhat Ranjan Dey
Dr. Begum Lufun Nahar
Dr. Md. Abdus Samad Azad
Dr. Md. Shamsur Rahman
Dr. Mojibul Haque

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Role of cardiotocography in High Risk Pregnancy- Study of 100 Cases in a Tertiary Hospital

Iffana Azam[1], M A Sabur[2], Sayed Miftah Uddin[3], Shahana Ferdous Chowdhury[4], Dipu Das[5]

Abstract

The antenatal cardiotocography (CTG) is essentially a screening test for fetal well-being. When an antenatal CTG is performed and interpreted as abnormal, this may result in a range of further actions. These could include further testing, hospital admission, induction of labour or cesarean section. This cross-sectional study was conducted in the Department of the Obstetrics and Gynaecology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet. Hundred high risk pregnancies women who got admitted in Jalalabad Ragib-Rabeya Medical College Hospital were the target population and those fulfill the inclusion and exclusion criteria were considered as study population. Majority (78%) were Lower Uterine Caesarean Section (LUCS) in abnormal CTG that is significantly higher than normal CTG. Meconium stained 38% in abnormal CTG that is significantly higher than normal CTG which was 16%. Placenta big and small 06% and 10% respectively in abnormal CTG that was significant higher than normal CTG that was 02(4%). Incidence of foetal distress (FD) in specific risk factor groups. Common risk factors in abnormal CTG were postdated, Pregnancy Induced Hypertension (PIH), PIH with Intrauterine Growth Retardation (IUGR), IUGR, Premature rupture of membrane (PROM), Bad Obstetrical History (BOH) and Oligohydramanious 78%, 36%, 10%, 14%, 28%, 16% and 12% respectively. Abnormal CTG, 13 had tachycardia among 03 (23.08%) was abnormal outcome, 2 reduced beat to beat variability 1(50%) found abnormal outcome, among 18 deceleration 15(83.3%) were abnormal outcome and 15 non-reactive 5 were abnormal outcome. Sixteen percent patients had birth asphyxia in abnormal CTG and 02% patients in normal CTG ($p<0.05$) that was statistically significant. Patients needed NICU admissions compared to group-A and group-B 36% and 08% respectively of those babies ($p<0.05$) that was statistically significant. In abnormal CTG group 08% were neonatal mortality. The proportion of immediate resuscitation and admission of the babies at neonatal care unit was higher among this group. Perinatal death was also significantly higher in this group. Thus overall outcome was better in normal CTG group. On the other hand the perinatal mortality (PNM) in the present study was significantly lower than the PNM of Bangladesh.

[OMTAJ 2016; 15(2)]

1. Registrar, Department of Obstetrics and Gynaecology, Sylhet Women's Medical College Hospital.
2. Professor, Department of Obstetrics and Gynaecology, Jalalabad Ragib-Rabeya Medical College, Sylhet.
3. Assistant Professor, Department of Anaesthesiology, Sylhet MAG Osmani Medical College.
4. Professor, Department Obstetrics and Gynaecology , Sylhet Women's Medical College.
5. Junior Consultant, Obstetrics and Gynaecology, Al Haramin Hospital, Sylhet .

Introduction

Now a days cardiotocography (CTG) become a popular method for monitoring of fetal wellbeing and it is assisting the obstetrician in making the decision on the mode of delivery to improve perinatal outcome. Fetal Heart Rate (FHR) monitoring plays the most important role in management of labouring patient when incidence of fetal hypoxia and progressive asphyxia increases.[1] Electronic fetal monitoring (EFM) has widely been adopted as fetal heart rate monitoring.[2] Although with intermittent auscultation the baseline FHR can be measured, other features of the fetal heart rate such as baseline variability, accelerations and decelerations are difficult to quantify. Therefore, the use of antepartum and intrapartum Cardiotocography (CTG), a continuous electronic record of the fetal heart rate obtained via an ultrasound transducer placed on the mother's abdomen has increased over the last 15 years. As a

consequence some authors attribute a considerable decrease in the overall perinatal mortality to the use of CTG and today CTG is a first line investigation for ante and intrapartum fetal assessment.[3] Since the 1990s, computerized fetal heart rate analysis systems have been developed to allow the automated evaluation of the CTG through numerical indices with the aim of bringing objectivity and reliability to CTG interpretation.[2] It is also thought that the computerized CTG analysis system may be able to extract more diagnostic information from the fetal heart rate signal than visual analysis alone.[4] The computerized CTG has been investigated in a range of clinical situations including fetal growth restriction, preterm rupture of the membranes, post-term pregnancy and in pregnancies without increased risk factors.[5-8] The antenatal CTG is essentially a screening test for fetal well-being. When an antenatal CTG is performed and interpreted as abnormal, this may result in a range of further actions. These could include further testing, hospital admission, induction of labour or cesarean section. Initial observational studies showed a strong correlation between an abnormal CTG and poor fetal outcome.[9-11] In high-risk pregnancies in particular, 'non-reactive' CTGs were associated with increased morbidity and mortality for the baby.[12-13] This observation has led to the belief that performance of a CTG would allow early identification of fetal heart rate changes associated with hypoxia and allow subsequent early intervention with improved outcomes. However, later studies have demonstrated a lack of specificity and high false positive rates when using the CTG to detect fetal compromise.[14-15] CTG is a non-invasive test to detect the current status of the foetus which is sensitive for detection of adverse foetal outcome. The only disadvantage is that the patient as well as the treating obstetrician may have a false sense of relief where admission CTG is reactive, but the status may change during the process of labour.[16]

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of the Obstetrics and Gynaecology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet. Hundred high risk pregnancies women who got admitted in Jalalabad Ragib-Rabeya Medical College Hospital was the target population and those fulfill the inclusion and exclusion criteria were considered as study population. After admission of the patient in the Department of Obstetrics and Gynaecology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet a rapid diagnostic work up was made from detailed history taking general, physical and obstetric examination. Gestational age was established from the history of last menstrual period in regular menstruating woman or by ultrasound at early pregnancy. High-risk cases were identified from history, antenatal records and clinical examination. Inform written consent was taken from the patients or guardians after full explanation of the purpose of the study. Antepartum cardiotocography (nonstress test) was done in all cases. The patient was placed in semi-recumbent position with slight Ieft lateral tilt to prevent supine hypotensive syndrome. Blood pressure was measured at the initiation of the test and 15 minutes thereafter. CTG was done with cardiotocography instrument. After palpation of abdomen, position of the fetus was confirmed and the position of transducer on the abdomen over the fetal side was selected. The transducer that perceives the fetal heart beat was placed over the uterus after applying the jelly where fetal heart was heard loudest. Uterine contractions were recorded by means of an external tocograph. The women recorded the fetal movements by pressing the event marker. The monitoring was done for 20 minutes and if there was no fetal movement or non reactive tracing in those 20 minutes, it was extended for another 20 minutes after shaking the fetus and changing the position of the woman. In doubtful cases tracing was repeated and time interval dependent on the nature of cases. Tracing was repeated after change of maternal position, maintaining adequate maternal hydration, after correction of maternal hypoxia by O_2 inhalation and stoppage of oxytocin infusion when there was uterine hypertonia. Tracings was taken and studied for interpretation with consideration of temperature, pulse and maternal drug ingestion. In a simple way, CTG was considered normal when the basal heart rate was 110- 150 bpm, beat to beat variability was 5-25 bpm and at least two acceleration of the fetal heart rate was present during a 20 minutes period, each at least 15 beats above the baseline rate and lasting at least 15 seconds. Abnormal CTG was include fetal tachycardia (FHR>150 bpm), fetal bradycardia (FHR <110bpm), reduced or absent beat to beat variability, late declaration and extreme variable decelerations. Pregnancy outcome and neonatal data were obtained from delivery note and neonatal chart review and co-relate with the FHR tracing.

Results

Table shows mean age was 23.45(±3.56) years in abnormal CTG (Group -A) and 24.21(±4.12) years in normal CTG (Group –B). Minimum age was 17 years and maximum age was 40 years (Table-I). Significant association of parity with CTG, in primi para 62% were in group A and 34% were group B, multi para 38% were in group A and 66% were in group B ($p<0.009$) (Table -II) . Term gestational age is significantly higher in abnormal CTG and post term gestation in higher in normal CTG ($p <0.05$) that was statistically significant (Table-III). Pulse and blood sugar were significantly higher in abnormal CTG than normal CTG (Table-IV). Table shows mode of delivery with the results of the admission test and occurrence of foetal distress (FD). Majority (78%) were LUCS in abnormal CTG that is significantly higher than normal CTG. Meconium stained 38% in abnormal CTG that is significantly higher than normal CTG which was 16%. Placenta big and small 06% and 10% respectively in abnormal CTG that was significant higher than normal CTG that was 02(4%) (Table-V). Incidence of foetal distress (FD) in specific risk factor groups. Data are expressed as number (N) and percentage. Common risk factors in abnormal CTG were postdated, PIH, PIH with IUGR, IUGR, PROM, BOH and Oligohydramanious 78%, 36%,10%,14%, 28%,16% and 12% respectively (Table-VI). Table shows abnormal CTG, 13 had tachycardia among 03(23.08%) was abnormal outcome, 2 reduced beat to beat variability 1(50%) found abnormal outcome, among 18 deceleration 15(83.3%) were abnormal outcome and 15 non-reactive 5 were abnormal outcome (Table-VII). Table shows abnormal CTG, 13 had tachycardia among 03(23.08%) was abnormal outcome, 2 reduced beat to beat variability 1(50%) found abnormal outcome, among 18 deceleration 15(83.3%) were abnormal outcome and 15 non-reactive 5 were abnormal outcome (Table-VIII). Table shows in abnormal CTG APGAR score 11(22%) between 4-5 were in 1 minute and normal CTG were 03(6%) that was statistically significant ($p<0.05$). APGAR score < 7 at 5 minutes 09(18%) patients was in abnormal CTG and 01 patients (2%) in normal CTG ($p<0.01$) that was statistically significant . Sixteen percent patients had birth asphyxia in abnormal CTG and 02% patients in normal CTG ($p<0.05$) that was statistically significant. Patients needed NICU admissions compared to group-A and group-B 36% and 08% respectively of those babies ($p<0.05$) that was statistically significant. In abnormal CTG group 08% were neonatal mortality (Table-IX).

Table I: Age group distribution of the study population

Age (Years)	Abnormal CTG (group-A)	Normal CTG (group-B)	Total
17-20	11	12	23
21-25	17	15	32
26-30	09	10	19
31-35	08	07	15
36-40	05	06	11
Mean ±SD	23.45(±3.56)	24.21(±4.12)	17-40 years

Table II: Distribution of parity of the study groups

Parity	Abnormal CTG (group-A)	Normal CTG (group-B)	Total	p value
Primi	31(62%)	17(34%)	48	0.009
Multi	19(38%)	33(66%)	52	
Total	50(100%)	50(100%)	100	

Table III: Gestational age

Gestational age	Abnormal CTG (group-A)	Normal CTG (group-B)	Total	P value
37-40 wks	42(84%)	29(58%)	71	0.004
>40 wks	08(16%)	21(42%)	29	
Total	50(100%)	50(100%)	100	

Table IV: Haemodynamics, hematocrit and blood sugar levels of the study patients

Parameters	Abnormal CTG Mean ±SD	Normal CTG Mean ±SD	p value
Pulse/minute	84.84±6.5	82.4±6.9	0.02*
Systolic Blood Pressure (mmHg)	124.9±17.3	123.9±16.9	0.77NS
Diastolic Blood Pressure (mmHg)	82.7±13.2	81.4±12.4	0.38 NS
Haemoglobin (gm %)	10.1± 1.7	10.4± 1.3	0.50 NS
Blood Sugar (mmol/l)	6.8± 1.8	6.3± 1.0	0.02*

Table V: Mode of delivery normal and abnormal CTG

Mode of delivery	Abnormal CTG (group-A)	Normal CTG (group-B)	p value
LUCS	39(78%)	13(26%)	<0.001
NVD	11(22%)	37(74%)	
Liquor amount			
Almost nil	06(12%)	00	0.09
Less amount	15(30%)	13(26%)	
Adequate	28(56%)	37(74%)	
Polyhydramnios	01(02%)	00	
Liquor Color			
Normal	31(62%)	42(84%)	0.003
Meconium stained	19(38%)	08(16%)	
Umbilical cord			
Normal	47(94%)	50(100%)	0.24
Compressed	03(06%)	00	
Placenta			
Normal	42(84%)	48(96%)	0.04
Big	03(06%)	00	
Small	05(10%)	02(04%)	

Table VI: Incidence of foetal distress (FD) in specific risk factors of the study groups

Risk factors	Abnormal CTG (group-A)	Normal CTG (group-B)	Total
Postdated	39(78%)	08(16%)	47
PIH	18(36%)	05(10%)	23
PIH with IUGR	05(10%)	03(06%)	08
IUGR	07(14%)	03(06%)	10
PROM	14(28%)	04(08%)	18
BOH	08(16%)	02(04%)	10
Oligohydramanious	06(12%)	02(04%)	08
Diabetes	-	-	05
Rh-ve pregnancy	03(06%)	01(02%)	04
Others	03(06%)	02(04%)	05

Table VII: Outcome of abnormal CTG

Types of abnormal CTG	Normal Outcome	Abnormal Outcome
Tachycardia (n=13)	10 (76.9%)	03(23.08%)
Bradycardia (n=2)	02(100%)	--
Absent/reduced beat to beat variability (n=2)	01(50.0%)	01(50.0%)
Deceleration (n=18)	03(16.67%)	15(83.3%)
Non reactive (n=15)	10(66.7%)	05(33.3%)
Total (n=50)	26(52.0%)	24(48.0%)

Table VIII: Outcome of abnormal CTG

Types of abnormal CTG	Normal Outcome	Abnormal Outcome
Tachycardia (n=13)	10 (76.92%)	03(23.08%)
Bradycardia (n=2)	02(100%)	--
Absent/reduced beat to beat variability (n=2)	01(50.0%)	01(50.0%)
Deceleration (n=18)	03(16.67%)	15(83.3%)
Non reactive (n=15)	10(66.7%)	05(33.3%)
Total (n=50)	26(52.0%)	24(48.0%)

Table IX: Fetal outcome of the study population

	Abnormal CTG (group-A) Mean ±SD	Normal CTG (group-B) Mean ±SD	P value
Birth weight (kg) (mean ±SD)	2.49±0.83	2.8±0.52	0.03
Apgar score 1 minutes mean ±SD)	6.2 ±1.5	7.0 ±0.9	0.003
Apgar score 5 minutes mean ±SD)	8.42 ±1.3	9.2 ± 1.4	0.01
Immediate resuscitation needed	32(64%)	19(37%)	0.009
Admission to NICU	18(36%)	04(08%)	0.002
Neonatal death	04(08%)	---	--

Discussion

This cross-sectional study was conducted in the Department of Obstetrics and Gynaecology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet during the period of six months after the acceptance of protocol with a view to explore the role of cardiotocography (CTG) in the management of high risk pregnancy. In present study showed mean age was 23.45(±3.56) years in abnormal CTG (Group -A) and 24.21(±4.12) years in normal CTG (Group –B). Minimum age was 17 years and maximum age was 40 years. The mean age was found 26.7±4.9 years in abnormal CTG and 26.7 ±4.9 years in normal CTG. The difference was not statistically significant between two groups (p=0.15).

In current study observed that term gestational age is significantly higher in abnormal CTG and post term gestation in higher in normal CTG (p <0.05) that was statistically significant. The purpose of this study was to test the ability of a CTG to predict pregnancy outcomes and early neonatal outcomes. CTG was taken from patients at > 32 weeks gestation because the likelihood of a non-reactive test is substantially increased early in third trimester. Both Lavin et al[7] and Druzin et al[8] have reported that 15% of NST remain non reactive between 28 and 32 weeks. After 32 weeks, the incidences of reactive and non-reactive tests are comparable to those seen at term and eliminate concern regarding the immature nervous system.[6] Before 27 weeks gestation, the fetal heart response to fetal movement may in fact be a bradycardia.[6]

In this study pulse and blood sugar were significantly higher in abnormal CTG than normal CTG. Khatun et al. haemodynamic parameters and haemoglobin were similar in both normal and abnormal CTG group however; blood sugar was higher among the patient with abnormal CTG (p<0.05).

In this study observed mode of delivery with the results of the admission test and occurrence of foetal

distress (FD). Majority (78%) were LUCS in abnormal CTG that is significantly higher than normal CTG. Meconium stained 38% in abnormal CTG that is significantly higher than normal CTG which was 16%. Placenta big and small 06% and 10% respectively in abnormal CTG that was significant higher than normal CTG that was 02(4%). Khatun et al. caesarian delivery and meconium staining liquor was significantly higher in abnormal CTG group ($p < 0.001$). It was also revealed that the proportion of adequate amount of liquor was higher among the women with normal CTG (74.0.0%) compared to abnormal CTG (56.0%), but the difference was not statistically significant ($p>0.05$). Similarly the proportion of normal placenta was significantly high among the women with normal CTG (96.0%) compared to abnormal CTG (84.0%). However, a statistically significant difference was found ($p<0.05$) in colour of liquor indicating the proportion of Meconium stained liquor was higher among the women with abnormal CTG (40.0%) compared to normal CTG (14.0%). No statistically significant difference was found in umbilical cord compression ($p>0.05$) though the compressed umbilical cord was found to be high among the women with abnormal CTG.

IN present study incidence of foetal distress (FD) in specific risk factor groups. Data are expressed as number (N) and percentage. Common risk factors in abnormal CTG were postdated, PIH, PIH with IUGR, IUGR, PROM, BOH and Oligohydramanious 78%, 36%,10%,14%, 28%,16% and 12% respectively. When the risk factors are more the overall outcomes are more among the abnormal CTG group.[03] In Amena Khatun et al.[3] study it was seen that when diabetic patients developed preeclampsia, intrauterine growth retardation, oligohydramnios, abnormal outcomes were more and risk factors are interrelated, one predispose to others. In respect to mode of delivery, there was a high incidence of cesarean section in this study. The reason for high incidence of cesarean section in this study in spite of normal test result was due to obstetrical indications, like history of previous cesarean section, cephalopelvic disproportion, severe preeclampsia and severe intrauterine growth retardation. Here cesarean delivery for fetal distress was significantly higher in abnormal CTG group, this finding is similar to the observation of Dellinger et al.[9] In this study showed abnormal CTG, 13 had tachycardia among 03(23.08%) was abnormal outcome, 2 reduced beat to beat variability 1(50%) found abnormal outcome, among 18 deceleration 15(83.3%) were abnormal outcome and 15 non-reactive 5 were abnormal outcome. CTG showing only tachycardia had 23.08% abnormal out comes. It is the early sign of fetal distress. [74] As interventions were taken early outcomes were good. Bradycardia was seen in 4% case outcome was normal. Druzin et al[4], Dashow et al[11] in their study observed bradycardia in 1- 2% of all CTG. Druzin et al[10] defined such bradycardia, a fetal heart rate of 90 beats per minute or a fall in the fetal heart rate of 40 beats per minute below the base line for one minute or longer. Druzin et al[10] in his study found that bradycardia was associated with increased morbidity and mortality; the causes were cord compression, IUGR and fetal malformation. In the current study 50% tracings with absent beat-to-beat variability showed abnormal outcomes. Studies done by Shields et al[12] and Langer et al[3] demonstrated that the fundamental component of ominous fetal heart rate pattern is absent or markedly decreased FHR variability. In this study non-reactive CTG were associated with 33.3% abnormal outcomes which is similar to the studies done by Kubli et al[14], Schifrin et al[15] Keogen et al[16], Flynn et al[7].

In this study abnormal CTG APGAR score 11(22%) between 4-5 were in 1 minute and normal CTG were 03(6%) that was statistically significant ($p<0.05$). APGAR score < 7 at 5 minutes 09(18%) patients was in abnormal CTG and 01 patients (2%) in normal CTG ($p<0.01$) that was statistically significant . Sixteen percent patients had birth asphyxia in abnormal CTG and 02% patients in normal CTG ($p<0.05$) that was statistically significant. Patients needed NICU admissions compared to group-A and group-B 36% and 08% respectively of those babies ($p<0.05$) that was statistically significant. In abnormal CTG group 08% were neonatal mortality. Fetal birth weight and APGAR score at 1 minute and 5 minute was significantly lower in abnormal CTG group. The babies needed immediate resuscitation and admission at neonatal care unit was higher in this group. The fetal death rate was high among the women with abnormal CTG. Reduced liquor volume before labour is considered an indication of placental insufficiency and reduced liquor volume during labour is associated with an increased incidence of FHR decelerations and may reduce the volume of the intervillous space and may predispose to umbilical cord occlusion, which increase the risk of fetal hypoxemia. In the past, the presence of meconium in the amniotic fluid was considered to be a sign of fetal hypoxia. In the study Apgar score was < 7 at 1 min among the babies of abnormal CTG group than normal CTG that was similar to the study done by Dellinger et al.[9] The difference in number of the babies with Apgar score < 7 at 5 min was very significant in normal CTG group than abnormal CTG group which was similar to many other studies. Apgar scoring has been the conventional means of evaluating the status of the infant at birth. It is usually assumed that this score reflects the degree of neonatal asphyxia. However recent studies 17 using

cord blood analysis and fetal scalp blood pH have cast serious doubts on the reliability of Apgar scoring for asphyxia. An analysis of several published works on the subject gives the following approximate indexes of accuracy for the Apgar score in the prediction of hypoxemia: In the present study no perinatal death was observed in normal CTG group which was similar to some other studies. Perinatal mortality (PNM) among total study population (n=100) was 40 per 1000. The PNM rate in the present study was significantly lower than the PNM in other teaching centers of Bangladesh. Timely intervention according to CTG results may be the reasons for such variation. The study did not attempt to demonstrate an ability to decrease cesarean delivery rates, nor did it attempt to link electronic fetal monitoring with long term neurologic function and cerebral palsy. It only attempted to show the pregnancy outcome and early neonatal outcomes in case of normal and abnormal CTG cases.

In conclusion, the present study showed that the mean Apgar score was low among the babies with mothers having abnormal than normal CTG. The proportion of immediate resuscitation and admission of the babies at neonatal care unit was higher among this group. Perinatal death was also significantly higher in this group. Thus overall outcome was better in normal CTG group. On the other hand the perinatal mortality (PNM) in the present study was significantly lower than the PNM of Bangladesh. Timely intervention according to CTG results may be the reasons for such variation. Many neonates in abnormal CTG group were not asphyxiated at birth.

Reference

1. Gupta M and Gupta P. Role of cardiotocography in high risk pregnancy and its correlation with increase cesarean section rate. Int J Reprod Contracept Obstet Gynecol 2017;6: 168-71.
2. Dawes GS, Lobb M, Moulden M, Redman CWG, Wheeler T. Antenatal cardiotocogram quality and interpretation using computers. British J Obstet Gynaecol 1992; 99: 791–7.
3. Rahman H, Renjhen P, Dutta S, Kar S. Admission cardiotocography: Its role in predicting fetal outcome in high risk obstetric patients. AMJ 2012, 5: 522-7.
4. Valensise H, Facchinetti F, Vasapollo B, Giannini F, Di Monte I, Arduini D. The computerized fetal heart rate analysis in post-term pregnancy identifies patients at risk for fetal distress in labour. European J Obstet Gynecol Reproductive Biol 2006; 125:185–92.
5. Soncini E, Ronzoni E, Macovei D, Grignaffini A. Integrated monitoring of fetal growth restriction by computerized cardiotocography and Doppler flow velocimetry. European J Obstet Gynecol Reproductive Biol 2006; 128: 222–30.
6. Kuhnert M, Hellmeyer L, Stein W, Schmidt S. Twenty-four-hour CTG monitoring: comparison of normal pregnancies of 25-30 weeks of gestation versus 36-42 weeks of gestation. Archives Gynecol Obstet 2007; 275:451–60.
7. Buscicchio G, Giannubilo SR, Bezzeccheri V, Scagnoli C, Rinci A, Tranquilli AL. Computerized analysis of the fetal heart rate in pregnancies complicated by preterm premature rupture of membranes (pPROM). J Maternal-Fetal Neonat Med 2006; 19: 39-42.
8. Bellver J, Perales A, Maiques V, Serra V. Can antepartum computerized cardiotocography predict the evolution of intrapartum acid-base status in normal fetuses?. Acta Obstetricia et Gynecologica Scand 2004; 83: 267-71.
9. Freeman, RK, Anderson G, Dorchester W. A prospective multi-institutional study of antepartum fetal heart rate monitoring. I. Risk of perinatal mortality and morbidity according to antepartum fetal heart rate test results. Am J Obstet and Gynecol 1982; 143: 771-7.
10. Freeman RK, Anderson G, Dorchester W. A prospective multi-institutional study of antepartum fetal heart rate monitoring. II. Contraction stress test versus nonstress test for primary surveillance. Am J Obst Gynecol 1982; 143: 778-81.
11. Phelan, JP. The nonstress test: a review of 3,000 tests. Am J Obstet Gynecol 1981; 139: 7-10.
12. Flynn AM, Kelly J. Evaluation of fetal wellbeing by antepartum fetal heart rate monitoring. BMJ 1977; 1: 936–9.
13. Boehm FH, Salyer S, Shah DM, Vaughn WK. Improved outcome of twice weekly nonstress testing. Obstet Gynecol 1986; 67: 566–
14. Sadovsky E, Weinstein D, Even Y. Antepartum fetal evaluation by assessment of fetal heart rate and movements. International J Gynecol Obstet 1981; 19: 21–6.
15. Trimbos JB, Keirse MJ. Significance of antepartum cardiotocography in normal pregnancy. Br J Obstet Gynaecol 1978; 85: 907–13.
16. Behuria S and Nayak R. Admission cardiotocography as a screening test in high risk pregnancies and its co-relation with peri-natal outcome. Int J Reprod Contracept Obstet Gynecol. 2016; 5: 3525-8.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Clinical Presentation Of Acute St Elevation Myocardial Infarction In Young Adult (Below 40 Years)

Ranjon kumer Roy[1], Md. Sohorab Hossain Bhuiyan[2], Suchanda Ray[3], Shishir Kumar Basak[4], Shah Emran[5], Syed Abul Foez[6], Md. Moyeen Uddin[1]

Abstract

This study is conducted on the clinical presentation of acute ST elevation myocardial infarction in young adult (below 40 years) during the period of March, 2007 to February , 2008 in Sylhet MAG Osmani Medical College Hospital. Total 100 consecutive patients admitted with acute transmural MI were included in the study. Most of the cases 87% were male. Among them 90% of the patient admitted with typical chest pain associated with sweating. The remaining presented atypically with dyspnoea, breathlessness, palpitation, vomiting, and abdominal pain. In context of different modalities clinical presentation of elevated ST level MI patients, this study has been designated for early diagnosis of cases and efficient management of acute MI as well as attempt to prevent complication.

[OMTAJ 2016; 15(2)]

Introduction

Cardiovascular diseases are leading cause of morbidity and mortality in industrialized countries and they are also emerging as a public health problem in developing countries[1].Myocardial infarction is the term applied to myocardial necrosis secondary to an acute interruption of the coronary blood supply[2]. It is almost always due to the formation of occlusive thrombus at the site of rupture of an athermatous plaque in a coronary artery[3].Despite a substantial reduction in age-adjusted rate of death from cardiovascular causes during the past 40-50 years, cardiovascular disease remains the single most common cause of natural death in developed nation. [4,5] The incidence of myocardial infarction is gradually increasing in Bangladesh at a rate of 0.5% per year[6]. In myocardial infarction the increased incidence of morbidity and mortality is attributed to various complications, which occurs in the early course of the disease. In about one quarter of all episodes of acute myocardial infarction, death occurs suddenly within minutes of onset. Innumerable works were done and still being continued, increasing the number of publication to explore the different aspects of this major killer disease in different corners of the world.

1. Assistant Professor, Department of Medicine, MAG Osmani Medical College, Sylhet.
2. Professor , Department of Pathology, Prime Medical College, Rangpur.
3. Associate Professor, Department of Pathology, Jalalabad Ragib- Rabeya Medical College, Sylhet.
4. Associate Professor, Department of Medicine, MAG Osmani Medical College, Sylhet.
5. Assistant Professor, Department of Endocrinology, MAG Osmani Medical College, Sylhet.
6. Assistant Professor, Department of Hepatology, MAG Osmani Medical College, Sylhet.

MATERIALS AND METHODS

A total number of 100 consecutive newly patients admitted with acute transmural myocardial infarction of both sexes being age below 40 years included in the study between March'2007 to last week of Feb' 2008. Diagnosis of acute myocardial infarction was made when two out of the following three criteria were fulfilled.

1) Typical chest pain suggesting myocardial infarction with or without radiation.
2) The development of pathological Q wave and ST-T wave changes that are typical of myocardial infarction.
3) Rise of Troponin-I, serum Aspertate amino transferase (AST) or Creatine kinase enzymes.

Patients presenting to the emergency department with a chief complaints of chest pain or an equivalent complaint such as pressure or heaviness in the chest were classified as having typical symptoms. Atypical symptoms included other chief complaints such as shortness of breath, weakness, syncope or anything else. Detailed history of chest pain, regarding its duration, character, location and radiation was taken.

Associated symptoms like sweating, breathlessness, weakness, nausea, vomiting palpitation etc. were also noted. Findings of ECG and indicative laboraratory investigations such as CK-MB, Troponin-I, serum cholesterol, blood sugar etc. are also collected.Patient having history of obstructive airway disease & valvular heart disease is excluded.

Results

In this study 100 patients were included with age range of 25 to 40 years.Among them 87% are male to female ration 8.7:1.3.The maximum incidence of MI was in the age group of 31-35years which comprises 60% of the total patients studied. 90(90%) patients were presented with typical chest pain. The commonest symptom associated with chest pain was sweating in 78 patients out of 90 cases. The commonest sign was irregular pulse (59%), followed by tachycardia (42%) and pulmonary oedema (28%).Nine patients(9%) had cardiogenic shock. Nine- five patients had transmural infarction and five had sub-endocardial infarction. Out of 95 cases transmural acute myocardial infarction, 58 had anterior MI, 35 (35%) had inferior MI, 1 (1%) had combind anterior and inferior MI.Among it, the anterior MI 30(30%) had extensive anterior MI, 16(16%) had anteroseptal MI and12(12%) had anterolateral MI.

Incidence of age group and distribution of symptoms are given below in Table I and Table II.

Table-I: Shows the incidence of different age group.

Age group (years)	Number of patients	Percentage
25-30	2	2%
31-35	60	60%
36- 40	38	38%

Table-II: Show the frequency of distribution of symptoms.

Symptoms	Number of Patients	Percentage (%)
Chest pain	90	90
Breathlessness	60	60
Palpitation	48	48
Vomiting	45	45
Sweating	78	78
Abdominal pain	5	5
Syncope	4	4
No chest pain	10	10

Note:Many patients had multiple symptoms

Discussion

In the series of 100 patients, the peak incidence of acute myocardial infarction was found in the age group 31-35 years (60 %). Comparable results were shown by Amanullah found in a study among the younger age group that maximum incidence (61 %) in the age group 31-35 years [7].In this study male and female ratio was 8.7:1.3. this is nearly similar to the finding of Malik et al [6] of 11.1, Khandaker et al [8] of 7.8:1. In western studies though the incidence is higher in male, the male and female ratio is much lower than that of us. A very low incidence in female in this part of the world may be due to fact that they do not get medical attention as the male. In our country, this male and female ration gradually declines that was shown in one study [9] from 8.1:6 to 7:1 which may be due to cope up with responsibilities, worries and anxieties, contraceptives and with more or less addiction to smoking.

Chest Pain, in its own characteristics special for acute myocardial infarction, is the commonest presenting feature. Chest discomfort usually described as pain of compressive nature is substituted by some patients with an equivalent complaints such as heaviness, pressure or tightness in the chest. In this present series, 90patients (90%) presented with the chief complaints of chest discomfort, majority describe pain and other 10 patients (10%) presenting with other symptom such as sweating and vomiting. Palpitation, abdominal pain and syncope was also present to certain extent.Breathlessness or dyspnoea in acute myocardial infarction is somehow related with pulmonary congestion and oedema,probably resulting from depressed cardiac function after acute trauma to the heart. Whenever this pulmonary oedema causing dyspnoea is out of proportion it may mask the chest pain, sometimes making the diagnosis difficult. From United States observed that in addition to high early mortality in hospital associated with acute myocardial infarction causing pulmonary oedema, in the long run, patients with pulmonary congestion have a higher 1-year mortality rate compared with patients without pulmonary oedema(28 % versus 5.5%).So breathlessness occurring after myocardial infarction caused by pulmonary oedema is not only a clinical presenting feature but a prognostic marker also[10].

As the present study was done on a small number of selected populations, it may not reflect the exact

picture but it may help in the more extensive studies in the future.

In conclusion, this prospective study in a consecutive group of 100 patients admitted with acute ST elevated myocardial infarction in young adult in Sylhet MAG Osmani medical college Hospital found that the incidence of myocardial infarction highest in the age group 31 to 35 years. The diagnosis of AMI was based on clinical, electrocardiographic and biochemical criteria. The cases were collected at random, serial ECG and continuous cardiac monitoring. The findings of the present series were correlated and discussed in relation to the findings of other studies at home and abroad. This observation has important implications as majority of the patients belonged to young and middle aged group, who are in the most active and productive stage of life. Still we have to study the problem in context of our situation in Bangladesh in an attempt to reduce the overall morbidity and mortality, our study exercise helps to prevent the occurrence of newer cases of IHD to the better possible treatment of the affected persons.

Reference

1. Malik A, Islam NM, Zafar A, Kader A, Ramijuddin M.. Clinical patterns of ischaemic heart diseases and its association with some known risk factors. Bangladesh heart J 1987; 21:1-9
2. Julian DG, Cowan JC. 2005. Cardiology, 8th edn, clinical presentation of coronary disease, W. B.Saunders Company, Philadelphia, pp-108,121,124.
3. Boon NA, Colledge NR,Walker BR.2006. Davidson's Principle and Practice of Medicine, 20th edn,Churchill Livingstone, Philadelphia, pp-519.
4. Myerburg RJ, Keisler KM, Castellanos A. Sudden cardiac death,epidemiology, transient risk and intervention assessment, Ann Intern med 1993;119:1187-97.
5. Myerburg RJ, Interian AJ. Mitrani RM, Kessler KM, Castellanos A. Frequency of sudden cardiac death and profiles of risk. Am J Cardiol 1997; 80:10F-19F.
6. Nabi MN, Yunus EB. Epidemiology of myocardial infarction, proceedings of the Bangladesh Japan joint conference on cardiovascular diseases published by Japan International cooperation agency, 1995.
7. Amanullah M, Thapa LB, Hasnat A, malik A.. A profile of 51 cases of myocardial infarction amongst young Banglees, Bangladesh Japan joint conference on Cardiology publication 1984;1:118-124.
8. Khandaker RK, Hossain D, Hossain M, Shamsuzzaman. Retrospective analysis of acute myocardial infarction, A 4 years study of 2690 patients. Bangladesh Heart J 1986;1:14I7.
9. National heart, lung and blood institute. Morbidity and mortality, chart blood on cardiovascular, lung and blood disease, Bethesda MD, US Department of Health and Human Services, Public Health Service National Institute of Health, May 1992.
10. Spodick DH, Flessas AP, Johnson TM. Association of acute respiratory symptoms with onset of acute myocardial infarction,prospective investigation of 150 consecutive patients and matched control patients, Am J Cardiol 1984; 53:481-2.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Diagnostic Performance Of MRI In The Evaluation Of Tuberculous Spondylitis

Farhana Chowdhury[1], Swajal Chandra Das[2], Tahmina Yasmin[3], Aliza Mamataz[4], N. K. Paul[5], MM. Ashikur Rahman[5], Md. Maksudul Azim[2], Sudipta Gope[2], Nahar-E-Zannat[2]

Abstract

MRI is a useful diagnostic modality for the diagnosis of suspected tubercular spondylitis. There are certain characteristics of tuberculous spondylitis seen on MRI. The MRI diagnosis of tuberculous spondylitis in this study was compared with histopathology findings. Aim and objectives is to assess the diagnostic performance of MRI in evaluation of tuberculous spondylitis. As the validity test results are higher, it can be concluded that the MRI is useful diagnostic modality in the evaluation of tuberculous spondylitis.

This cross sectional study was carried out in the Department of Radiology & Imaging, in collaboration with Department of Neurosurgery and Department of Pathology of Dhaka Medical College Hospital, Dhaka, during July 2014 to June 2016, to evaluate the MRI findings of a suspected tuberculous spondylitis and to compare the MRI findings of tuberculous spondylitis with that of histopathology and also to find out the sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for evaluation of tuberculous spondylitis.

The mean age was found 34±14.7 years with range from 8 to 75 years. Male female ratio was 1.9:1. Majority (87.2%) patients had back pain followed by 35(74.5%) had neurologic deficit, 27(57.4%) had fever and 17(36.2%) had spinal deformity/kyphosis.

Almost two third (63.8%) patients had thoracic spinal involvement. Almost half (46.8%) of the patients had involvement two vertebrae, 4(8.5%) had single, 17(36.2%) had three and 4(8.5%) had involvement more than three vertebrae. All (100.0%) patients had altered marrow signal intensity of involved vertebrae, followed by 43(91.5%) vertebral collapse, 32(68.1%) end plate disruption, 25(53.2%) spinal deformity/ kyphosis and 8(17.0%) posterior element involvement.

Majority (89.4%) patients had disc involvement and 42(89.4%) had signal change. More than three fourth (78.7%) patients had well defined paraspinal abnormal signal and 10(21.3%) had ill defined paraspinal abnormal signal. Majority (46.8%) patients showed cord compression and 8(17.0%) showed cord compression with myelopathy.

More than two third (68.1%) patients was diagnosed as having tuberculous spondylitis, 12(25.5%) pyogenic spondylitis and 3(6.4%) as spinal metasteses by MRI. Two third 31(66.0%) patients was diagnosed of tuberculous spondylitis, 13(27.6%) pyogenic spondylitis and 3(6.4%) were metastasis by histopathology.

The validity of MRI in the evaluation of tuberculous spondylitis showed sensitivity of 93.5%, specificity of 81.3%, accuracy of 89.4%, positive predictive values of 90.6% and negative predictive values of 86.7%.

MRI is a useful diagnostic modality for the diagnosis of suspected tubercular spondylitis. It should be the radiographic examination of choice in looking for or determining the extent of spinal disease.

[OMTAJ 2016; 15(2)]

Introduction

Tuberculosis of the spine accounts for more than 50% musculoskeletal tuberculosis. In the developing countries, the disease commonly afflicts children and young adults and tends to be more aggressive in extent and abscess formation. Consequently, neurologic complications and spinal deformities are seen frequently. In the developed countries, musculoskeletal tuberculosis is uncommon, but its

1. Radiologist, Radiology & Imaging, Sylhet MAG Osmani Medical College, Sylhet.
2. Assistant Professor of Radiology & Imaging, Sylhet MAG Osmani Medical College, Sylhet.
3. Radiologist, Radiology & Imaging, Sir Salimullah Medical College & Mitford Hospital, Dhaka.
4. Medical Officer; Radiology & Imaging, Kurmitola General Hospital, Dhaka Cantonment.
5. Associate Professor of Radiology & Imaging, Sylhet MAG Osmani Medical College.

incidence is reported to be greater in older individuals[1].

Tuberculous spondylitis is typically more indolent than pyogenic osteomyelitis. Onset is often insidious, and symptom duration frequently ranges from months to years. Untreated patients develop progressive vertebral collapse with anterior wedging and gibbus formation [2]. Paraplegia may be a result of spinal cord compression or kyphosis deformity in the late state of disease. Therefore, early diagnosis and establishment of treatment are necessary for avoiding this long term disability [3] .The lower dorsal and lumber spine are affected in nearly three quarters of all cases of tuberculous spondylitis; the cervical spine is an uncommon site[4]. Vertebral collapse and kyphosis results from progressive bone destruction. The spinal canal can be narrowed by abscesses, granulation tissue, or direct dural invasion, leading to spinal cord compression and neurologic deficits[5].

Different modalities of imaging plays an important role in the overall evaluation of these lesions. MR imaging has become an established imaging technique in the diagnosis of spinal infections and their sequlae. MRI has been shown to be at least as sensitive as bone scintigraphy for detecting infections of the vertebrae and is comparable to CT for visualizing extent of disease. High contrast resolution, direct multiplanar imaging, usefulness in detecting marrow infiltration, and ease by which intradural disease can be assessed are definite advantages. On MR images, vertebral intraosseous abscesses, paraspinal soft-tissue abscesses that extend beyond the confines of disc collapse, spinal deformity (gibbus), skip lesions, encroachment on spinal canal and nerve roots, and involvement of the posterior elements are all readily detectable. Enhanced MR studies are particularly useful for characterizing tuberculous spondylitis. Rim enhancement around intraosseous and paraspinal soft tissue abscess had not been demonstrated in other spinal infection [6].

MRI has been reported to be useful in the early detection of spondylitis. The two most reliable MRI findings suggesting tuberculous spondylitis were thin and smooth enhancement of the abscess wall and well defined paraspinal abnormal signal [7].

Plain-radiography and CT scan has limited use in the diagnosis of tuberculous spondylitis because they cannot provide detailed information regarding extension, other associated abnormalities. Magnetic resonance imaging has become the modality of choice for evaluating patients with spinal complaints. Though histopathology is gold standard, it is an invasive procedure. MRI, though relatively expensive for the developing world, is safe, non invasive and devoid of radiation hazards. Early diagnosis and prompt treatment of tuberculous spondylitis is very much necessary. This can minimize residual spinal deformity or permanent neurologic defect of a large number of people. MRI can play an important role in diagnosing tuberculous spondylitis. As for this purpose, if the hypothesis is tested we can radiologically help in early evaluation and management of tuberculous spondylitis.

MATERIALS AND METHODS

This cross sectional study was carried out in the department of Radiology and Imaging of DMCH, in collaboration with Department of Neurosurgery and Department of Pathology of DMCH during the period of July 2014 to June 2016. Patients were selected who have fulfilled the inclusion criteria. Patients with clinically suspected tuberculous spondylitis who were referred to the Department of Radiology and Imaging for imaging investigation were included in my study. Finally histopathological reports were available in 47 cases who were the study population. The demographic and clinical findings of these 47 patients were analyzed and their MRI findings and histopathological diagnosis were compared. Patients with clinically suspected tuberculous spondylitis referred to Department of Radiology and Imaging, DMCH, either by OPD or in-patient Department of Neurosurgery, DMCH, for imaging investigation.

MRI was performed in the department of Radiology and Imaging, Dhaka Medical College, Dhaka using 0.3 Tesla open MRI machine (AIRIS-II-HITACHI). Images were taken of T1WI, T2WI and T1 contrast sequence in sagittal, axial and coronal plane. After intravenous infusion of 0.1 mmol/kg of gadolinium DTPA, T1 weighted axial, sagittal and coronal image were obtained immediately. The reviewer evaluated the presence or absence of individual imaging criteria in MRI and made on an overall assessment of the type of spondylitis. Refused to do MRI or undergo operative treatment and non availability of the histopathological reports were excluded.

The post-operative resected tissues or biopsy materials were examined histopathologically and reports were being compared with MRI findings.

Prior to commencement of this study an approval of the research protocol was obtained from ethical committee of committee of Dhaka Medical College,

Dhaka. Informed written consent was also taken from each patient.

The diagnostic value of MRI scan for the diagnosis of tubercular spondylitis was determined by calculating sensitivity, specificity, accuracy, positive and negative predictive values.

Measures of variables (Demographic and imaging variables)

Age of the patients, gender of the patients, clinical features, vertebrae involvement with site, number, vertebral collapse, altered marrow signal intensity of involved vertebrae, posterior element involvement, spinal deformity/Kyphosis, end plate disruption, intervertebral disc involvement, Soft tissue involvement, cord compression/ myelopathy.

RESULTS

The age of the patients ranged from 8 to 75 years with the mean age of 34 ± 14.7 years. Highest incidence was found in between 31 to 40 years of age group (13, 27.6 %) (Table-I).

Table I: Distribution of the study patients by age (n=47)

Age (in year)	Number of patients	Percentage
≤10	1	2.1
11-20	9	19.1
21-30	9	19.1
31-40	13	27.6
41-50	10	21.3
51-60	3	6.4
>60	2	4.3
Mean±SD	47	34.0 ±14.7

Table II: Distribution of the study patients by sex (n=47)

Sex	Number of patients	Percentage
Male	31	66
Female	16	34

Table II shows the distribution of the study patients according to sex, it was observed that 31(66%) patients were male and 16(34%) patients were female. Male female ratio was 1.9:1.

Table III: Distribution of the study patients by clinical presentation (n=47)

Clinical presentation	Number of patients	Percentage
Back pain	41	87.2
Neurologic deficit	35	74.5
Fever	27	57.4
Spinal deformity/Kyphosis	17	36.2

Table III shows the clinical presentation of the study patients, it was observed that 41(87.2%) patients had back pain followed by 35(74.5%) had neurologic deficit, 27(57.4%) had fever and 17(36.2%) had spinal deformity/kyphosis.

Table IV: Distribution of the study patients by site of spinal involvement (n=47)

Site of spinal involvement	Number of patients	Percentage
Lumbar	15	31.9
Thoracic	30	63.8
Cervical	2	4.3

Table IV shows the site of spinal involvement of the study patients, it was observed that 30(63.8%) patients had thoracic spinal involvement, 15(31.9%) had Lumbar and 2(4.3%) had cervical spinal involvement.

Table V: Distribution of the study patients by number of vertebral involvement (n=47)

Number of vertebral involvement	Number of patients	Percentage
Single	4	8.5
Two	22	46.8
Three	17	36.2
More than three	4	8.5

Table V shows the number of vertebral involvement of the study patients, it was observed that 22(46.8%) patients had involvement of two vertebrae, 4(8.5%) single (figure 3), 17(36.2%) three and 4(8.5%) more than three vertebral involvement.

Table VI: Distribution of the study patients by MRI findings of involved vertebrae (n=47)

Vertebrae involvement	Number of patients	Percentage
Altered marrow signal intensity of involved vertebrae	47	100.0
Vertebral collapse	43	91.5
End plate disruption	32	68.1
Spinal deformity/Kyphosis	25	53.2
Posterior element involvement	8	17.0

Table VI (Figure-1) shows the MRI findings of involved vertebrae, it was observed that all (100.0%) patients had altered marrow signal intensity of involved vertebrae, followed by 43(91.5%) vertebral collapse, 32(68.1%) end plate disruption, 25(53.2%) spinal deformity/kyphosis and 8(17.0%) posterior element involvement.

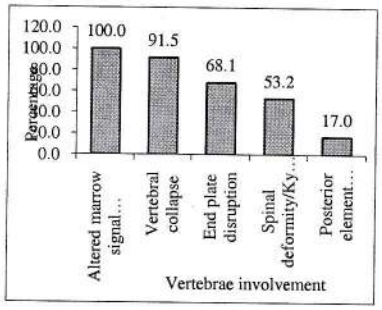


Figure 1: Bar diagram shows MRI findings of involved vertebrae of the study patients

Table VII: Distribution of the study patients by intervertebral disc involvement (n=47)

Disc involvement	Number of patients	Percentage
Yes	42	89.4
No	5	10.6

Table VII shows the intervertebral disc involvement of the study patients, it was observed that 42(89.4%) patients had disc involvement.

Table VIII: Distribution of the study patients by signal change of intervertebral disc (n=47)

Signal change	Number of patients	Percentage
Yes	42	89.4
No	5	10.6

Table VIII shows the signal change of intervertebral disc of the study patients, it was observed that 42(89.4%) showed signal change of intervertebral disc. Figure 2 shows paraspinal abnormal signal of the study patients, it was observed that 37(78.7%) patients had well defined paraspinal abnormal signal and 10(21.3%) ill defined paraspinal abnormal signal.

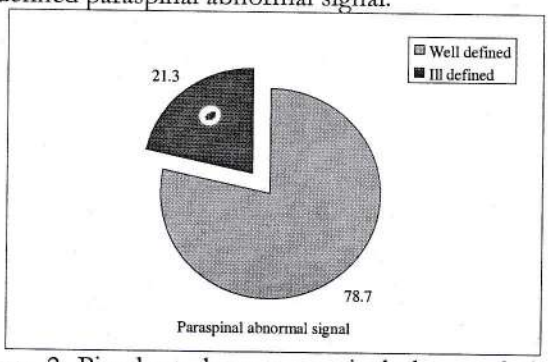


Figure 2: Pie chart shows paraspinal abnormal signal of the study patients

Table IX: Distribution of the study patients by margin of abscess wall after contrast administration (n=47)

Margin of abscess wall after contrast administration	Number of patients	Percentage
Thin smooth	33	70.2
Thick irregular	14	29.8

Table IX shows margin of abscess wall after contrast administration of the study patients, it was observed that 33(70.2%) patients had thin smooth and 14(29.8%) thick irregular margin of abscess wall after contrast administration.

Table X: Distribution of the study patients by cord compression (n=47)

Cord compression	Number of patients	Percentage
Yes	22	46.8
No	25	53.2

Table X shows cord compression of the study patients, it was observed that 22(46.8%) patients had cord compression.

Table XI: Distribution of the study patients by cord compression with myelopathy (n=47)

Cord compression with myelopathy	Number of patients	Percentage
Yes	8	17.0
No	39	83.0

Figure3: Dorsal spine shows compression collapse of D9 dorsal vertebrae.

Table XI shows cord compression with myelopathy of the study patients, it was observed that 8(17.0%) patients had cord compression with myelopathy.

Table XII: Distribution of the study patients by MRI diagnosis (n=47)

MRI diagnosis	Number of patients	Percentage
Tuberculous spondylitis	32	68.1
Pyogenic spondylitis	12	25.5
Spinal metasteses	3	6.4

Table XII shows MRI diagnosis of the study patients, it was observed that 32(68.1%) patients diagnosed as tuberculous spondylitis, 12(25.5%) pyogenic spondylitis and 3(6.4%) was spinal metasteses.

Table XIII: Distribution of the study patients by histopathological diagnosis (n=47)

Histopathological diagnosis	Number of patients	Percentage
Tuberculous spondylitis	31	66
Pyogenic spondylitis	13	27.6
Spinal metasteses	3	6.4

Table XIII shows histopathological diagnosis of the study patients, it was observed that 31(66%) patients diagnosed as tuberculous spondylitis, 15 (27.6 %) pyogenic spondylitis and 3(6.4%) was spinal metasteses.

Table XIV: Comparison between histopathological diagnosis and MRI diagnosis (n=47)

MRI diagnosis	Histopathological diagnosis	
	Positive (n=31)	Negative (n=16)
Positive (n=32)	29 (True positive)	3 (False positive)
Negative (n=15)	2 (False negative)	13 (True negative)

Table XIV shows the relationship of MRI and histopathological diagnosis of study patients. Out of all cases 32 were diagnosed as tuberculous spondylitis by MRI and among them 29 were confirmed by histopathologically. They were true positive. Three cases were diagnosed as having tuberculous spondylitis by MRI but not confirmed by histopathological finding. They were false positive. Out of 15 cases of non tuberculous spondylitis which were confirmed by MRI, 2 were confirmed as tuberculous spondylitis and 13 were non tuberculous spondylitis by histopathological findings. They were false negative and true negative respectively.

Table XV: Test of validity.

Validity test	Percentage
Sensitivity	93.5
Specificity	81.3
Positive predictive value	90.6
Negative predictive value	86.7
Accuracy	89.4

The validity of MRI in the evaluation of tuberculous spondylitis was seen by comparing with histopathological test.

DISCUSSION

In this present study it was observed that more than one fourth (27.6%) patients with Tuberculous spondylitis were in 4th decade and the mean age was found 34.0±14.7 years with range from 8 to 75 years. Similarly, Khalequzzaman & Hoque [8] found the mean age was 33.33±5.23 years varied from 11-69 years. Older age group 7th decade rather least affected 4.35%, which closely resembling with the current study. Tuberculous spondylitis can occur in any age. Middle aged adults are the most frequently affected by tuberculous spinal infection[6].

In this series it was observed that 41(87.2%) patients had back pain followed by 74.5% had neurologic deficit 57.4% had fever and 36.2% had spinal deformity/kyphosis. Back pain remained the most frequently recorded symptom and fever was found to be more regular finding than developed country as these symptoms mostly compel the patient in our setting to pay a visit to doctor [9,10]. Another study Ogle et al[11] showed that kyphosis was found to be a valuable marker for spinal tuberculosis and this study also showed that 1.33% of patients had kyphosis. Similar findings also was observed by Alvi et al [12] which was back pain 84.9%, fever 71.4% and neurological deficit 63%.

In the study, 30(63.8%) patients had thoracic spinal involvement, 31.9% had Lumbar and 4.3% had cervical spinal involvement,very much similar to the study done by others.[7,8] In this present study it was observed that 22(46.8%) patients had involvement of two vertebrae, 8.5% single, 36.2% three and 8.5% more than three vertebral involvement, which is similar with the study performed by others [7,8,13,14,16].

In this current study it was observed that all (100.0%) patients had altered marrow signal intensity of involved vertebrae, followed by 91.5% vertebral collapse, 68.1% end plate disruption, 53.2% spinal deformity/kyphosis and 17.0% posterior element involvement, 42(89.4%) patients had disc involvement and 42(89.4%) showed signal change of intervertebral disc almost similar to Bajwa and Hasan [14,15].

In this study it was observed that 37(78.7%) patients showed well defined paraspinal abnormal signal and 21.3% ill defined paraspinal abnormal signal ,almost close to the study by Jung et al [7],not similar to Hasan[15] found paraspinal abnormal signal 95% cases.

In this current study it was observed that the validity of MRI in the evaluation of tuberculous spondylitis sensitivity was 93.5%, specificity was 81.3%, accuracy was 89.4%, positive predictive values was 90.6% and

negative predictive values was 86.7%. which are very much consistent with the study done by Jung et al[7] Khalequzzaman & Hoque [8] reported that sensitivity, specificity and accuracy of MRI were 95.2%, 75% and 93.5% respectively. The above studies findings are consistent with the current study.

The limitation of the study that the study population was selected from one selected hospital in Dhaka city, so that the results of the study may not reflect the exact picture of the country.The present study was conducted at a very short period of time with small sample size.Therefore, in future further study may be under taken with large sample size.

In conclusions MRI is a useful diagnostic modality for the diagnosis of suspected tubercular spondylitis among the Bangladeshi population. There are certain characteristics of tuberculous spondylitis seen on MRI. The MRI diagnosis of tuberculous spondylitis in this study was compared with histopathology findings. As the validity test results are higher, it can be concluded that the MRI is useful diagnostic modality in the evaluation of tuberculous spondylitis. However, further study with larger study population involving several investigators at multiple centers may give more precise results which may be the logical recommendation of this study.

REFERENCES

1. Moorthy S, Prabhu NK, Spectrum of MR Imaging Findings in Spinal Tuberculosis. AJR, 2002;179: 979-83.
2. Osborn AG, Diagnostic Neuroradiology, India: Elseviers, 2004.
3. Danchaivijitr N, Temram `S, Thepmongkhol K and Chiewvit P, Diagnostic Accuracy of MR Imaging in Tuberculous Spondylitis. J Med Assoc Thai 2007; 90:8: 1581-9.
4. Renton P, Periosteal Reaction; Bone and Joint Infections; Sarcoid, In: Sutton D, editor. Text book of Radiology and imaging.7th ed, London; Churchill Livingstone 2003; 2.
5. Hidalgo JA, Pott Disease, 2012, Available from:http://emedicine.medscape. com /article/226141-overview# showall, (Accessed on 12.02.15)
6. Sharif HS, Role of MR imaging in the management of the spinal infections. AJR 1992;158:1333-45.
7. Jung NY, Jee WH, Ha KY, Park CK and Byun, JY. Discrimination of Tuberculous Spondylitis from Pyogenic Spondylitis on MRI. AJR 2004;182:1405-10.
8. Khalequzzaman S and Hoque HW, Tuberculosis of Spine Magnetic Resonance Imaging (MRI) Evaluation of 42 Cases. Medicine today 2012; 24: 53-62.
9. Maeda Y, Izawa K, Nabeshima T and Yonenobu K , Tuberculous spondylitis in elderly japanese patients. J Orthop Sci 2008;13: 16–20.
10. Cormican L, Hammal R, Messenger J and Milburn H , Current difficulties in the diagnosis and management of spinal tuberculosis. Postgrad Med J 2006;82: 46–51.
11. Ogle JW, Wilson FC and Mcconnachie CC , Angular kyphosis as an indicator of the prevalence of pott's disease in Transkei. S Afr Med J 1994;84: 614–8.
12. Alvi AA, Raees A, Rehmani MAK, Aslam HM, Saleem S and Ashraf J, 'Magnetic Resonance Image findings of Spinal Tuberclosis at first presentation. International Archives Med 2014; 7: 2-7.
13. Loke TK, Ma HTG and Chan CS, Magnetic Resonance Imaging of the tuberculous spinal infection. Australas Radiol 1997; 1: 7-12.
14. Bajwa GR , Evaluation of the role of MRI in spinal Tuberclosis: A study of 60 cases. Park J Med Sci 2009; 25; 6: 944-7.
15. Hasan, NMA , Pedicle involvement in tuberculous spondylitis and pyogenic spondylitis: Comparative magnetic resonance imaging study. Egyptian J Radiol Nuclear Med 2014;45: 455-60.
16. National survey of tuberculosis notifications in England and Wales in 1983: characteristics of disease, 1987, 'Report from the Medical Research Council Tuberculosis and Chest Diseases Unit'. Tubercle 1983; 68: 19-32.

ORIGINAL ARTICLE

The Osmani Medical
Teachers Association Journal
Volume 15 Number 2

Prospective Comparative Study Of Operative And Non-Operative Management Of Peptic Ulcer Perforation In Selected Cases

Muhammad Mofazzal Hossain[1], AKM Shafiul Alam[2], Mohammad Mahbubul Haque[3], Syed Abul Foez [4], Showkat Uddin Ahmed[5], Abdullah Md. Abu Ayub Ansary[6], Ranjon Kumer Roy[7]

Abstract

This prospective clinical trial was carried out to compare the outcome between non-operative and operative management of perforated peptic ulcer. A total of 100 such cases were included in the study, out of them 49 received non-operative treatment and 51 operative treatment. The mean age 36.3 ± 12.1 years with male and female ratio being 24:1 in non-operative group and 6:1 in operative group. 25.5% of the patients in the non-operative group got admitted in less than 6 hours and 52.9% between 6 – 12 hours of onset of pain, while in operative group nearly 64% admitted in the hospital 12 hours after onset of pain. The difference between the groups with respect to time lapsed between onset of pain and admission was statistically significant ($p < 0.001$). About 60% the former group did not take anything orally after perforation. In the operative group majority (81.6%) received some amount of food and drinks and oral medications. The pulse, systolic and diastolic blood pressures and respiratory rate were significantly stable in the non-operative group than those in the operative group ($p<0.001$, $p<0.001$, $p=0.002$ and $p<0.001$ respectively). Nearly half (49%) of the non-operative group exhibited mild dehydration, 41.3% moderate dehydration and 7.8% severe dehydration. In contrast, 42.9% of the operative group had moderate dehydration and 57.1% severe dehydration. The difference between the groups in terms of urine output in 1st 6 hours of observation was statistically significant ($p < 0.001$). Hospital stay was significantly less in non-operative group than that in operative group (6.7 vs. 10.3 days, $p < 0.001$). Recovery does not depend on age of the patients ($p = 0.950$) and the sex also did not influence outcome (86.8% vs. 77.8%, $p = 0.809$). Uneventful recovery was significantly less in patients who delayed in getting admitted to the hospital compared to those who presented earlier ($p < 0.001$). Intake of anything orally after perforation was also observed as a barrier to uneventful recovery ($p < 0.001$).

[OMTAJ 2016; 15(2)]

1. Registrar, Department of Surgery, 300 Beded Hospital, Narayangonj.
2. Senior consultant, Department of Surgery, 300 Beded Hospital, Narayangonj.
3. Senior consultant, Department of Medicine, 300 Beded Hospital, Narayangonj.
4. Assistant Professor, Department of Hepatology, Sylhet MAG Osmani Medical College.
5. Junior Consultant (Surgery), UHC, South Surma, Sylhet.
6. Resident Surgeon, Shaheed Suhrawardy Medical College Hospital.
7. Assistant Professor, Department of Medicine, Sylhet MAG Osmani Medical College.

Introduction

Peptic ulcer disease (PUD) is a very common abdominal condition especially in middle aged male[1]. Because of changing pattern of life styles and dietary habits the incidence is increasing in this subcontinent. More over smoking and use of certain drugs such as NSAIDs, steroids also play role as aggravating factor.[2] Duodenal ulcer perforation is one of the gravest acute abdominal condition among its complications like peritonitis, septicaemia, gross water and electrolyte imbalance which increases the mortality and morbidity of the patient unless treated energetically.[3] Early diagnosis is important, as delayed diagnosis usually results in intraperitoneal sepsis.[4]

Despite dramatic improvements in peptic ulcer management in the last two decades the frequency of emergency surgery for perforated gastro-duodenal ulcer remains stable or little changed.[5] A number of opinions have been put forward in the management of duodenal perforation. Most authorities recommend surgical closure of the perforation.[6] But it has been well established that non-operative management often

named as conservative management with nasogastric suction, circulatory support, anti-secretory drugs and antibiotics can be an effective treatment of perforated ulcer[7]; Non-surgical treatment of the patient with self-sealing can be undertaken with the assurance that the seal will be secure and that the incidence of septic intra-abdominal complications will be very low. This option is particularly attractive in a case considered to be at high surgical risk because of age and/or associated disease. A surgical procedure performed to close an already-sealed perforation is unnecessary. Repeated clinical examinations to assure early progressive resolution of evidence of peritonitis are mandatory. If the physician is unable to conduct such examinations, non-surgical treatment is contraindicated[8]. Although each case must be individualized and not withstanding the foregoing, if a Gastrografin upper gastrointestinal series shows continuing free perforation despite the best conservative management, surgery may well become indicated.[9]

MATERIALS AND METHODS

This prospective clinical trial were conducted in surgery department of Sylhet MAG Osmani Medical College Hospital from August 2006 to December 2008. Age more than 18 years irrespective of sexes, early or late presentation but comparatively soft abdomen with minimum tenderness and distension and haemodynamically stable patients who responds to initial six hours treatment were included. Whereas similar illness following fever, suspected perforation with extreme shock, rigid abdomen and significant tenderness who did not respond to initial management, concurrent any other illness specially diabetes, any confusion in diagnosis, perforations due to trauma were excluded. Data presented on categorical scale were compared between groups with the help of Chi-square (χ^2) or Fisher's Exact Probability Test and quantitative data were compared between groups using Student t-Test.

RESULTS

Out of 100 cases of perforated peptic ulcers 51 received non-operative treatment and 49 operative treatment. There is no significant difference was observed between the groups in terms of age ($p = 0.698$). Majority of the patients in both groups (98%) informed that pain commenced in empty stomach ($p = 0.742$). In non-operative group over one-quarter (25.5%) of the patients admitted in less than 6 hours and 52.9% between 6 – 12 hours of onset of pain, while in operative group nearly 64% admitted in the hospital 12 hours after onset of pain and statistically significant ($p < 0.001$).

Table I. Comparison of clinical characteristics between groups

Clinical presentation	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
Vomiting#	12(23.5)	7(14.3)	0.239
H/O taking NSAIDs*	2(3.9)	5(10.2)	0.202
Muscle guard/rigidity *	2(3.9)	5(10.2)	0.202
H/O peptic ulcer disease#	50(98.0)	41(83.7)	0.013

Data were analysed using **Chi-square (χ^2)**; data were analysed using **Fisher's Exact Test**
Figures in the parentheses denote corresponding **percentage.**

Table II. Comparison of clinical characteristics between groups (n = 100)

Clinical characteristics	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
Distension			
Mild	29(56.9)	0(0.0)	
Moderate	21(41.2)	32(65.3)	< 0.001
Severe	1(2.0)	17(34.7)	
Fluid thrill	1(2.0)	6(12.2)	0.050
Liver dullness			
5th	16(31.4)	0(0.0)	
6th	32(62.7)	36(73.5)	< 0.001
7th	3(5.9)	13(26.5)	
Shifting dullness	20(39.2)	22(44.9)	0.565
Bowel sound	40(78.4)	28(57.1)	0.023

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

In the non-operative group about 60% of the patients did not take anything orally after perforation of peptic ulcer, 37.2% took limited amount of food and drinks and 5.9% took everything liberally. In the operative group majority (81.6%) received limited amount of food and drinks, 16.7% received food and drinks without restriction and only 2% did take nothing. As muscle guard/rigidity was compared between groups, it was observed that in 80.6% of the cases of non-operative group it was restricted to upper abdomen and subsided with time, while in operative group majority (77.6%) of the cases exhibited generalized rigidity at initial presentation. In 19.4% of cases of non-operative group exhibited generalized rigidity but that gradually subsided and restricted to upper abdomen within two hours of initial management. On the other hand 22.4% of the cases of operative group muscle guard/rigidity was restricted to upper abdomen but they were haemodynamically unstable so that they were allocated to operative group. Comparison of haemodynamic state shows that pulse, systolic and diastolic blood pressures and respiratory rate were significantly stable in the non-operative group than those in the operative group ($p < 0.001$, $p < 0.001$, $p = 0.002$ and $p < 0.001$ respectively). Nearly half (49%) of the non-operative group exhibited mild dehydration, 41.3% moderate dehydration and 7.8% severe dehydration. In contrast, 42.9% of operative group had moderate dehydration and 57.1% severe dehydration.

Table III. Comparison of urine output 6 hours between groups (n = 100)

Urine output in 6 hours	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
≤ 300 ml	1(2.0)	16(32.7)	
301 – 600 ml	41(80.4)	33(67.3)	< 0.001
> 600 ml	9(17.6)	0(0.0)	

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

Plain X-ray of abdomen in erect posture shows that over half (51%) of the non-operative group had small amount of free gas under diaphragm, while majority (73.5%) of the operative group exhibited moderate and 26.5% huge amount of gas under diaphragm ($p < 0.001$).

Table IV. Comparison of outcome between groups

Outcome	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
Cured	45(88.2)	41(83.7)	0.511
Complications developed	6(11.8)	8(16.3)	

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

Table V. Comparison of type of complications between groups

Type of complications	Group		p-value
	Non-operative (n = 6)	Operative (n = 8)	
Residual abscess	6(100.0)	2(25.0)	0.009
Wound infection	0(0.0)	6(75.0)	

Data were analysed using **Fisher's Exact Test.**
Figures in the parentheses denote corresponding **percentage.**
Hospital stay was significantly less in non-operative group than that in operative group (6.7 vs. 10.3 days, $p < 0.001$)

DISCUSSION

Perforation is the second most important and dramatic complication of PUD that deserves immediate treatment[10]. Once the diagnosis of perforation has been made, it is generally agreed that emergency surgery should be performed[11]. Conservative treatment is reserved for patients considered to be too ill to stand the stress of surgery[12]. However, controversies surround the issue and surgeons sometimes confuse to take decision which treatment options would be correct in an individual patient. The main reason that confuses a surgeon is that in many cases when abdomen is opened for surgical repair and peritoneal toileting, the perforation has already been sealed. This phenomenon convinced us to undertake the present study to see whether there is any difference in outcome between conservative and

operative management and, if so, what factors influence the outcome.

The present study did not find any difference in outcome of treatment between the two groups because non-operative treatment was continued if patient is adult>18 years, symptoms are mild and when patients were responds favorably. Majority of the subjects in either group experienced uneventful recovery and most of them were followed up to three months. Only 14 cases developed complications, and factors like delayed presentation (>12 hours of time lapsed between onset of pain and admission) and liberal intake of foods and drinks after perforation were found to influence the outcome irrespective of mode of treatment. The age and sex of the patients were not observed to be the determinants of outcome.

However, a retrospective study conducted in the Department of Surgery of Khulna Medical College Hospital, Bangladesh on 491 patients of perforated peptic ulcer from July 1992 to November 2002 put different experience. In that study two options of treatment were carried out: simple closure and peritoneal lavage in 364 cases, and 127 patients managed by non-surgical methods. The outcome evaluation showed that mortality in the surgical group was significantly higher (6.8%) than that in the non-surgical group (0.02%) ($p< 0.001$). This results go in favor of non-operative management of perforated peptic ulcer and in case if the patient does not respond to conservative treatment and deteriorates, surgical treatment can be tried.[13]

Although it was documented as early as 1843 that gastroduodenal ulcers tend to seal spontaneously,[14] it was not until 1935 that Wangensteen[15] reported the success of non-operative management of patients of perforated peptic ulcer. A review of cases in 1963 demonstrated that up to 80% of patients could be managed nonoperatively.[16] Despite numerous reports of success with non-operative management, surgical intervention became the treatment of choice for perforated peptic ulcer disease because prognosis is relatively certain.[17]

Since that time, there have been 2 prospective randomized controlled trials and 3 prospective validation studies on the utility of non-operative management of perforated peptic ulcer disease. A prospective randomized controlled trial by Cocks et al[18] in 1989 demonstrated successful non-operative management in 79 (68%) of 116 patients with perforated peptic ulcer disease. This was further substantiated by a prospective randomized controlled trial by Croft et al[19] of 83 patients in which 72% in the non-operative group were successfully managed without surgery and had equivalent morbidity and mortality rates compared with the operative group. These studies have been validated in 3 further prospective uncontrolled trials by Marshall et al,[20] Gul et al,[21] and Songne et al.[22]

In our study all the patients of non-operative group were successfully managed. No one required surgical intervention. The reason of such a successful outcome may be that the patients of this group had more stable haemodynamic state than that of operative group. Hydration status was also comparatively good and urine output in first 6 hours of observation was adequate. Moreover, majority of the patients in the non-operative group were admitted in hospital within 12 hours and without generalized peritonitis, they took nothing or limited amount of food or drinks after onset of perforation.

Thus the success of non-operative management of perforated peptic ulcer lies in careful selection of patients. As pulse, blood pressure, hydration, urine output, extent of peritonitis and early presentation (<12 hours) are very much influenced by the severity of illness, they act as prognostic signs in decision making. Besides these, there are several other risk factors for failure of non-operative management like intake of food and fluid following incidence of perforation increase the extent of peritonitis. Here the role of primary health care personnel is important in the sense that they can give them proper advice not to take anything by mouth and refer immediately to nearby hospital where facilities for proper management exists. Thus we can avoid the operative assault of the patient, minimize the workload of the hospital, hospital stay and cost of the patient for treatment.

In conclusion, from the findings of the study and discussion it could be concluded that there is no significant difference in the outcome of treatment between non-operative and operative management of perforated peptic ulcers in selected cases. The findings of the study suggest that non-operative treatment should be started in almost all adult patients who are haemodynamically stable and present early in hospital with minimal signs and symptoms. If the condition deteriorates or not responds to non-operative management in few hours of observation, immediate surgical intervention should be considered.

The study has got some limitation including non randomized trial and limited size of sample. A fare and randomized trial with a bigger sample size to recommend the criteria and formulate a scoring system for the management of the patients suffering from peptic ulcer perforation is suggested.

References

1. John NP. Stomach and Duodenum. In: R.C.G. Russell, Norman S. Williams & Christopher J.K. Bulstrode, editors. Bailey & Love's Short Practice of Surgery. 24th Edition. London: ARNOLD; 2004. p. 1026-61.
2. Gupta PKS and Azad MR. Trends of clinical presentation of peptic ulcer disease at DNMIH in recent years. Med J 1996; 32:10-4.
3. Miah MAG, Selim S, Islam S. Intestinal perforation – a clinical study in district hospital. Mymensingh Med J 1999; 8:18-21.
4. Cheefook C, Lawrence J. Conservative management of duodenal perforation following endoscopic sphincterotomy Digestive Endoscopy 2005;17:168–71.
5. Bucher P, Wassila O, Philippe M, Frederic R, Olivier H. Results of conservative treatment for perforated gastroduodenal ulcer in patients not eligible for surgical repair. Swiss Med WKLY 2007;137:337-40.
6. Graham RR. The treatment of perforated duodenal ulcers. Surg Gynecol Obstet.1937;64:235-8.
7. Crofts TJ, Park KG, Steele RJ, Chung SS, Li AK. A randomized trial of nonoperative treatment for perforated peptic ulcer. N Engl J Med 1989; 321:1050-1.
8. Arthur JD, Thomas VB, John AD. Perforated Duodenal Ulcer: An Alternative Therapeutic Plan. Arch Surg 1988;33:133-7.
9. Jamieson GG. Current Status of Indications for Surgery in Peptic Ulcer Disease. World J Surg 2000; 24:256–8.
10. Matthias H. Seelig, Stefanie KS, Christian Behr and Klaus Schonleben Comparison Between Open and Laparoscopic Technique in the Management of Perforated Gastroduodenal Ulcers. J Clin Gastroenterol 2003; 37:226–9.
11. Tanphiphat C, Tanprayoon T. Surgical Treatment of perforated duodenal ulcer: a prospective trial between simple closure and definitive surgery. Br J Surg 1985; 72:70-2.
12. Keane E, Dillon B. Conservative management of perforated duodenal ulcer. Br J Surg 1988; 75:583-4.
13. Rahuman MM, Saha AK, Rahim A. Experience of peptic ulcer perforation over a decade in a teaching hospital of southern Bangladesh. Ceylon Med J 2003;48:53-5.
14. Crisp E. Cases of perforation of the stomach with deductions there from relative to the character and treatment of that lesion. Lancet 2009; 2:639.
15. Wangensteen OH. Nonoperative treatment of localized perforations of the duodenum. Minn Med 1935;18:477-80.
16. Fontaine R, Hiebel G, Lang G, Barth G. Continuous aspiration by Taylor's method for gastroduodenal ulcer perforated into the free peritoneum: complementary statistics from July, 1953 to the end of 1962 [in French]. Lyon Chir. 1963;59:682-95.
17. Lau WY, Leow CK. History of perforated duodenal and gastric ulcers. World J Surg 1997;21:890-6.
18. Cocks JR, Kernutt RH, Sinclair GW, Dawson JH, Hong BH. Perforated peptic ulcer: a deliberative approach. Aust N Z J Surg. 1989; 59:379-85.
19. Crofts TJ, Park KG, Steele RJ, Chung SS, Li AK. A randomized trial of non-operative treatment for perforated peptic ulcer. N Engl J Med 1989; 320:970-3.
20. Marshall C, Ramaswamy P, Bergi FG, Rosenberg IL, Leaper DJ. Evaluation of a protocol for the non-operative management of perforated peptic ulcer. Br J Surg. 1999; 86:131-4.
21. Gul YA, Shine MF, Lennon F. Non-operative management of perforated duodenal ulcer. Ir J Med Sci. 1999; 168:254-6.
22. Songne B, Jean F, Foulatier O, Khalil H, Scotte M. Non-operative treatment for perforated peptic ulcer: results of a prospective study. Ann Chir 2004; 129:578-82.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal
Volume 15 Number 2

Problems identified and perceived benefits of post natal care

Samira Haque[1], Ferdousi Begum,[2] Lipy Bakshi[3] , Afroza Akhter[4], Fayeza Karim[5], Farida Yeasmin[6]

Abstract

The objective of the study was to determine post natal problems and patient's perception of benefit of post natal care. A cross sectional study was conducted in Ad-din women's medical college and hospital on one hundred patients attending post natal care center for various reason from July to December 2011. They had been selected purposively.Most of the mothers belong to 21-30 year of age. Among them, maximum completed higher secondary study. Mode of delivery was most of cases by caesarian section. The morbidities found were lower abdominal pain (16%), wound infection (11%) and breast engorgement (9%) of mothers. In case of neonate common problems were neonatal jaundice (11%) and umbilical sepsis (8%). Twenty one percent mothers and fifty two percent neonates were found healthy. Regarding perception of benefit of post natal care, mothers expressed that they were benefited by getting routine checkup, treatment for their illness, and vitamin A capsule. Both the mothers and newborns were benefited by receiving post natal care in terms of maintenance of health and treatment of complications. Efforts should be enhanced to ensure post natal care for all mothers and newborns.

[OMTAJ 2016; 15(2)]

Introduction

The puerperium (postpartum period), generally lasts 6 weeks and it is the period of adjustment after delivery when the anatomic and physiologic changes of pregnancy are reversed and the body returns to the normal non pregnant state[1] Globally, approximately four million babies die annually in the first 28 days of life. Neonatal mortality now accounts for approximately two-thirds of all infant mortality and 38% of deaths of children under five mortality[2] Ninety-nine percent of these occur in middle and low-income countries[3] Puerperal infectious morbidity affects 2-8% of pregnant women and are among the most prominent puerperal complications. These complications are more common in those of low socio-economic status, those who have undergone operative delivery, those with premature rupture of the membranes, those with prolonged labors and those with repeated vaginal examination during labour.[4]
Maternal mortality in Bangladesh is 194 in 1,00,000 live birth [5]. Again majority of maternal and neonatal deaths and complications occur during the postpartum period and over 90% of deliveries take place in the home and most attended by untrained providers. By the end of the first week postpartum more than two-thirds of all maternal deaths and two-thirds of all neonatal deaths have occurred.[6]

Post natal care is an important programme to ensure healthy mother and neonates and for identification and treatment of complications. Efforts are needed to improve the use of this programme by advocacy, awareness raising and ensuring the service provision. The present study was undertaken to evidence of the problems of mother and child in post natal period and perceived benefits of post natal care by the mothers.

Materials and Methods

A cross- sectional study was carried out in the dept. of Obstetrics and gynaecology in Ad-din Women's Medical college Hospital located in Moghbazar which is a tertiary care hospital, has a separate centre for post natal care.

The study was carried on during the period of July 2011 to December 2011 on one hundred mother at

1. Assistant Professor, Obstetrics and Gynaecology, Aichi medical college
2. Professor,Obstetrics and Gynaecology, Ibrahim Medical College
3. Registrar,Obstetrics and Gynaecology, Dhaka National Medical College
4. Lt. Colonel,Obstetrics and Gynaecology, CMH, Sylhet
5. Assistant Professor, Physiology, Aichi medical college
6. Assistant Professor, Anatomy, Tairunnessa Memorial Medical College

post natal period those were purposively selected among the mothers presenting within 1st day – 6wks after delivery in PNC centre of Ad-din Women's Medical college Hospital. Data were collected by interviewing the mothers using structured, pre-tested questionnaire.

Both primi and multi who delivered by normal vaginal delivery or caesarean section &the neonates of 42 days were included in this study. Patient with known medical diseases, with history of infection, morbidities prior to delivery & who did not agree to participate in the study were excluded.

RESULT

Mean age of post natal mothers are 24.53 and SD is 4.36 .Majority of the patients belongs to age group 21 to 30 years and 68% patients belongs to age group 20.17 to 28.89 years. 56% of mother completed higher secondary education, 31% of post natal

Table I: Geographical distributions

Age group(year)	Percentage (%)
<=20	21
21-30	68
>30	11
Mean age= 24.53 years	Mean+ SD =24.53+ 4.36
Educational status	
Illiterate	2
Primary	11
Secondary	31
Higher secondary & above	56
Nutritional status	
Got adequate nutrition – Yes	71
No	29
Took adequate rest – Yes	70
No	30

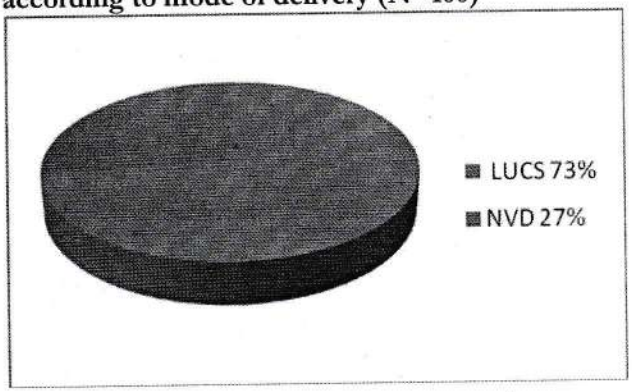
mother completed secondary education, 11% mothers up to primary level and only 2% are illiterate. Only 28% of mothers were employed.71% mother got adequate nutrition and 70% of post natal mother got adequate rest .

Table: II Distribution of postnatal mother according to parity (N=100)

Parity(no)	Percentage (%)
1	64
2	27
3	6
4	3
Mean parity 2.5	Mean+SD=2.5+1.29

Majority mothers belongs to pirmipara with mean parity was 2.5 and SD was1.29 and 73% mothers delivered their baby by LUCS .

Figure: I Distribution of post natal mother according to mode of delivery (N=100)



So it seems that the mothers who underwent caesarean section are more interested to come to postnatal care in comparison to those who had normal vaginal delivery.

74% patient had no complain in immediate past pregnancy. Most common complication among rest of the patient was Preeclamptic Toxaemia (PET) (10%), second most common complication was Premature Rupture of Membrane (PROM) and oligohydramnios was found in 5%. Anti Partum Haemorrhage (APH) and Gestational Diabetes Mellitus (GDM) contributed 1% of the pregnancy. Only 1 patient present with Intrauterine Death (IUD) who delivered a dead male baby. There was two twin female babies.

99% of postnatal mother delivered live born healthy babies. Among them 53% were female and 48% were male baby. There were 2 female twins and 1% was male dead baby. The common morbidities that was observed in post natal mothers were lower abdominal pain 16%, wound infection 11%, breast engorgement 9%, Peptic ulcer disease 8%, urinary tract infection 6%, constipation 4%, diarrhoea 4%, cracked nipple 3%, fever 3%, respiratory tract infection 2%, hypertension 2%, low back pain 1%, headache 1%, pain in episiotomy wound 1%, allergic reaction 1%. Multiple morbidities were present in 9 patients. 22 mothers had no complication.

Table: III Problems of mother & child in post natal period (N=100)

Problem(mother)	Number	Percentage (%)
Lower abdominal pain/ pain in incision area	17	16
Wound infection	12	11
Breast engorge	10	9
Peptic ulcer disease	9	8
Urinary tract infection	7	6
Diarrhoea	5	4
Constipation	5	4
Fever	4	3
Crack nipple	4	3
Respiratory tract infection	3	2
Hypertension	3	2
Pain in episiotomy wound	2	1
Spinal headache	2	1
Low back pain	2	1
Allergic reaction	2	1
No complication	22	21
Problem		
Low birth weight	21	
Jaundice	11	
Umbilical sepsis	8	
Respiratory distress	4	
Common cold	3	
Fever	1	
Whole body rash	1	
No complication	52	

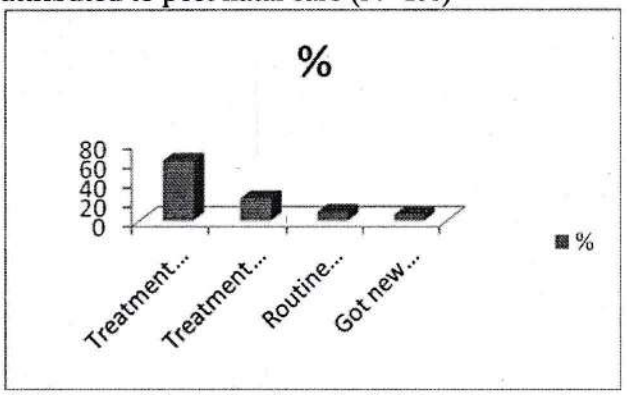
Table: IV type of services received during postnatal care (N=100)

Source	Percentage (%)
Routine history taking & counseling of mother	99
Routine history taking & counseling on baby	95
Information about care of new born	94
Vitamin A capsule	99

52% of neonates were present in post natal care center with no complication. Most common problems were low birth weight 21%, neonatal jaundice 11%.Umbilical sepsis 8%, respiratory tract infection 4%. 3% presented with common cold, 1% with fever and 1% with rash. One neonate had multiple complications.

Routine history was taken from 99% post natal mother and counseled them about their health, 95% mother counseled on baby, 94% mother got information about care of new born, 99% mother got Vitamin A capsule, 1 mother did not got Vitamin A capsule because she delivered a dead babyFigure II shows 42% mother got information about post natal care from doctor or nurse during delivery, 31% mother were informed from doctor or nurse during ANC, rest of them received information on PNC from other source such as relatives, neighbours, friends etcFigure III shows 52% mother went for checkup as they were sick in post natal period. 19% for their babies as they were sick, 11% mother went to PNC for routine checkup as somebody told for seeking post natal checkup and 4% went for other reason such as information about their health, new born care, to get Vitamin A capsule , 21 patients goes for post natal checkup as both mother and baby were sick.

Figure II: Perception of mothers about benefits attributed to post natal care (N=100)



62% postnatal mother benefited from post natal care by getting treatment for their illness, among them 12% post natal mother got dressing for their wound infection, 23% mother got treatment for their neonates, 9% mother benefited by checkup for them and their new born. 6% mother got information about health care for both mother and their new born, also benefited by getting Vitamin A capsule. 17 mothers received multiple benefits.

DISCUSSION

This study was conducted in Obstetrics and Gynaecology department of Ad-din Women medical college, to evaluate the problems of post natal period of both mother and neonates, and perceived benefits of post natal care. The study was conducted over a period of six month (July 2011-Dec 2011) and 100 patients who visited at post natal care center for various reasons were selected.

In the study, 68% of the post natal mother belongs to age group of 21-30 years and above 30 year the percentage was 11%. In a study by JHPN showed that 62.3% mothers were in the age group of 20-34 years.[7] Another study by Tanzina on post natal morbidities showed the age incidence was revealed that 55% of cases were in the age group of 21-30 year and 15% mother belongs to age group >30 years[8] that was almost consistent with present study.

In this study 56% mother were educated up to higher secondary and above, only 2% was illiterate. This results revealed that educated mother were more conscious about post natal check up; whereas only 10% mother were from middle class family with higher education and 70% were from lower class family in a study conducted on 2007 on post natal problem.[8] That is percentage of educated mother rise almost 4 fold who were availing post natal care.

Inspite of good proportion of higher educated mother, the employment status was no so higher. Only 28% post natal mother were employed.

Analyzing the nutritional status of mother during pregnancy it was revealed that 71% mother got adequate nutrition and 70% mothers got adequate rest. In a precursory study on link between nutrition and pregnancy in 1950 women who consumed minimal amounts had a higher mortality or disorder rate concerning their offspring than who ate regularly.[9]

In this study, about 97% mothers are belongs to para 1-3 among them primipara were 64%. A study on post natal complication by Tanzina, about 90% patients belongs to para 1-3.[8] This observation almost similar with the findings of Akter who has worked on patients of early puerperal complication in Dhaka medical college and Sir Salimullah medical college and hospital.[10]

The study revealed that 73% mother delivered by LUCS whereby NVD 27% . The survey report BDHS 2011 showed that c-section increased from 52% (2010) to 60 %(2011*)*. So the study finding is increase percentage of C section indicate women access skill care for complicated delivery and more percentages of caesarian section, more complication in post natal period.

Most common complication during pregnancy was PET which was about 10%, Second most complication was PROM 5%, and oligohydramnios 5%, UTI not also uncommon which is 3%. 74% pregnancies were uncomplicated.

In this study 99% mother delivered healthy live baby among them 53% were female baby and 48% male baby. Male female ratio is almost 1:1. There was a dead male baby and 2 twin female babies.

The most commonpost natal complications were lower abdominal pain or pain in incision area which were 16%. A study on post natal complication showed in Bangladesh 19% post natal mothers suffering from lower abdominal pain.[11] This findings was consistent with present study. Then in this study wound infection present on 11% of post natal mother. A study showed the common morbidities were wound infection in 10% patient[8] similar finding with present study. Another study showed that 20-40% of mother had post caesarian complications including infection of uterus, wound infection or UTI. Caesarian mother are twice as likely to have severe complication compared to vaginal delivery.[12] In this study the incidence of wound infection are quite higher in comparison to many other studies of developed countries, where the incidence of infectious morbidities varies between 2-8% [4] and very common in those who have undergone operative delivery. [5]Post partum infection are responsible for much morbidities associated with child birth and contribute to death about 8% of all pregnant women in each year.[2]

Another quite common morbidity was breast problem including breast engorgement 9% and cracked nipple 3%. In the Grampian study 33% of all women experienced breast problem in 1st 2 weeks of post partum and 28% in the weeks there after. [24] In this study breast problem was lowerer than other study due to possibly effective breast feeding practice.

UTI was another complication that was observed in present study and it contributes 6% of post natal mother. A study revealed 2-4% women develop post partum UTI.[2] Another study by Tanzina showed 7% mother developed post partum UTI, [8] which is similar to present study. The percentage of UTI in post partum is increasing due to catheterization.[13] UTI in post natal period is common in caesarian section showed in a study by Schwartz MA.[14]

This study showed 1% post natal mother suffered from spinal headache. Birmingham sample reported 3.6% patient had frequent headache. There was a relation between headaches with accidental dural puncture.[15] In present study the percentage was slightly lower than other study possibly due to skilled practice of anesthesia. Another complains was backache that contribute 1% in this study. In

Birmingham the percentage was 14% and in Grampian studies more than 20%. These were association with caesarian section.[16, 17]

1% postnatal mother complains of pain in episiotomy wound or perineal pain. In Egypt 2.1% of women reported dyspareunia after child birth and in Grampian study 8% women experience perineal pain among them 16% in primipara[18]

Use of absorbable suture material can reduce short term perineal complaints[19,20,21] that's why there was low rate of episiotomy wound pain in present study. Constipation was another problem in post natal period. In this study it contributed about 5%. In Grampian study the rate is quite higher 7%[22,23,24] which almost consistent with present study.

In this study other morbidity was PUD 8%, Diarrhoea 4%, Fever 3%, respiratory tract infection 2%, hypertension 2%, allergic reaction1%, and 9 patients complains of multiple morbidities. 22 mothers had no complication.

In our study there was no case of Vesicovaginal Fistula (VVF) which is more common in developing world similar with the study conducted by Tanzina.[8] It was due to now there was early management of obstructed labour so chance of developing VVF is less. Also there was no case of thromboembolism which is a common occurrence in developed world. In other studies conducted in this country, no case of thromboembolism has been recorded.[8]

According to the morbidities of new born in post natal period the most common morbidities were low birth weight which is about 21%. A study showed, in Bangladesh low birth weight (LBW) rate is quite high (36%), more common in low socio economic group. In present study most of the patient from middle class family that's why rate of LBW was lower than other study. [25]

Another common complication was neonatal Jaundice that contributes 11%. A study on post natal complication showed the percentage was 15%.[11] It was more or less similar with this study. In this study 8% new born present with umbilical sepsis. A study revealed in developed countries the incidence is 1-10 in 1000pre term infants.[11] In the present study the percentage was high due to Bangladesh is a developing country and all the health facilities are not adequate in developing countries. Another complication was respiratory distress 4%, common cold 3%, Fever 1%, whole body rash 1%, 2 new bon had multiple complication. 56% new born were healthy.

99% postnatal mother counseled about health care of mother also routine history taking and checkup was done. 95% mother counseled on their new born, routine checkup of baby was done, and 94% mother got information about care of new born, 99% post natal mother got vitamin A capsule 1 mother didn't get because she delivered a dead baby. The rate of supplement of vitamin A capsule is very high because in Ad-din hospital every post natal mother who came to post natal checkup must administered with 200000 IU of vitamin A capsule.

42% mother got information about post natal care from health care providers (Doctor and Nurse) during delivery. Now a days 32% delivery occurs by TBA,[26] so they give information about PNC during delivery. 31% post natal mother got information during ANC from doctor or nurse. A study showed 30% mother received ANC regular or irregularly,[8] so from there they got the information.

In this study majority post natal mother came for post natal check up due to they were sick in post natal period, 19% mother came due to their babies were sick. 11% mother came for routine checkup as somebody told for seeking post natal checkup and 4% goes for other reason such as information about their health, new born care, to get Vitamin A capsule, 21% came for multiple reasons.

There were many more benefits of post natal checkup. But according to the mothers the perception attributed to post natal care was 62% post natal mother got adequate treatment for them illness, 23% mother thought instead of post natal checkup they couldn't get treatment for the new born. Only 9% post natal mother perceived that they and their babies were thoroughly checked up and identified their problems. 6% mother said that they had no information about health care of both mother and their new born before coming for post natal checkup, and also got vitamin A capsule which they thought quite beneficial for them and their new born.

In conclusion, post natal care is important and the post natal mothers are benefited by post natal care in terms of information and treatment of complication. It is surprising that postpartum care has received so little attention compared with antenatal and intrapartum care. Most of the mother didn't feel necessity for regular post natal checkup, 52% mother came in post natal care center due to their sickness, not for routine checkup. All patients told that they were extremely benefited after coming for post natal checkup. They

have received treatment for mother and babies, got vitamin A capsules. But before then they were unaware about the benefits. All mother should counseled about regular post natal checkup. Every maternity hospital should have separate ante natal and post natal care center Research should be carried out to indentify the justification of increasing caesarian section day by day. Comparative study may done to find out the rate of LBP and headache after epidural and spinal anaesthesia.

References

1. Novy J. Lipsconb K. The normal puerperium. InDecherney A H, Nathan L, eds. Current obstetrics and Gynaecologic Diagnosis and Treatment 9th Eds.New York, MC Graw Hill; 2003.Pp234-58.
2. Saving new born lives. Sate of the world's new borns. Washingtong DC, Save the children-USA. 2001(1):1-49.
3. Lawn JE, Cousens S Zupam J. 4 million neonatal death: When? Where? Why? Lancet 2005;365:891-900.
4. Poggi BH. Kapernick S. Postpartum Haemarrhage& the abnormal Puerperium. InDecherney AH, Nathan L eds. Current obstetrics and Gynaecologic Diagnosis and Treatment 9thEdn. New York, MC Graw Hill. 2003: pp. 531-52.
5. Anonymous. Bangladesh Maternal Mortality and Health care survey (BMMs),2010(2);86-7
6. Kabir A. Country fact file on maternal and child health situation in Bangladesh, Dhaka.Jhon Snow international research and training institute 2004(4) :03-04.
7. JHPN. Study in PNC in Bangladesh2006, Descember,24(4);506-518.
8. Sultana T, Early puerperal complication following emergency caesarian section , A clinical study in Comilla Medical College Hospital. Comilla. J BCPS 2007; 32;33-6.
9. RasmussanKM .The influence of maternal Nutrition on lactation Annual Review of Nutrition.2012(12) 103-10,114
10. AkterT.Early puerperal complication. A prospective study on caesarian deliveries in DMCH, SSMC and Mitford Hospital.(D BCPS L3556) 2004;41-44.
11. Safe mother hood, Post purtum care of the mother and new born: a practical guide report of a technical working group .WHO/RHT/MSM/ 1998(3);1-16.
12. National child birth VII: C –section risk and complication September,2011(29):5-6.
13. Gerber B, Schnidt H, Ohde A. Diagnosis of Urinary tract infection in puerperium. Universitals Fravenkilnk, Roskoch. Glburtshilfe Frauenheilked 1994 54:524-28
14. Schwartz MA. Wang CC, Eckert L, Cirtchlow CW. Risk factors for urinary tract infection in the post partum period. Am J Obstet Gynecol 1999; 1813: 547-53.
15. MacArthur C, Lewis M, Knox EG. Accidental dural puncture in obstetric patients and long term symptoms. Br Med J 1993;306:883-5.
16. MacArthur C, Lewis M, Knox EG. Investigation of long term problems after obstetric epidural anaesthesia. Br Med J 1992; 304:1279-83
17. Russel R, Groves Ph, Taub N, O'Dowd J, Reyonlds F. Assessing long term backache after childbirth. Br Med J 1993;306:1299-1303.
18. Sleep J, Grant A, Garcia J, Elbourne D, Spencer J, Chalmers I. West Berkshire perineal management trial. Br Med J 1984;289: 587-90.
19. Mahomed K, Grant AM, Ashurst H, James D. The Southmead perineal suture study.A randomized comparison of suture materials and suturing techniques for repair of perineal trauma. Br J Obstet Gynaecol 1989;96: 1272-80.
20. Isager-Sally I, Legarth J, Jacosen B, Bostofte E. Episiotomy repair immediate and long-term sequelae. A prospective randomized study of three different methods of repair.Br J Obstet Gynaecol 1986;93: 420-5.
21. Johanson RB. Absorbable vs non-absorbable sutures for perineal repair. In: Keirse MJNC, Refrew MJ, Neilson JP, Crowther C (eds.) The Coachrane and Childbirth, Database Pregnancy. The Cochrane Collaboration Issue 2, Oxford BMJ Publishing Group, London.1995(2):7-8
22. Zuspan FP. A double blind laxative study on postpartum patients.Am J Obstet Gynecol 1960; 80: 548-50.
23. Shelton MG. Standardisedsenna in the management of constipation in thepuerperium a clinical trial. S Afr Med J 1980;57: 78-80.
24. Diamond RA, Gall SA, Spellacy WN. Bisox T0. Rasmussan KM. The influence of maternal Nutrition on lactation Annual Rev Nutri 1992; 12:282-99
25. Khatun S and Rahman M. Socio economic determinants of low birth weight in Bangladesh A multivariate approach. 2008(34),81-86.
26. BDSH 2011, Bangladesh demographic and Health survey report (BHHS) 2011;6:22-4

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Comparative study of the outcome of treatment for closed comminuted unstable tibial diaphyseal fracture by Ilizarov external fixator and minimally invasive plate osteosynthesis.

Kamrul Alam,[1] Lutfor Rahman Khan,[2] M A Hannan,[3] Mohsenuzzaman Khan,[4] F M Mahfuz Anwar,[5] Mirza Omar Beg.[6]

Abstract

The purpose of the study was to know the outcome of treatment of closed comminuted unstable tibial diaphyseal fracture by Ilizarov external fixator & minimally invasive plate osteosynthesis. This cross sectional comparative study was carried out in the Department of Orthopaedic surgery at Sylhet MAG Osmani Medical College Hospital, Sylhet from 1st July 2012 to 30th June 2014. A total 12 patient presented with closed comminuted unstable tibialdiaphyseal fractures considering inclusion and exclusion criteria. Then the study sample was randomly allocated into group-A and group-B. The patients of group-A were treated with Ilizarov external fixator and patients of group-B were treated with minimally invasive plate osteosynthesis.Each of the patients was evaluated clinically and radiologically. The patients were evaluated clinically with respect to pain on full weight bearing and knelling, tenderness, abnormal mobility, infection, range of movement of knee & ankle joints and limb length discrepancy. Radiologically the patients were evaluated with respect to union of fracture, alignment, implant failure and infection. 1 patient was lost during follow up, and therefore 11 patients were available for follow up for a period of 6 months. Male was more predominant in both groups. Motor Vehicle Accident was the main mechanism of trauma. Mean duration of operation of group-A was 106.00 (SD 4.18) minutes and group-B was 85.0 (SD 4.47) minutes. Duration of operation was significantly longer in patient of group-A than that of group-B (X^2=11.000; $p<0.01$).There was no statistically significant difference of age between two groups (t=1.153; $p>0.05$), injury treatment interval (X^2=2.933; $p>0.05$), duration of hospital stay(t=1.610; $p>0.05$), duration of fracture union t=1.397; $p>0.05$, post operative infections X2=0.917; p=0.338, post-operative anterio-posterior angulation10^0 X^2=1.320; =0.251, The functional outcome (X^2=2.037; $p>0.05$), the final outcome (X^2=0.917; $p>0.05$).The functional outcome ($p>0.05$), final outcome ($p>0.05$) complications ($p>0.05$) and time of union ($p>0.05$) were almost identical in both groups by using Gustilo criteria. The duration of operation ($p<0.001$) was significantly longer in Ilizarov external fixator group. Considering the duration of operation it may be concluded that both the techniques were equally safe and effective for the treatment of closed comminuted unstable tibial diaphyseal fracture.

[OMTAJ 2016; 15(2)]

Introduction

Tibia is the most commonly fractured long bone of the body. The average age of patients with tibial shaft fracture is approximately 37 years, and teenage males are reported to have the highest incidence[1].An understanding of the diagnosis and treatment of the tibial shaft fractures is of importance to primary care physicians and orthopedic surgeons. Often the primary care provider first comes to contact with tibial shaft fracture and must take the diagnosis and early

1. Assistant Professor, Orthopaedic Surgery, Sylhet MAG Osmani Medical College
2. Professor, Orthopaedic Surgery, Sylhet MAG Osmani Medical College
3. Jr. Consultant, Orthopaedic Surgery,Gowainghatupazila health complex
4. Jr. Consultant, Orthopaedic Surgery, Golapganjupazila health complex
5. Registrar, Orthopaedic Surgery, Sylhet MAG Osmani Medical College
6. Registrar, Orthopaedic Surgery, Sylhet MAG Osmani Medical College

treatment decisions. High-speed lifestyle with motor vehicles (eg. motor cycles) as well as the growing popularity of extreme sports, contribute to the increasing occurrence of tibial shaft fractures in today's society[2]. The limited soft tissue coverage, subcutaneous location and poor vascularity render the tibial fractures very challenges to the orthopaedic surgeon. More chances of open type of fracture, greater tendency of displacement and increased chances of post-operative infection, delayed union and non union[3]. The treatment of tibial shaft fractures of all forms has been fraught with complications. Delayed union, nonunion and infection are relatively common complications of tibial shaft fractures [4].

Uniaxial external fixator is not a good option for close fracture, but it can be a useful option in open fractures with soft tissue injury. It can lead to pin-track infections, septic arthritis, mal-alignment and delayed union. Conventional plating techniques if applied to multifragmentary fractures, requires anatomic reduction, wide surgical exposure and the fractured fragments are stripped off the soft tissue attachments resulting in a variety of complications like delayed union or non-union, infections and implant failure.[5] Closed interlocking intramedullary nailing is still considered a gold standard treatment for long bone diaphyseal fractures. However, the procedure has a tendency to result in angular malunion and anterior knee pain problems when used to treat tibial fractures. Narrow medullary canals can impede nailing, and repeated reaming through this bone can cause significant thermal injury to the shaft[6]. It is difficult or impossible in comminuted fractures & not suitable for oblique fractures. Ilizarov fixator was chosen to primarily fix those fractures that produced high rate of complications with most conventional methods of fixation. The structure is stable and enables the patient to bear weight on the affected limb straight away even in much comminuted fractures, not easily achievable by other methods of fixation. The Ilizarov device is minimally invasive with little interference in the biology of fracture while at the same time providing optimal skeletal stability[7].

Recently, MIPO is widely applied with excellent results as an alternative treatment option for tibial fracture. This subcutaneous but extraperiosteal plating technique uses indirect reduction methods and thereby provides biomechanical stable construct and preserves the vascularity of the surrounding tissues and fracture haematoma[6].

Materials and Methods

This cross sectional comparative study was carried out in the Department of Orthopaedic surgery at Sylhet MAG Osmani Medical College Hospital, Sylhet from 1st July 2012 to 30th June 2014. A total 12 patient presented with closed comminuted unstable tibial diaphyseal fractures, were enrolled in this study during the study period. Inclusion criteria were Spiral, wedge, and multi-fragmentary fracture. Age of the patient in between 18 to 60 years irrespective of sex, site, side and causes of injury. Exclusion criteria were transverse, segmental, short oblique, pathological and open fracture. Fractures involved the articular surface, fractures more than three weeks old, infected fractures and fractures with bad skin condition, compartment syndrome, impaired circulation and unstable medical illness. Primary selection of patients was done on the basis of inclusion and exclusion criteria. Then the study sample was randomly allocated into group-A and group-B. The patients of group-A were treated with Ilizarov external fixator and patients of group-B were treated with minimally invasive plate osteosynthesis. Each of the patients was evaluated clinically and radiologically. The patients were evaluated clinically with respect to pain on full weight bearing and knelling, tenderness, abnormal mobility, infection, range of movement of knee & ankle joints and limb length discrepancy. Radiologically the patients were evaluated with respect to union of fracture, alignment, implant failure and infection. A total number of 12 patients were selected, of which 1 patient was lost during follow up, and therefore 11 patients were available for follow up for a period of 6 months. Half were treated by treated with Ilizarov external fixator and half were treated with minimally invasive plate osteosynthesis.

Surgical procedure:

Ilizarov External Fixator: After the selection of the cases for surgery, patient was immobilized with LLBS to relieve swelling. Surgery was avoided until the swelling subside. The patient was prepared for elective surgery to be conducted in main operation theatre. All patients received pre-operative intravenous antibiotics before 30 minutes of surgery. Under spinal anesthesia with all aseptic precaution a measured preconstructed Ilizarov assembly was inserted in the leg and manual traction applied for reduction of fracture. Some minor adjustments made according to the X-ray findings during the procedure. The positions of the rings were

checked again and final adjustments were made by adjusting the distant between rings by turning the nuts on connecting rods. Usually two 1.8 mm K-wires with bayonet tip were used to fix one ring at diaphysis & three 1.8 mm K-wires with trocher tip were used to fix one ring at metaphysis. For stabilization 3 or 4 rings were used. The most proximal ring was 5/8th in circumference so that there is adequate clearance for the knee flexion. Limbs were transfixed with K-wires by introducing it in the tibia with a low RPM electric drill machine in the safe zone. Wires were tensioned with a local made mechanical tensioner. Then wires were tightened with the ring by fixation bolts and nuts. Patients were encouraged for weight bearing immediately after the day of surgery.

MIPO: Fracture was approached by the MIPO technique, reduction done under C-arm and the fracture fragments were fixed with the locking compression plate and locking head screws. Tourniquet was then released haemostasis achieved and wound closed followed by application of above knee plaster of Paris slab. Post-operatively limb was kept elevated, antibiotics and analgesics were administered and regular assessment of the distal neurovascular status was done. Check X-rays in AP and Lateral view were taken to note the position of fragments. Patients were encouraged for isometric limb exercise immediately after the day of surgery.

RESULTS

Results of the study shown in the undermention tables

Table I. Showing age distribution of both the groups

Age group	Study group			P
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	Total (n=11) Frequency (%)	
21-30 years	2 (40.0)	1 (16.7)	3 (27.3)	
31-40 years	2 (40.0)	2 (33.3)	4 (36.4)	
41-50 years	1 (20.0)	1 (16.7)	2 (18.2)	
51-60 years	0 (0.0)	2 (33.3)	2 (18.2)	
Mean in years	33.0 (SD 11.1)	42.0 (SD 4.1)	37.9 (SD 13.1)	p>0.05

* t = 1.53

Table I showed the distribution of patients according to age. The age of patients of fracture shaft of the tibia was ranging from 22 to 60 years with the mean age of 37.9 (SD 13.1) years. The mean age of patients in group-A was 33.0 (SD 11.1) years, whereas the mean age of the patients in group-B was 42.0 (SD 14.1) years. The mean age of the patients in both groups was statistically similar (t=1.153; p>0.05).Most of the patients in group–A (80.0%) and group-B (50.0%) were at or below 40 years; indicating younger age group was more affected in fracture shaft of the tibia and there was no statistically significant difference between the age group of the patients in group-A and group-B.

Table II. Showing sex distribution of both the groups

Sex ratio	Study group	
	Group-A (n=5)	Group-B (n=6)
Male: Female	4: 1	5 : 1

Table II showed the distribution of patients according to sex. There were 4 (80.0%) male and 1 (20.0%) female with a ratio of male: female 4 :1 in the group-A; whereas 5 (83.3%) male and 2 (18.2%) female with a ratio of male : female 5 :1 in group-B.

Table III. Showing cause of injury of both the groups

Cause of injury	Study group			P
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	Total (n=11) Frequency (%)	
RTA	3 (60.0)	3 (50.0)	6 (54.5)	p>0.05
Assault	2 (40.0)	3 (50.0)	5 (45.5)	
Total	5 (100.0)	6 (100.0)	11 (100.0)	

X^2=0.110

Table III showed the distribution of the patients by cause of injury. Out of 11 patients with fracture shaft of the tibia the cause of injury was road traffic accident (RTA) in 54.5% of patients and assault in 45.5% of patients. In Group-A the cause of injury was road traffic accident (RTA) in 60.0% of patients and assault in 40.0% of patients. Whereas in the Group-B the cause of injury was road traffic accident (RTA) in 50.0% of patients and assault in 50.0% of patients. So, the cause of injury should not interfere the final outcome (X^2=0.110; p>0.05).

Table IV. Showing level of fracture of both the groups

Level of fracture	Study subjects			p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	Total (n=11) Frequency (%)	
Proximal third	3 (60.0)	0 (0.0)	3 (27.3)	p>0.05
Middle third	1 (20.0)	4 (66.7)	5 (45.5)	
Distal third	1 (20.0)	2 (33.3)	3 (27.3)	
Total	5 (100.0)	6 (100.0)	11 (100.0)	

X^2=5.084

Table IV showed the distribution of the patients according to level of fracture. Proximal third (60.0%) was relatively more involved in group-A; while middle third (66.7%) was relatively more involved in group-B. Ultimately the distribution of patients by level of fracture in the both group was almost similar (X^2=5.084; p>0.05).

Table V. Showing injury treatment interval of both the groups

Duration of fracture before surgery	Study group		p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
5-10 days	0 (0.0)	2 (33.3)	
10-15 days	4 (80.0)	2 (33.3)	
15-20 days	1 (20.0)	2 (33.3)	
Mean in days	13.6 (SD 2.4)	12.8 (SD 4.7)	p>0.05

t= 0.328

Table V showed the distribution of the patients according to injury treatment interval. The injury treatment interval ranged from 11 to 17 days with the mean of 13.6 (SD 2.4) days in group-A; whereas the injury treatment interval ranged from 8 to 20 days with the mean of 12.8 (SD 4.7) days in group-B. The mean duration ofinjury treatment interval of the patients in both groups was statistically similar (t=0.328; p>0.05).

Table VI. Showing duration of operation of both the groups

Duration of operation	Study group		p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
75-90 minutes	0 (0.0)	6 (100.0)	
90-105 minutes	3 (60.0)	0 (0.0)	
105--120 minutes	2 (40.0)	0 (0.0)	
Mean in minutes	106.00 (SD 4.1)	85.0 (SD 4.4)	p<0.001

t=7.980

Table VI showed the distribution of the patients according to duration of operation. In group-A 3 (60.0%) patients had duration of operation was 90-105 minutes and 2 (40.0%) patient had 105-

120 minutes; while in group- B, 6 (100.0%) patients had duration of operation was 75-90 minutes. The duration of operation ranged from 100 to 110 minutes with the mean of 106.00 (SD 4.18) minutes in group-A; whereas the duration of operation ranged from 80 to 90 minutes with the mean of 85.0 (SD 4.47) minutes in group-B. The duration of operation was shorter in group-B than that of group-A (t=7.980; p<0.001).

Table VII. Showing time of union of both the groups

Time of union	Study group		p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
12--16 weeks	4 (80.0)	3 (50.0)	
16--20 weeks	1 (20.0)	2 (33.3)	
20 or more weeks	0 (0.0)	1 (16.7)	
Mean in weeks	12.80 (SD 1.7)	14.67 (SD 3.2)	p>0.05

t=1.397

Table VII showed the distribution of the patients according to time of union. In group-A, 4 (80.0%) patients had time of union of fracture 12 weeks and 1 (20.0%) patient had 16 weeks; while in group-B, 3 (100.0%) patients had time of union of fracture 12 weeks, 2 (33.3%) patient had 16 weeks and 1 (16.7%) patient had 20 weeks. The mean time of union of fracture was 12.80 (SD 1.7) weeks in patients of group-A; whereas the mean duration of fracture group-B was 14.67 (SD 3.2) weeks. The mean duration of fracture union in both groups was almost identical (t=1.397; p>0.05).

Table VIII. Showing different types of post operative complications of both the groups

Postoperative of complications	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	p
Superficial infection	0 (0.0)	0 (0.0)	
Deep infection	0 (0.0)	1 (16.7)	p>0.05
Anterio-posterior angulation 10^0	1 (20.0)	0 (0.0)	p>0.05
Shortening of the limb	0 (0.0)	0 (0.0)	
Total	1 (20.0)	1 (16.7)	p>0.05

X^2=1.320

Table VIII showed the distribution of the patients according todifferent types of post operative complications. Post operative complications were almost equal in both groups [1 (20.0%) versus 1 (16.7%); p=0.887]. The individual variable of post operative complications were also similar in group-A (IEF) & group-B (MIPO) such as deep infection [0 (0.0%) vs 1 (16.7%); X^2=0.917; p=0.338] and anterio-posterior angulation 10^0 [1 (20.0%) vs 0 (0.0%); X^2=1.320; =0.251].

Table IX. Showing frequency of follow upof both the groups

Frequency of follow up	Study group		p-value
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
3-4 times	0 (0.0)	2 (33.3)	p>0.05
5-6 times	5 (100.0)	4 (66.7)	
Total	5 (100.0)	6 (100.0)	

X^2=2.037

Table IX showed Distribution of patients by frequency of follow up. In group-A, frequency of follow up was 5-6 times in 5 (100.0%) patients; while in group-B, frequency of follow up was 5-6 times in 4 (66.7%) patients and 3-4 times in 2 (33.3%) patients. There was no statistically significant difference of frequency of follow up between group-A and group-B (X^2=2.037; p>0.05).

Table X. Showing final follow up of both the groups

Variables	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	P
Pain			
No pain	4 (80.0)	5 (83.3)	p>0.05
Mild pain	1 (20.0)	1 (16.7)	
Knee flexion			
≥135^0	4 (80.0)	5 (83.3)	p>0.05
<135^0	1 (20.0)	1 (16.7)	
Ankle flexion			
≥45^0	4 (80.0)	5 (83.3)	p>0.05
<45^0	1 (20.0)	1 (16.7)	
Ankle extension			
≥15^0	4 (80.0)	5 (83.3)	p>0.05
<15^0	1 (20.0)	1 (16.7)	

Table X showed the distribution of the patients according to final follow up.At final follow up there was no statistically significant difference of both the groups.

Table XI. Showing duration of hospital stay of both the groups

Duration of hospital stay	Study group		p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
≥20 days	2 (40.0)	5 (83.3)	
<20 days	3 (60.0)	1 (16.7)	
Mean in days	24.00 (SD 7.)	18.67 (SD 3.1)	p>0.05

t=1.610

Table XI showed the distribution of the patients according to duration of hospital stay. The mean duration of hospital stay of the patients was 24.00 (SD 7.1) days in group-A; whereas the mean duration of hospital stay of the group-B was 18.67 (SD 3.1) days. Any method of fixation was not superior to other in respect of length of stay (t=1.610; p>0.05).

Table XII. Showing functional outcome of both the groups

Functional outcome	Study group		p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
Excellent	4 (80.0)	5 (83.3)	p>0.05
Good	1 (20.0)	0 (0.0)	
Poor	0 (0.0)	1 (16.7)	
Total	5 (100.0)	6 (100.0)	

X^2=2.037

Table XII showed the distribution of patient according to functional outcome. Functional outcome was excellent [4 (80.0%) vs 5 (83.3)]; good [1 (20.0%) vs 0 (0.0%)] and poor [0 (0.0%) vs 1 (16.7%)] patients in the group-A and group-B respectively. The functional outcome of either method of fixation did not differ significantly (X^2=2.037; p>0.05).

Table XIII. Showing final outcome of both the groups

Final outcome	Study group		p
	Group-A (n=5) Frequency (%)	Group-B (n=6) Frequency (%)	
Satisfactory	5 (100.0)	5 (83.3)	p>0.05
Unsatisfactory	0 (0.0)	1 (16.7)	
Total	5 (100.0)	6 (100.0)	

X^2=0.917

Table XII showed the distribution of patients according to final outcome. In group-A final outcome was satisfactory in 5 (100.0%); whereas in the group-B, final outcome was satisfactory in 5 (83.3%), and non-satisfactory in 1 (16.7%) patients. The final outcome was almost similar in both groups (X^2=0.917; p>0.05).

DISCUSSION

This cross sectional comparative study was conducted in the Department Orthopaedics, Sylhet M A G Osmani Medical College, Sylhet. A total 12 patients with closed comminuted unstable tibialdiaphyseal fracture were selected during the period from 1st July 2012 to 30th June 2014. In the current study the age of patients of closed comminuted unstable tibialdiaphysial fracture ranging from 21 to 60 years with the mean age of 37.9 ± 13.1 (mean ± standard deviation) years. The mean age of patients in group-A was 33.0 ± 11.1 years; whereas the mean age of the patients in group-B was 42.0 ± 14.14 years ($p>0.05$).This result was supported by the previous study of Mollah.[8] treated with IERF with a mean age of patients was 34.1 years. El-Sayed and Atef.[9]. treated with IEF with a mean age of 36 years. Shrestha[10] using MIPO with a mean age of years was 30.9 years. This study indicating younger age group was more affected in fracture shaft of the tibia and there was no statistically significant difference between the age group ($p>0.05$). The higher incidence in young adult age group points to the higher rate of mobility as well as social violence in this age group.

The current study showed male predominance 9 (81.8%). This result was almost similar to the study of Tantray[3] by MIPO where 84% male, Study of Shabbir[5] by MIPO where 90% male. Study of Sidharthan[7] by IEF where 90.24% male. The sex difference between the patients of group-A and group-B did not show any statistically significant difference ($p>0.05$) in this study. Male being the major working group in our society and thus are more consistently exposed to the external environment that may be the cause of male predominance.

The current study showed that 3 (27.3%) of patients were driver 2((18.2%) of patients were house wife, 2((18.2%) of patients were service holder, and 2 (18.2%) of patients were day labor. People of different occupations were found to be injured in this study. Almost all type of people especially highly mobile group suffered the injury. ($p>0.05$). This result was supported by the previous study of Mollah[8] treated with IEF showed that 26.66% of patient was student, 20% of patient was businessman,13.33% of patient was farmer, 13.33% of patient was labor, 13.33% of patient was service 6.66% are driver and 6.66% are housewife.

The present study showed that the cause of injury was road traffic accident (RTA) in 6 (54.5%) of patients and assault in 5 (44.5%) of patients. In group-A road traffic accident (RTA) in 3 (60.0%) of patients and assault in 2 (40%) of patients, where as in the group-B the cause of injury was road traffic accident (RTA) in 3 (50%) of patients and assault in 3 (50%) of patients. The cause of injury should not interfere the final outcome ($X^2=0.110$; $p>0.05$). This result was supported by the study of MIPO by Shrestha[10] was 50%, Tantray[3] was 56%, Shabbir[5] was 72.6% RTA. Study of IEF by Mollah[8] was 70.58% RTA. The major causes of fractures of this study are high speed RTA which is the major cause of the tibial fracture worldwide.

The present series of 11 patients, proximal third was involved in 27.3%, middle third was involved in 45.5% and distal third was involved in 27.3% of the patients. Proximal third (60%) was relatively more involved in group-A, where as middle third (66.7) was relatively more involved in group-B. Ultimately the distribution of respondents by site of fracture in the both group was almost similar ($p>0.05$). This result was correlated with the study of Tantray[3] (MIPO) with middle third was involved in 60%, distal third was involved in 24% and proximal third was involved in 16% of the patients with fracture shaft of the tibia. Giri[11] found that proximal third was involved in 25.5%, middle third was involved in 45.5% and distal third was involved in 29.0% of the patients with close comminuted unstable fracture of tibial diaphysis.

In the current study the mean duration of injury treatment interval was 13.6 (SD 2.4) days in patients of group-A; whereas the mean duration of injury treatment interval group-B was 12.8 (SD 4.7) days. The mean duration of injury treatment interval in both groups was almost identical ($t=0.328$; $p>0.005$). There was no statistically significant difference of duration of fracture before surgery between group-A and group-B. The mean duration of injury treatment interval of Shabbir[5], Shrestha[10] and Tantray[3] (MIPO) was 12.5, 4.45 and 8 days respectively. This result was correlated with this study.

In the current study, the duration of total operation time was 106.00 (SD 4.18) minutes of patients in group-A; whereas the mean duration of operative procedure was 85.0 (SD 4.47) minutes of patients in group-B. The duration of total operation time was shorter in group-B than that of group-A ($t=7.980$; $p<0.001$).There was statistically significant difference

of duration of operation between group-A and group-B. The mean operative time of El-Sayed and Atef.[9] IEF was 55 min. The mean operative time of Shabbir[5] and Shrestha[10] MIPO was 90 and 81.33 minutes respectively. This result was correlated with this study.

In this study, found that mean time of union was 12.80 (SD 1.79) and14.67 (SD 3.27) weeks in patients of group-A and group-B respectively. The mean time of union in both groups was almost identical (t=1.397, p=0.285). There was no statistically significant difference of time of union of fracture between group-A and group-B. El-Sayed.and Atef[9]found that a mean union time for patients treated withIlizarov external fixator was 14 weeks (range 13-18 weeks). Shabbir[5]found that a mean union time for patients treated with MIPO was 13 weeks (range 9 -16 weeks).Shrestha[10] also found that a mean union time for patients treated with MIPO was 18.5 weeks (range: 12-28weeks).Lau[12] found that a mean union time for patients treated with MIPO was 18.7 weeks (range: 12-44 weeks). These results were correlated with this study.

The infection developed in 1(16.7%) patient in group A& anterior-posterior angulation in 1(20.0%) patient in group B. There was no statistically significant difference of post-operative complications between group-A and group-B (p>0.05). El-Sayed and Atef[9] in IEF found pin tract infection in 81 (25%), pin breakage and removal in 14 (4.32%). Shrestha[10] in MIPO found delayed union in 1 (5%), deep infection in 1 (5%), superficial infection 2 (10%), and ankle stiffness in 1 (5%). secondary procedure1 (5%). Lau[12] in MIPO found delayed union in 5 (10.41%), wound infection in 8 (16.66%), and secondary procedure in 1 (2.08%). Ahmad[13] in MIPO found delayed union in 3 (16.66%), superficial infection 1 (5.56%), chronic wound infection in 1 (5%), and ankle stiffness in 1 (5.56%). Implant failure in1 (5.56%). Mushtaq[14] found delayed union in 1 (5%), deep infection in 1 (5%), superficial infection 2 (10%), and ankle stiffness in 1 (5%). secondary procedure1 (5%).

Present study also showed the distribution of patients by frequency of follow up. In group-A, 3 (60.0%) patients had got 5 times follow up, 1 (20.0%) patient had got 6 times follow up and another 1 (20.0%) patient had got 7 times follow up; while in group-B, 2 (33.3%) patients had got 3, 4 and 5 times follow up in each. The times of follow up was not differ significantly between group-A and group-B (X^2=6.160; p>0.05).

In the current study, the functional outcome was assessed according to Gustilo[15] and in this study it was excellent [4 (80.0%) vs 5 (83.3)]; good [1 (20.0%) vs 0 (0.0%)] and poor [0 (0.0%) vs 1 (16.7%)] patients in the group-A and group-B respectively. The functional outcome of either method of fixation was almost similar and did not differ significantly (p>0.05). Mollah[8] found excellent outcome in 47.06%, good in 35.29%, fair 11.76% and poor5.88% of patients with close comminuted unstable fracture of tibial diaphysis treated with IEF. Shabbir[5] found excellent outcome in 93.1%, excellent to good in 6.9% of patients with close comminuted unstable fracture of tibial diaphysis treated with MIPO. Tae Hun Kim[16] found excellent outcome in 31.25%, good in 62.5% and fair 6.25% of patients with close comminuted unstable fracture of tibial diaphysis with MIPO using. Tantray[3] found good outcome in 84%, excellent in 8% and poor in 8% of patients with close comminuted unstable fracture of tibial diaphysis treated with MIPO.

In this study, the final outcome was satisfactory in 100% in group-A; whereas in the group-B, final outcome was satisfactory in 5 (83.3%), and non-satisfactory in 1 (16.7%) patients. The final outcome was almost similar in both groups (p>0.05).

For prevention of any post-immobilization stiffness of the ankle and knee joints immediate weight bearing exercise started from 1st POD in IEF group. Active and passive ankle and knee range of motion were measured by goniometer and compared with the normal side and recorded in each post operative follow up. Range of movement of operating sides were less and painful in early period in group-A due to Ilizaov frame. Gradually the pain would decrease and ROM would increase. After removal of implants of four patients ROM of knee and ankle joints of operating sides were mildly less in comparative to the normal sides. During final follow up the ROM of knee and ankle joints of operating sides were similar to the normal sides & within the normal range of movement. One patient did not attend in follow up in 1st three months, than attend with anterior angulation up to 10 degree. Angulation was corrected at 5th month. In MIPO group only one patient developed deep infection and the infection is controlled by conservative treatment. The fracture healed & ROM was near similar to the normal side. The ROM of the fracture sides of other patients in MIPO group was similar to the normal sides & within the normal range of movement.

In conclusion, considering the duration of operation it may be concluded that both the techniques are equally safe and effective and considering the result of the both techniques we do recommend the minimally invasive plate osteosynthesis technique is the feseable treatment of closed comminuted unstable tibial diaphyseal fracture.

REFERENCES

1. Court-Brown CM.,McBirnie J. The epidemiology of tibialfractures. J Bone Joint Surg Br1995: 77: 417-21.
2. Konowalchuk BK, Wills NJ and Swiontkowski M. Tibial Shaft Fractures. Emedicine Orthopedic Surgery, 2009 Last updated on May 15, 2009. Available from: http://www. emedicine medescape.com.viewarticle/overview [Accessed on 20 December 2009].231- 45
3. Tantray M, Kuchay G, Wani A, Habib, Sharma S. &Habib M., A Clinical Study For Management Of Tibial Diaphyseal Fractures In Adults With Locking Compression Plate Using Mippo Technique. I J Ortho Surg 2011; 18: 1-7.
4. Whittle AP. Fracture of the lower extremity. In: ST Canale JH Beaty eds. Campbell's Opearative Orthopaedics. Vol-3. 11th edition. Pheladephia: Mosby Elsevier, 2008, 3117- 46.
5. Shabbir G, Hussain S, Ahmed Nasir Z, Shafi K. & Khan AJ. Minimally invasive plate osteosynthesis of close fracture of distal tibia". 1st ed.[ebook] J Ayub Med Coll Abbottabad: pubfacts. 2011, Available at: 6 apr .2011].1 - 3
6. He G, Wang H, Wang Q, Chen Z. &Cai X. Effect of minimally invasive percutaneous plates versus interlocking intramedullary nailing in tibial shaft treatment for fractures in adults: A meta-analysis. Clinics 2014;69: 234 - 40.
7. Sidharthan S, Sujith A, Rathod AK & Pathak RH. Management Of High Energy Tibial Fractures Using Ilizarov Apparatus Internet J Ortho Surg. 2005 ; 3:1- 4.
8. Mollah MMU. Evaluation of the treatment of Gustilo IIIA open tibial shaft fracture by transosseus osteosynthesis technique with IEF. Dhaka University Thesis, 2010;1-48
9. El-Sayed M. &Atef A. Management of simple (types A and B) closed tibial shaft fractures using percutaneous lag-screw fixation and Ilizarov external fixation in adults. SICOT 2012; 36:2133-8.
10. Shrestha D, Acharya BM. &Shrestha PM. Minimally Invasive Plate Osteosynthesis with Locking Compression Plate for Distal Diametaphyseal Tibia Fracture, Katmandu Univ Med J 2011; 34:62-8.
11. GiriSK, Adhikari BR, Gurung GB, RC DR, Bajracharya AR, Khatri K. Mini-open reduction and intramedullary interlocking nailing of fracture shaft of tibia without an image intensifier. Nepal Med Coll J 2008; 10: 123- 5.
12. Lau T, Leung F, Chan C and Chow S. Wound complication of minimally invasive plate osteosynthesis in distal tibia fractures. International Orthop 2007; 32: 697-703.
13. Ahmad M, Sivaraman A, Zia A, Rai A & Patel A, Percutaneous Locking Plates for Fractures of the Distal Tibia: Our Experience and a Review of the Literature. J Trauma Inj InfectCrit Care 2010; Available at:http://dx.doi.og/10.1097/ ta. 0b013e3181f140b3 [Accessed 11 Oct. 2014].
14. MushtaqA, Shahid R, Asif M &Maqsood M. Distal Tibial Fracture Fixation with Locking Compression Plate (LCP) Using the Minimally Invasive Percutaneous Osteosynthesis (MIPO) Technique. Eur J Trauma EmergSurg 2009; 35: 159- 64.
15. Gustilo RB, Kyle RF &Templeman DC eds, Fractures and Dislocations. USA. Mosby-Yeaf Books Inc 1993; 1:197-214
16. Kim TH, Chung SH. MIPO for fracture distal tibia. Kosin Med J 2014; 29: 23-9

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Utility of Serum Ascites Albumin Gradient (SAAG) and Ascitic Fluid Total Protein (AFTP) in Differentiating Cirrhotic and Non-cirrhotic Ascites

Md. Enayet Hossain[1], Arunangshu Raha[2], Ferdaus Ahammed[3], S.M. Sajjadul Haq[4]

Abstract

Traditionally ascites was classified as transudative and exudative based on the total protein concentration of the ascitic fluid. On the Basis of SAAG ascites is now being classified as "high gradient" and "low gradient". The difference between serum and ascitic fluid albumin concentration correlates directly with portal pressure.The present study is designed to determine the utility of SAAG and ascitic fluid total protein (AFTP) in the differentiation of cirrhotic ascites and non-cirrhotic ascites.

It was a cross-sectional prospective study conducted in Department of Medicine, Sylhet M.A.G. Osmani Medical College Hospital, Sylhet from July 2015 to December 2015.

One hundred adult patients with ascites fulfilling the inclusion and exclusion criteria were included in this study.

The mean age was 49.3 ± 13.6 years with male to female ratio of 1.7:1. The most common cause of ascites was cirrhosis [63 (63.0%)]; and other causes were abdominal tuberculosis [20 (20.0%)] and malignancy. Ascitic fluid total protein level was 21.0 gm/ L (SD ± 9.8) in ascites of cirrhosis; while ascitic fluid total protein level was 38.8 gm/ L (SD ± 9.6) in ascites of non-cirrhotic causes.Ascitic fluid albumin was 9.0 gm/L (SD ± 6.7) in ascites of cirrhosis; while ascitic fluid albumin was 25.0 gm/L (SD ± 6.2) in ascites of non-cirrhotic causes. Serum albumin was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes.Serum-ascitic albumin gradient was 22.3 gm/L (SD ± 7.9) in ascites of cirrhosis; while serum-ascitic albumin gradient was 9.2 (SD ± 2.3) in ascites of non-cirrhotic causes. Serum-ascitic albumin gradient was significantly higher in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes (Z=9.927; p<0.01). Sensitivity and specificity of ascitic fluid total protein at a cut off value of <25 gm/L in differentiating ascites of cirrhotic causes from ascites of non-cirrhotic causes was 71.4% and 89.2%. Positive and negative predictive values were 91.8% and 64.7% respectively. The overall accuracy was 78.0%. Sensitivity and specificity of serum-ascites albumin gradient (SAAG) at a cut off value of ≥11 gm/L in differentiating ascites of cirrhotic from non-cirrhotic causes was 93.7% and 83.8%. Positive and negative predictive values were 90.8% and 88.6% respectively. The overall accuracy was 90.0%.

So, diagnostic accuracy of serum-ascitic albumin gradient (SAAG) is higher than that of ascitic fluid total protein in distinguishing ascites of cirrhotic causes from non-cirrhotic causes.

[OMTAJ 2016; 15(2)]

Introduction

Ascites denotes the condition of excessive, abnormal and pathologic free fluid accumulation in the peritoneal cavity.[1-4]The most common cause of ascites is cirrhosis (75% of patients). Ten percent of cases are due to malignancy, 3% are due to cardiac failure, 2% are due to tuberculosis, and the remainder is due to other causes.[5]

The cause of ascites in cirrhotic patients has been well described.[6] Portal hypertension develops secondary to resistance at the splanchnic and arteriolar level, and the sinusoidal resistance is not relieved by the subsequent development of porto-systemic collaterals.[7]

The cause of ascites development in oncology patients differs from patients with cirrhosis. In 50% of patients with malignancies, ascites development is secondary to invasion of the parietal or visceral peritoneum; 15% are due to liver invasion and portal venous

1. Associate professor of Medicine, Sylhet MAG Osmani Medical College.
2. MD(Gastroenterology) 3rd part student, Dhaka Medical College.
3. Lecturer, Department of Pathology), Sylhet MAG Osmani Medical College.
4. Medical Officer, Upazilla Health Complex, Jagannatpur,Sunamgonj.

compression, 15% are a combination of the first two, and the remaining 20% are attributed to chylous ascites secondary to lymphatic invasion.[8]

Earlier, ascites was classified as transudative and exudative based on the total protein concentration of the ascitic fluid. The traditional concept of high protein ascites(> 2.5 g/dl) as exudative was questioned because the normal peritoneal fluid protein concentration is sometimes > 4 g/dl; the ascitic fluid protein concentration increases in cirrhotic patients with diuresis and albumin infusion; some transudative ascites like cardiac ascites have high protein concentration while some traditionally exudative ascites like malignant ascites have low concentration of protein; and moreover cirrhosis may be the most frequent cause of high protein ascites.[9]

To overcome the shortcomings ascites is now being classified as "high gradient" and "low gradient". The difference between serum and ascitic fluid albumin concentration correlates directly with portal pressure.[9]Several studies have demonstrated the superiority of the serum-ascites albumin concentration gradient (SAAG) in the discrimination of ascitic fluid, compared with the exudate-transudate concept. It was shown that such a classification has a validity rate of 90% or more in detecting the ascites of portal hypertension.[10,11,12,13]Serum-ascites albumin gradient (SAAG) is defined as the difference between serum and ascitic fluid albumin concentrations. If the SAAG is greater than 1.1 g/dL, it is termed a "high" SAAG, and if it is less than 1.1 g/dL, it is termed a "low" SAAG. A high SAAG occurs in portal hypertension, which may be related to either liver disease or cardiac disease. When the SAAG is greater than 1.1 g/dL, the concentration of total proteins in ascitic fluid helps to distinguish cardiac causes from hepatic causes. In cardiac disease, the total protein concentration in ascitic fluid is usually greater than 2.5 g/dL, whereas in liver disease, it is less than 2.5 g/dL. A low SAAG typically is associated with carcinomatosis, tuberculosis, pancreatic ascites, and nephritic syndrome.

The test has been performed in a number of studies demonstrating a lower value for patients with malignant neoplasm and tuberculous peritonitis (TBP) compared with LC group.[14,10,11,15] This biochemical parameter is simple and could be routinely performed in developing countries where ascites is common. However, the diagnostic value of the SAAG has poorly been evaluated in our population until now. The present study is, therefore, undertaken to determine prospectively the causes of ascites and to find out the utility of SAAG and ascitic fluid total protein in the differentiation of ascites of cirrhotic and non-cirrhotic cause in the North-East part of Bangladesh.

Materials and Methods

It was a cross-sectional prospective study conducted in Department of Medicine, Sylhet M.A.G. Osmani Medical College Hospital, Sylhet from July 2015 to December 2015. All admitted patients in different Medicine units, Sylhet M.A.G Osmani Medical College Hospital, Sylhet, who were diagnosed as a case of ascites during the study period, were the study population in this study.

Inclusion criteria:

- Clinical features suggesting ascites.
- Ultrasonography suggesting free fluid in the peritoneal cavity.
- Age 14 years and above, irrespective of gender.

Exclusion criteria:

- Hepatocellular malignancy if it was on the top of cirrhosis
- Diagnosis of ascites was non-conclusive.
- The patients who were very sick or having hepatic encephalopathy.
- Patients who refused to take part in this study.

Sample size: One hundred patients with ascites fulfilling the inclusion and exclusion criteria.

Sampling method: Purposive sampling was employed as sampling technique in this study.

Procedures of collecting data:

Patients with suspected ascites of aged 14 years or above age, irrespective of sex, who were admitted in different units of Department of Medicine, Sylhet M.A.G. Osmani Medical College Hospital, Sylhet from July 2015 to December 2015 were the study population in this study.

Informed written consent was taken from each of the patients before taking any interview. The consent form clearly describes the purpose and methods of the study, confidentiality of the interviews, risks and benefits of participating in the study, their rights to participate voluntarily and to refuse at any point in time without consequences.

Each patient was assessed thoroughly by taking complete history including present or previous jaundice, fever, abdominal distension and rapid weight

gain, generalized swelling, swelling of legs, shortness of breath, puffy face, and scanty micturition. Meticulous clinical examination and necessary investigations were also done to confirm ascites. Those who met the inclusion criteria were taken as sample and those, who met the exclusion criteria by detail history, clinical examination and relevant investigations were excluded from the study.

A total of 100 patients with ascites were selected consecutively. Before any therapeutic intervention, diagnostic paracentesis of abdomen were done. The samples of ascitic fluid and venous blood samples were obtained in the same setting.

Further investigations were done to identify the causes of ascites such as urine for routine examination, x-ray chest P/A view, liver function test-prothrombin time, serum total protein and serum albumin, serum albumin globulin ratio,renal function test, serum cholesterol, ultrasonogrphy, Tuberculin test, endoscopy of upper GIT, barium meal and follow through, colonoscopy, where necessary. Ascitic fluid study was done in all cases for protein, albumin, cytology, malignant cell and ADA where appropriate. Serum ascites albumin gradient was calculated.

One hundred patients with ascites were divided into two groups according to presence or absence of cirrhosis. Ascites due to cirrhosis of liver were enrolled as cirrhotic ascites while ascites due to other causes were enrolled as non-cirrhotic ascites.

Relevant data from history, physical examination and investigations were recorded in predesigned case record form which was filled up by the investigator himself.

Results

Age distribution of the patients:

The age of the patients ranged from 18 to 70 years with the mean age of 49.3 ± 13.6 years. Figure 1 showed the age distribution of the patients.

In this study 25.0% patients belonged to age group of 51 to 60 years, 24.0% to age group of 41 to 50 years, 21.0% to age group of 61 to 70 years, 17.0% to age group of 31 to 40 years, 9.0% to age group of 21 to 30 years and 4.0% to age group of up to 20 years.

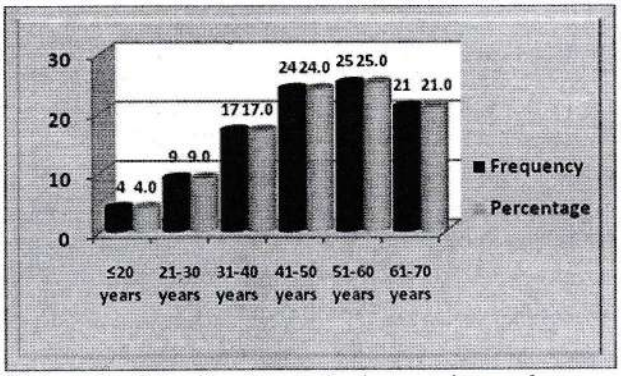


Figure-1: Distribution of the patients by age (n=100)

Distribution of the patients by sex:

In this study, 63 (63.0%) patients were male and 37 (37.0%) patients were female with male to female ratio of 1.7:1. Distribution of patients according to sex is shown in Figure-2.

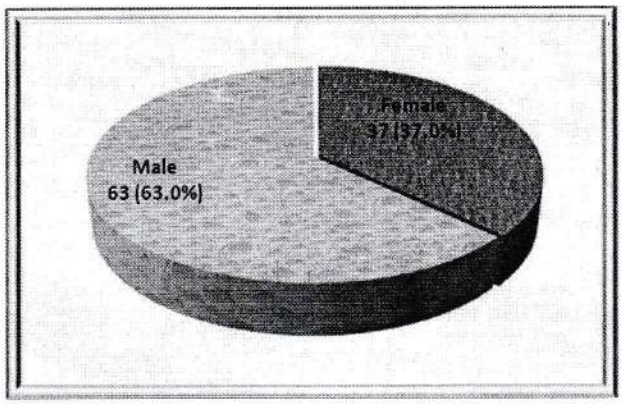


Figure-2: Distribution of the patients by sex (n=100)

Clinical presentation (symptoms):

Clinical presentation (symptoms) of ascites patients was shown in table-I. Most common clinical presentations were distension of abdomen (75.0%) followed by loss of appetite (63.0%), pain abdomen (32.0%), fever (19.0%), bowel disturbance (22.0%) and yellow colouration of sclera (18.0%).

Table-I: Clinical presentation (symptoms) of ascites patients (n=100)

Cause of ascites	Symptoms					
	Distension of abdomen	Fever	Pain abdomen	Loss of appetite	Bowel disturbances	Yellowish discolouration of sclera
Cirrhosis (n=63)	50	3	7	31	10	18
Abdominal tuberculosis (n=20)	11	14	16	17	8	0
Malignancy (n=17)	14	2	9	15	4	0
Total (n=100)	75	19	32	63	22	18

Clinical presentation (signs):

Clinical presentation (signs) of ascites patients was shown in table-II. Clinical presentations (signs) were pallor (27.0%), icterus (18.0%), splenomegaly (37.0%), hepatomegaly (2.0%) and pedal edema (26.0%).

Table-II: Clinical presentation (signs) of ascites patients (n=100)

Causes of ascites	Signs				
	Icterus	Pallor	Pedal edema	Hepatomegaly	Splenomegaly
Cirrhosis (n=63)	18	12	17	2	37
Abdominal tuberculosis (n=20)	0	5	5	0	0
Malignancy (n=17)	0	10	4	0	0
Total (n=100)	18	27	26	2	37

Causes of the ascites:

The most common cause of ascites was cirrhosis [63 (63.0%)]; and other causes were abdominal tuberculosis [20 (20.0%)] and malignancy. Causes of asciteswere shown in figure-3.

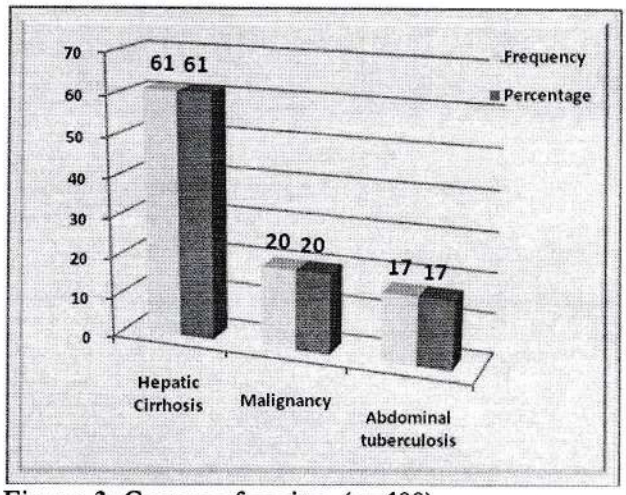


Figure-3: Causes of ascites (n=100)

Characteristics of ascitic fluid in different aetiological type of ascites:

Characteristics of ascitic fluid in different aetiological type of ascites were shown in Table-III.

Table-III: Characteristics of ascitic fluid in different aetiological type of ascites (n=100)

Aetiology	Gross appearance				AFTP Mean (SD) gm/L	AFAlb Mean (SD) gm/L	SAlb Mean (SD) gm/L	SAAG Mean (SD) gm/L
	Straw	clear	Turbid	Haemorrhagic				
Cirrhosis (n=63)	27	36	0	0	21.0 (9.8)	9.0 (6.7)	32.3 (.72)	22.3 (7.9)
Abdominal tuberculosis (n=20)	14	3	0	3	36.6 (11.4)	24.2 (7.5)	35.1 (51)	9.5 (1.5)
Malignancy (n=17)	9	0	0	8	41.4 (6.3)	26.0 (4.3)	34.2 (3.7)	8.8 (2.9)
Total (n=100)	40	39	0	11	27.6 (13.0)	14.9(10.1)	33.3 (6.4)	17.5 (9.0)

AFTP: Ascitic fluid total protein
AFAlb: Ascitic fluid albumin
S Alb: Serum Albumin
SAAG: Serum-ascitic albumin gradient

Comparison of ascitic fluid total protein between cirrhotic and non-cirrhotic causes of ascites:

Ascitic fluid total protein level was 21.0 gm/ L (SD ± 9.8) in ascites of cirrhosis; while ascitic fluid total protein level was 38.8 gm/ L (SD ± 9.6) in ascites of non-cirrhotic causes. Ascitic fluid total protein level was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes (Z=-8.823; p<0.01). Comparison of ascitic fluid total protein between cirrhotic and non-cirrhotic causes of ascites was shown in table-IV.

Table-IV: Comparison of ascitic fluid total protein between cirrhotic and non-cirrhotic causes of ascites (n=100)

Ascitic fluid total protein (g/dl)	Type of ascites		*p- value
	Cirrhotic (n=63)	Non-cirrhotic (n=37)	
Mean	2.10	3.88	p<0.01
Standard deviation	± 0.98	± 0.96	

*Z was applied to analyses the data.

Comparison of ascitic fluid albumin between cirrhotic and non-cirrhotic causes of ascites:

Ascitic fluid albumin was 9.0 gm/L (SD ± 6.7) in ascites of cirrhosis; while ascitic fluid albumin was 25.0 gm/L (SD ± 6.2) in ascites of non-cirrhotic causes. Ascitic fluid albumin was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes (Z=-11.876; p<0.01). Comparison of ascitic fluid albumin between cirrhotic and non-cirrhotic causes of ascites was shown in Table-V.

Table-V: Comparison of ascitic fluid albumin between cirrhotic and non-cirrhotic causes of ascites

Ascitic fluid albumin (g/dl)	Type of ascites		*p- value
	Cirrhotic (n=63)	Non-cirrhotic (n=37)	
Mean	0.90	2.50	p<0.01
Standard deviation	± 0.67	± 0.62	

*Z was applied to analyses the data

Comparison of serum albumin between cirrhotic and non-cirrhotic causes of ascites:

Serum albumin was 32.3 gm/L (SD ± 7.2) in ascites of cirrhosis; while serum albumin was 35.0 gm/L (SD ± 4.4) in ascites of non- cirrhotic causes. Serum albumin was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes (Z=-2.038; p=0.044). Comparison of serum albumin between cirrhotic and non-cirrhotic causes of ascites was shown in table-VI.

Table-VI: Comparison of serum albumin between cirrhotic and non-cirrhotic causes of ascites

Serum albumin (g/dl)	Type of ascites		*p-value
	Cirrhotic (n=63)	Non-cirrhotic (n=37)	
Mean	3.23	3.50	p=0.044
Standard deviation	± 0.72	± 0.44	

*Z was applied to analyses the data

Comparison of serum-ascitic albumin gradient between cirrhotic and non-cirrhotic causes of ascites:

Serum-ascitic albumin gradient was 22.3 gm/L (SD ± 7.9) in ascites of cirrhosis; while serum-ascitic albumin gradient was 9.2 (SD ± 2.3) in ascites of non-cirrhotic causes. Serum-ascitic albumin gradient was significantly higher in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes (Z=9.927; p<0.01). Comparison of serum-ascitic albumin gradient between cirrhotic and non-cirrhotic causes of ascites was shown in Table-VII.

Table-VII: Comparison of serum-ascitic albumin gradient between cirrhotic and non-cirrhotic causes of ascites

Serum-ascitic albumin gradient (g/dl)	Type of ascites		*p-value
	Cirrhotic (n=63)	Non-cirrhotic (n=37)	
Mean	2.23	0.92	P <0.01
Standard deviation	± 0.79	± 0.23	

*Z was applied to analyses the data

Validity of ascitic fluid total protein in differentiating ascites of cirrhotic and non-cirrhotic causes:

Table-VIII showed the cross tabulation of ascitic fluid total protein and type of ascites. In this study, sensitivity and specificity of ascitic fluid total protein at a cut off value of <25 gm/L in differentiating ascites of cirrhotic causes from ascites of non-cirrhotic causes was 71.4% and 89.2%. Positive and negative predictive values were 91.8% and 64.7% respectively. The overall accuracy was 78.0%.

Table-VIII: Cross tabulation of ascitic fluid total protein at cut off point 25 gm/L and type of ascites (n=100)

Ascitic fluid total protein	Ascites type		Total
	Cirrhotic	Non-cirrhotic	
<25 gm/L	45 (a)	4 (b)	49
≥ 25 gm/L	18 (c)	33 (d)	51
Total	63 (a+c)	37 (b+d)	100

Sensitivity=a/(a+c)X100=45/(45+18) X100=71.4%
Specificity=d/(b+d)X100=33/(33+4) X100=89.2%
Positive predictive value=a/(a+b) X100=45/(45+4) X100=91.8%
Negative predictive value=d/(c+d) X100=33/(33+18) X100=64.7%
Accuracy=
(a+d)/(a+b+c+d)=(45+33)/(45+4+18+33) X100=78.0%

Validity of serum-ascites albumin gradient in differentiating ascites of cirrhotic and non-cirrhotic causes:

Table-IX showed the cross tabulation of serum-ascites albumin gradient (SAAG) and type of ascites. In this study, sensitivity and specificity of serum-ascites albumin gradient (SAAG) at a cut off value of ≥11 gm/L in differentiating ascites of cirrhotic from non-cirrhotic causes was 93.7% and 83.8%. Positive and negative predictive values were 90.8% and 88.6% respectively. The overall accuracy was 90.0%.

Table-IX: Cross tabulation of serum-ascites albumin gradient at cut off point 11 gm/L and type of ascites (n=100)

Serum-ascites albumin gradient	Ascites type		Total
	Cirrhotic	Non-cirrhotic	
≥11 gm/L	59 (a)	6 (b)	65
< 11 gm/L	4 (c)	31 (d)	35
Total	63 (a+c)	37 (b+d)	100

Sensitivity=a/(a+c)X100=52/(59+4) X100=93.7%
Specificity=d/(b+d)X100=31/(31+4) X100=83.8%
Positive predictive value=a/(a+b) X 100=59/(59+6) X100=90.8%
Negative predictive value=d/(c+d) X 100=31/(31+4) X100=88.6%
Accuracy= (a+d)/(a+b+c+d)=(59+31)/(59+6+4+31) X100=90%

Discussion

Earlier, ascites was classified as transudative and exudative based on the total protein concentration of the ascitic fluid. The traditional concept of high protein ascites(> 2.5 g/dl) as exudative was questioned because: (a) the normal peritoneal fluid protein concentration is sometimes > 4 g/dl; (b) the ascitic fluid protein concentration increases in cirrhotic patients with diuresis and albumin infusion); (c) some transudative ascites like cardiac ascites have high protein concentration while some traditionally exudative ascites like malignant ascites have low concentration of protein; and (d) moreover cirrhosis may be the most frequent cause of high protein ascites.

To overcome the shortcomings, ascites is now being classified as "high gradient" and "low gradient". When the difference between serum albumin and ascitic fluid albumin is >l.lg/dl it is called high gradient ascites, whereas if the difference is < 1.1 g/dl it is termed as low gradient ascites. The SAAG is based on oncotic hydrostatic balance. Portal hypertension results in an abnormally high hydrostatic pressure gradient between the portal bed and the ascitic fluid. There must be a similarly large difference between ascitic fluid and intravascular oncotic pressure than other proteins. The difference between serum and ascitic fluid albumin concentration correlates directly with portal pressure.[10]

This cross-sectional study was carried out in the different units of Medicine in Sylhet MAG Osmani Medical College Hospital, during the period from July 2015 to December 2015 with a view to find out the utility of serum ascites albumin gradient and ascitic fluid total protein in differentiating cirrhotic and non-cirrhotic ascites. The outcome of the study was discussed below:

In our study the age of the patients ranged from 18 to 70 years with the mean age of 49.3 ± 13.6 years. This result was similar to the study of Khan, [16] that mean age of the patients with ascites was 52.9 (SD± 14.75) years. Mahmoodet al.[17] found that the age of the

patients ranged from 13 to 61 years with the mean age of 40 (SD± 5) years. Khan et al.[18]found the age of the patients ranged from 30 to 90 years with the mean age of 57.0 years.

This study also showed that 87.0% patients belonged to age group of 31 to 70 years, 9.0% to age group of 21 to 30 years and 4.0% to age group of up to 20 years. This result was almost similar to the study.[19,20,21] Aminuddin,[19] found most of the patients were between 19 to 59 years (68.0%). Hoque,[20] reported 77.0% of their ascites patients were aged between 21 to 60 years. Mandal,[21]reported that most of the patients were between 21 to 60 years (81.3%).

In this study, 63.0% of patients were male and 37.0% of patients were female with mate to female ratio of 1.7:1. This result was consistent with the study of Khan,[10] that 67.3% of patients were males and 32.7% of patients were females of their series of ascitic patients. Mahmoodet al.[17]also supported the present study result that 68% of cases were male and 32% of cases were female. Aminuddin,[19] reported that 66.0% of their ascites patients were male and 34.0% were female. Hoque,[20] found 74.0% of their ascites patients were male and 26.0% were female. Khan et al.[18]found 72.0% of their ascites patients were male and 28.0% were female.

The current study showed that the most common clinical presentations were distension of abdomen (75.0%) followed by loss of appetite (63.0%), pain abdomen (32.0%), fever (19.0%), bowel disturbance (22.0%) and yellow colouration of sclera (18.0%). This results was consistent with other studies.[18, 19, 20]

This study showed that the clinical presentations (signs) were pallor (27.0%), icterus (18.0%), splenomegaly (37.0%), hepatomegaly (2.0%) and pedal edema (26.0%). This result was almost similar to the other studies.[18,19,20]

In the present study, most common cause of ascites was cirrhosis (63.0%), and other causes were abdominal tuberculosis (20.0%) and malignancy (17.0%). This result was supported by Khan,[16] that liver cirrhosis 62.0%, malignancy related ascites in 22.0%, tuberculous ascites in 8.0%, Heart failure in 7.0%, nephrotic syndrome in 3.0% and others in 2.0%. This result was also similar to the study of Mahmood et al.[17]that the commonest cause of ascites is cirrhosis of liver 68%, tubercular peritonitis 9%, hepatocellular carcinoma 8%, congestive cardiac failure 6%, malignancy 4%, nephrotic syndrome 3%, lymphoma 2% and others 4%. Hoque,[20] found that the commonest cause of ascites is cirrhosis of liver 50.0%, congestive cardiac failure 13.0%, peritoneal carcinomatosis 9.0%, hepatocellular carcinoma 6.0%, nephrotic syndrome 6.0%, tubercular peritonitis 5.0%, others 11.0%. Aminuddin,[19] found that the commonest cause of ascites is cirrhosis of liver 50.0%, congestive cardiac failure 26.0%, hepatocellular carcinoma 8.0%, nephrotic syndrome 8.0%, tubercular peritonitis 6.0% and carcinoma stomach 2.0%.

In this study ascitic fluid total protein level was 21.0 gm/ L (SD ± 9.8) in ascites of cirrhosis; while ascitic fluid total protein level was 38.8 gm/ L (SD ± 9.6) in ascites of non-cirrhotic causes. Ascitic fluid total protein level was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes ($p<0.01$). This result was supported by the study of Sharatchandraet al.[22] that ascitic fluid total protein was significantly lower in cirrhosis than tuberculosis ($p<0.01$) and malignancy ($p<0.01$) but not between tuberculosis and malignancy (not significant). In this regard Gupta et al.[23] found ascitic fluid total protein was significantly lower in cirrhotic patients with ascites than that of malignancy related ascites [21 ± 10 vs 45 ± 10 gm/L; $p<0.001$).

In the present study ascitic fluid albumin was 9.0 gm/L (SD ± 6.7) in ascites of cirrhosis; while ascitic fluid albumin was 25.0 gm/L (SD ± 6.2) in ascites of non-cirrhotic causes. Ascitic fluid albumin was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes ($p<0.01$). This result was supported by the study of Sharatchandraet al.[22] that ascitic fluid total protein was significantly lower in cirrhosis than tuberculosis ($p<0.01$) and malignancy ($p<0.01$) but not between tuberculosis and malignancy (not significant). In this regard Gupta et al.[23] found ascitic fluid albumin was significantly lower in cirrhotic patients with ascites than that of malignancy related ascites [12 ± 8 vs 32 ± 7 gm/l; $p<0.001$). Khan et al.[18] reported that patients with malignancy as the cause of ascites were found to have higher ascites albumin levels ($p<0.0001$).

In this study serum albumin was 32.3 gm/L (SD ± 7.2) in ascites of cirrhosis; while serum albumin was 35.0 gm/L (SD ± 4.4) in ascites of non- cirrhotic causes. Serum albumin was significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes ($p=0.044$). This result was in accordance with the study of Sharatchandraet al.[22] that ascitic fluid total protein was significantly lower in cirrhosis than tuberculosis ($p<0.01$) and malignancy ($p<0.01$) but not between tuberculosis and malignancy (not significant).

In our study serum-ascitic albumin gradient was 22.3 gm/L (SD ± 7.9) in case of cirrhosis; while serum-ascitic albumin gradient was 9.2 (SD ± 2.3) in cases of non-cirrhotic causes. Serum-ascitic albumin gradient was significantly higher in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes ($p<0.01$). This result was supported by Gupta et al.[23] that serum-ascitic fluid albumin gradient was significantly higher in cirrhotic patients with ascites than that of malignancy related ascites [23 ± 08.2 vs 7.8 ± 10 gm/dl; $p<0.001$). Khan et al.[18] reported that patients with malignancy as the cause of ascites were found to have lower SAAG than other patients ($p<0.0001$).

In this study, sensitivity and specificity of ascitic fluid total protein at a cut off value of <25 gm/L was 71.4% and 89.2% respectively in the differentiation of ascites of cirrhotic causes from ascites of non-cirrhotic causes. Positive and negative predictive values were 91.8% and 64.7% respectively. The overall accuracy was 78.0%. Al-Knawy[24] supported this result that the efficiency in correctly diagnosing patients with ascites caused by liver disease and those related to non-liver disease (malignancy and peritoneal tuberculosis) was 84% for ascitic fluid total protein at a cut off value of >25 g/L. This result was also supported by Gupta et al.[23] that sensitivity and specificity of ascitic fluid total protein at cut off point <25 gm/L in differentiating cirrhotic ascites and malignant ascites was 76% and 100%. Positive and negative predictive values were 100% and 80% respectively. The overall accuracy was 88%. Anchinmane and Puranik[25] also reported the sensitivity and specificity of ascitic fluid total protein in differentiating transudative from exudative ascites was 84% and 70%. Positive and negative predictive values were 77% and 73.6% respectively. The overall accuracy was 81.4% at a cutoff point <30 gm/L.[25]

In this study, sensitivity and specificity of serum-ascites albumin gradient (SAAG) at a cut off value of <11 gm/L in differentiating ascites of cirrhosis from other ascites were 93.7% and 83.8% respectively. Positive and negative predictive values were 90.8% and 88.6% respectively. The overall accuracy was 90.0%. Al-Knawy[24] supported this result that the efficiency in correctly diagnosing patients with ascites caused by liver disease and those related to nonliver disease (malignancy and peritoneal tuberculosis) was 91% for SAAG at a cut off value of <11 gm/L. This result was also supported by Gupta et al.[22] that sensitivity and specificity of serum-ascites albumin gradient (SAAG) at cut off point >11 gm/L in differentiating cirrhotic ascites and malignant ascites was 94% and 91%. Positive and negative predictive values were 91% and 93.5% respectively. The overall accuracy was 92%. Anchinmane and Puranik[25] also reported the sensitivity and specificity of ascitic fluid total protein in differentiating transudative from exudative ascites was 88% and 92%. Positive and negative predictive values were 90% and 73.6% respectively. The overall accuracy was 88.4% at a cutoff point >12 gm/L. In this regard Khan et al.[18] reported sensitivity and specificity for detecting malignancy (exudative) using SAAG <11 g/L were 66.7% (18/27) and 91.5% (140/153), respectively.

In conclusion, the most frequent cause of ascites is cirrhosis of liver (63.0%) followed by abdominal tuberculosis (20.0%) and malignancy (17.0%).

Ascitic fluid total protein ($p<0.01$), ascitic fluid albumin ($p<0.01$) and serum albumin ($p=0.044$) were significantly lower in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes ($p<0.01$); while serum-ascitic albumin gradient was significantly higher in ascites of cirrhotic causes than that of ascites of non-cirrhotic causes ($p<0.01$).

The efficiency in correctly diagnosing patients with ascites of cirrhotic causes and those with ascites of non-cirrhotic causes (malignancy and peritoneal tuberculosis) was (90.0%) for serum-ascites albumin gradient at a cut off value of <11 gm/L, and 78.0% for ascitic fluid total protein at a cut off value of <25 gm/L.

Our study showed that the diagnostic accuracy of serum-ascitic albumin gradient is higher than that of ascitic fluid total protein in distinguishing ascites of cirrhotic causes from non-cirrhotic causes.

References

1. Jha R, Shrestha HG, Sayami G, Pradhan SB. Study of effusion cytology in patients with simultaneous malignancy and ascites. Kathmandu University Med J 2006; 4: 483-7.
2. Snyder CL. Ascites. eMedicine Pediatrics: Surgery. Available at: http://www.emedicine. medscape. com /article/ 779774. Accessed on 20 September 2010.
3. Shah R, Fields JM. Ascites. eMedicine Gastroenterology. Available at: http://www. emedicine. medscape.com/ article/ 779774. Accessed on 20 September 2010.
4. Khan Z, Khan I, Uddin J, Subhan F, Khan B, Khan H. Frequency of spontaneous bacterial peritonitis in cirrhotic patients with ascites due to hepatitis c virus

and efficacy of ciprofloxacin in its treatment. Gomal J Med Sc 2009; 7: 149-54.
5. Reynolds TB. Ascites. Clin Liver Dis 2000; 4: 157–68.
6. Gines P, Cardenas A, Arroyo V, Rodes J. Current concepts: management of cirrhosis and ascites. N Engl J Med 2004; 350: 1646–54.
7. Rosenberg SM. Palliation of Malignant Ascites. GastroenterolClin N Am 2006; 35: 189–99.
8. Enck RE. Malignant ascites. Am J HospPalliat Care 2002; 19: 7–8.
9. Das B, Acharya U, Purohit A. Comparative Utility of Sero Ascites Albumin Gradient and Ascitic Fluid Total Protein for Differential Diagnosis of Ascites. Indian Pediatr 1998; 35: 542-4.
10. Gurubacharya DL, Mathura KC, Karki DB. Correlation between serum-ascites albumin concentration gradient and endoscopic parameters of portal hypertension. Kathmandu Univ Med J 2005; 3: 327-33.
11. Akriviadis EA, Kapnias D, Hadjigavriel M, Mitsiou A, Goulis J. Serumlascitesalbumin gradient: its value as a rational approach to the differential diagnosis of ascites. Scand JGastroenterol 1996; 31: 814-7.
12. Runyon BA, Montano AA, Arkiviadis EA, Antillon MR, Irving MA, McHutchison JG. The serum-ascites albumin gradient is superior to the exudate-transudate concept in the differential diagnosis of ascites. Ann Intern Med 1992; 117: 215-20.
13. Kajani MA, Yoo YK, Alexander JA, Gavaler JS, Stauber RE, Dindzans VJ, et al. Serum-ascites albumin gradients in nonalcoholic liver disease. Dig Dis Sci 1990; 35: 33-7.
14. Malabu UH, Olubuyide IO, Shaibu ME, Olawuyi F. Ascites in Ibadan, Nigeria- usefulness of albumin gradient in its etiologic diagnosis. Biomed Res 2006; 17 : 105-9
15. Das BB, Purohit A, Acharya U, Treskova E. Serum-Ascites Albumin Gradient: A Predictor of Esophageal Varices with Ascites. Indian J Pediatr 2001; 68: 511-4.
16. Khan F Y. Ascites in the state of Qatar: aetiology and diagnostic value of ascitic fluid analysis. Singapore Med J. 2007; 48: 434–9.
17. Mahmood G, Debnath CR, Mandal AK. Evaluation of 100 Cases of Ascites. Mymensingh Med J 2009; 18: 62-6.
18. Khan J, Pikkarainen P, Karvonen AL, Mäkelä T, Peräaho M, Pehkonen E, et al. Ascites: aetiology, mortality and the prevalence of spontaneous bacterial peritonitis. Scand J Gastroenterol 2009; 44: 970-4.
19. Aminuddin M. Etiological basis of ascites- a study on 50 cases [dissertation]. Bangladesh College of Physicians and Surgeons, Dhaka; 2007.
20. Hoque MI. Aetiology of ascites in hospital admitted patients- a study in 100 cases [Dissertation]. Bangladesh College of Physicians and Surgeons, Dhaka; 2002.
21. Mandal AK. Ascites: Clinical presentation and causes: a study of 100 cases. [Dissertation]. Bangladesh College of Physicians and Surgeons, Dhaka; 2007.
22. Sharatchandra LK, Ningsen R, Singh YI, Narendra RK, Ranjan S, Singh WG. Serum Ascites Lipid Gradients in Alcoholic Liver Cirrhosis, Tuberculosis, and Malignancy. JIACM 2005; 6: 306-11
23. Gupta R, Misra SP, Dwivedi M, Misra V, Kumar S, Gupta SC. Diagnosing ascites: value of ascitic fluid total protein, albumin, cholesterol, their ratios, serum-ascites albumin and cholesterol gradient. J GastroenterolHepatol 1995; 10: 295-9.
24. Al-Knawy BA. Etiology of ascites and the diagnostic value of serum-ascites albumin gradient in non-alcohol liver disease. Ann Saudi Med 1997; 17: 26-28.
25. Anchinmane VT, Puranik GV. The Diagnostic Separation of Transudates and Exudates in Ascitic Fluid and Pleural Fluid. Bombay Hosp J 2001; 53: 166-72.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Evaluation of Risk Factors and Angiographic Pattern of Patients with Acute Myocardial Infarction in Young

Fakhrul Islam,[1] Muhammad Shahabuddin,[2] S M Habibullah,[3] Mohammad Sirajur Rahman,[4] Bhabani Prashad Roy,[5]

Abstract

Acute myocardial infarction is a major health problem and account for a large proportion of the total number of hospitalizations all over the world. The purpose of this study was to determine the risk factors and angiographic pattern of acute myocardial infarction in young patients. This was a cross sectional comparative study done in the department of Cardiology in Sylhet MAG Osmani Medical College Hospital from January 2010 to December 2011. Consecutive 100 patients has taken as sample and divided into two groups randomly. Group-A includes 50 young patients (≤40 years) and group-B includes 50 old (>40 years) patients of acute myocardial infarction after considering the inclusion and exclusion criteria. The difference in risk factors and angiographic pattern of two groups were analyzed and compared.

Comparing the risk factors and angiographic characteristics smoking, positive family history , Triglyceride(TG) level were significantly higher and High Density lipoprotein(HDL) level was significantly lower in group-A patients whereas hypertension, Total cholesterol(TC), Low Density lipoprotein(LDL), and diabetes mellitus(DM) were significantly higher in group-B patients. Younger patients preponderance of normal coronary artery (24.0% vs 8.0%; p<0.05) and single vessels disease (66.0% vs 26.0%; p= 0.001), whereas DVD (6.0% vs 34.0%; p=0.001) and TVD (4.0% vs 32.0%; p=0.001) were significantly higher in older patients. In young patients LAD is the highest involved vessels (48%), than RCA (40%) and LCX (20%).

Young patients have a different risk factors profile in comparison with older patients. Smoking is a strong and quite common coronary risk factor in the young patients. The majority of young patients have normal coronaries and single vessels disease.

[OMTAJ 2016; 15(2)]

1. Assistant Registrar, Department of Cardiology, Sylhet MAG Osmani Medical College Hospital.
2. Professor, Department of Cardiology, Sylhet MAG Osmani Medical College
3. Assistant Professor, Department of Cardiology, Sylhet MAG Osmani Medical College
4. Assistant Professor, Department of Cardiology, Sylhet MAG Osmani Medical College
5. Senior Consultant (Cardiology), Lakshmipur Sadar Hospital.

Introduction

Acute Myocardial infarction is defined as myocardial cell death due to prolonged myocardial ischemia. According to the WHO criteria, acute MI is diagnosed by ischemic chest pain, ECG changes and elevation of cardiac biomarkers. But recently in the universal definition some additional criteria are included like sudden cardiac death (SCD), post PCI and post CABG elevation of biomarkers and pathological finding of acute MI[1].

Acute myocardial infarction comprises acute ST elevation and Non-ST elevation MI (NSTEMI). Patients with ST-segment elevation or new or suspected new left bundle branch block on the admission ECG defined ST-segment elevation MI (STEMI). The remaining patients categorized as non-STEMI. NSTEMI is diagnosed on the basis of abnormal levels of serum biomarkers of myocardial necrosis— usually cardiac troponin I, & T, or the CK-MB without ST elevation[2]. The South Asian countries of India, Pakistan, Bangladesh, Srilanka, and Nepal contribute the highest proportion of the burden of cardiovascular diseases (CVDs) compared to any other region globally[3]. There is a common clinical perception that myocardial infarction is uncommon in young population. Epidemiological survey from many countries indicates its increasing incidence in young patients. The clinical picture of coronary artery diseases(CAD) does not differ between young and elderly patients. However, differences in rate of in-hospital complications and risk factors prevalence have been claimed by several authors[4].

Premature CAD has been defined as having an age of onset ranging from 30 to 56 years in different studies. Age cut-off value for young CAD will be defined 40 years in this study, because most studies this cut-off value selected as young[5].

Myocardial infarction without previous angina pectoris is common in young patients with coronary artery disease. The incidence of myocardial infarction (MI) and symptomatic CAD in the young is only about 3% of all CAD cases occur in age 40 or below this[6]. The incidence of CAD in the young has been reported to be 12% - 16% in Indians. 25% of acute MI in India occurs under the age of 40 years[7]. Young patients with acute coronary syndrome in Thailand comprised of 5.8% (544 patients) of which ST segment elevation myocardial infarction (STEMI) is 67%, non-ST segment elevation myocardial infarction is (NSTEMI) 20%[8].

Though data from large-scale epidemiological study are lacking, some small-scale observational studies have been done to see the incidence and prevalence of cardiovascular disease in our country. Three studies were conducted to see the prevalence of ischemic heart disease (IHD) among rural population. The prevalence of IHD was observed to be 3.3 to 13/1000 populations (average 6.56/1000)[9].

Young patients with acute myocardial infarction are most commonly seen in male and most frequent risk factor is smoking. Majority patient has single vessel disease and most frequently involve vessel is Left anterior descending artery (LAD)[10]. Identifying the risk factors such as smoking, high plasma triglyceride (TG) and low HDL cholesterol level and family history, are associated with premature CAD[11]. Smoking which has been traditionally recognised as the most common risk factor for the heart disease, has been shown to be increasingly prevalent in young adult and adolescents reaching up to 9%[12]. But in elderly the most important risk factors are diabetes mellitus, hypertension, obesity, family history, smoking and dyslipidaemia. Epidemiologic data also suggest that risk factors may be different in young vs older patients[13].

In the presence or absence of atherosclerosis, young adults can experience a myocardial infarction and are at increased risk to be misdiagnosed since they do not frequently have traditional coronary risk factors. Compared with the young acute MI significantly higher mortality and cardiovascular events in the elderly[14,15].

Coronary angiography (CAG) performed in young patients with acute myocardial infarction has identified a preponderance of single vessel disease and relatively complex stenosis morphologic features and less extensive CAD[11]. This observational study will be designed to demonstrate in detail the risk factors and angiographic profile of young patients (≤40 years) of acute MI

Materials and Methods

This cross sectional comparative study was carried at the department of Cardiology in Sylhet MAG Osmani Medical College Hospital from January 2010 to December 2011. After considering the inclusion (acute ST elevation myocardial infarction and non-ST elevation myocardial infarction) and exclusion (prior MI, structural heart diseases eg; valvular, congenital heart diseases, cardiomyopathy, major non-cardiovascular disorder such as renal impairment, any systemic infection) criteria a total of 100 cases were selected, out of which 50 cases up to the age of 40 years were included in group-A and rest 50 cases were >40 years of age included in group-B. Patients with chest pain attending the CCU initially evaluated by history taking and physical examination. ECG was done on admission. Troponin-I was measured after 6 hours of chest pain and fasting blood glucose & fasting lipid profile was measured within 24 hours of admission. Coronary angiography was done before discharge or within one month of discharge. Coronary angiographies were visually assessed by two independent experienced interventional cardiologists blinded to identify and clinical characteristics of the patients.

Results

Among the study population male was 97.0% in group-A and 90.0% in group-B. Female was 6.0% and 10.0% in group-A and B respectively.

Table I. Sex distribution of the study patients (n=100)

Sex	Group A (n=50)	Group B (n=50)	*P* Value
	n	n	
Male	47 (94.0)	45 (90.0)	0.357[ns]
Female	3 (6.0)	5 (10.0)	

Table II. Age distribution of the study patients (n=100)

Age (in years)	Group A (n=50)		Group B (n=50)		*P* value
	n		n		
≤25	4	(8.0)	0	(0.0)	0.058[ns]
26-30	8	(16.0)	0	(0.0)	0.002[s]
31-40	38	(76.0)	0	(0.0)	0.001[s]
41-50	0	(0.0)	19	(38.0)	0.001[s]
51-60	0	(0.0)	20	(40.0)	0.001[s]
61-70	0	(0.0)	10	(20.0)	0.001[s]
>70	0	(0.0)	1	(2.0)	0.500[ns]

Table III shows Smoking 82.0% in group A and 64.0% in group B. Hypertension 14.0% and 66.0% in group A and group B respectively. Dyslipidemia 22.0% in group A and 78.0% in group B. DM 10.0% and 64.0% in group A and group B respectively. Family history 38.0% in group A and 18.0% in group B.

Table III. clinical characteristics of the study patients (n=100)

Parameters	Group A (n=50)		Group B (n=50)		*P* value
	n		n		
Mean age Mean (SD)	35.04	(5.25)*	55.28	(8.74)	[a]0.001[s]
Smoking					
Smoker	41	(82.0)	32	(64.0)	[b]0.042[s]
Not Smokier	9	(18.0)	1 8	(36.0)	
Hypertension					
Hypertensive	7	(14.0)	33	(66.0)	[b]0.001[s]
Normotensive	43	(86.0)	17	(34.0)	
Dyslipidemia					
Present	11	(22.0)	39	(78.0)	[b]0.001[s]
Absent	39	(78.0)	11	(22.0)	
Diabetes mellitus					
Diabetics	5	(10.0)	32	(64.0)	[b]0.001[s]
Non-diabetics	45	(90.0)	18	(36.0)	
Family History					
Positive	19	(38 .0)	9	(18.0)	[b]0.025[s]
Negative	31	(62.0)	41	(82.0)	

Table IV. shows The mean fasting blood sugar 119.5 mmol/L in group A and 172.5 mmol/L in group B, TC 184.9 mg/dl and 234.9 mg/dl in group A and group B respectively, LDL 136.08 mg/dl in group A and 151.6 mg/dl in group B, TG 192.8 mg/dl in group A and 172.9 mg/dl in group B, HDL 36.0 mg/dl in group A and 42.1mg/dl in group B

Table IV. Mean distribution of blood sugar and lipid profile of the study patients (n=100)

Lab parameter	Group A (n=50)	Group B (n=50)	*P* value
	Mean(SD)	Mean(SD)	
Fasting blood sugar (mmol/L)	119.5(103.7)*	172.5(70.4)	0.003[s]
Total cholesterol (mg/dl)	184.9(31.4	234.9(58.5)	0.001[s]
LDL (mg/dl)	136.08(26.0)	151.6(27.2)	0.004[s]
TG (mg/dl)	192.8(41.6)	172.9(25.5)	0.004[s]
HDL (mg/dl)	36.0(6.12)	42.1(7.37)	0.001[s]

Table V. Clinical diagnosis of study patients (n=100)

Clinical diagnosis	Group A (n=50)	Group B (n=50)	*P* value
	n	n	
Acute STEMI (ST elevation myocardial infarction)	46 (92.0)*	35 (70.0)	0.005[s]
Acute NSTEMI (Non-ST elevation myocardial infarction)	4 (8.0)	15 (30.0)	

Acute STEMI 92.0% in group A and70.0% in group B, NSTEMI 8.0% and30.0% in group A and group B respectively

Table VI. Comparative analysis of involvement of major vessels (n=100)

Variables	Group A (n=50)	Group B (n=50)	*P* value
	n	n	
LAD (Left anterior descending artery)	24 (48.0)*	34 (68.0)	0.042[s]
LCX (Left circumflex artery)	10 (20.0)	28 (56.0)	0.001[s]
RCA (Right coronary artery)	20 (40.0)	27 (54.0)	0.160[ns]
LM (Left main)	2 (4.0)	2 (4.0)	0.691[ns]
Normal	12 (24.0)	4 (8.0)	0.029[s]

Table VII. Comparative analysis of angiographic profile between the two groups (n=100)

Angiographic profile Vessel No. of vessel Score	Group A (n=50)	Group B (n=50)	*P* value
	n	n	
0 Normal coronary artery	12 (24.0)*	4 (8.0)	0.029[s]
1 Single vessel disease (SVD)	33 (66.0)	13 (26.0)	0.001[s]
2 Double vessel disease (DVD)	3 (6.0)	17 (34.0)	0.001[s]
3 Triple vessel disease (TVD)	2 (4.0)	16 (32.0)	0.001[s]

Discussion

In this study mean age was 35.04(SD5.25) years in Group-A and 55.28(SD8.74) years in group. In a study showed that the mean age of young patient were 33.00(SD6.66) years and that elderly were 56.10(SD 9.37) years which closely agrees with present study.

In this study it was observed that in group-A 94.0% was male and rest 6.0% was female patients. In group-B 90.0% was male & 10.0% was female patients. Similar higher male prevalence was observed in other study where the authors observed male 91.32% in group-A & 90.26% in group-B.

In this study acute STEMI was 92.0% in group-A & 70.0% in group-B & NSTEM was found 8.0% in group- A & 30% in group- B. However STEMI was significantly higher ($p<0.05$) in both groups.

Regarding the risk factors in this current study it was observed that smoker 82.0% in group-A and 64.0% in group-B. Hypertension 14.0% in group-A, 66.0% in group-B. DM 10.0% in group-A, 64.0% in group-B. Dyslipidemia 22.0% in group-A & 78.0% in group-B. Family history of acute myocardial infarction was 38.0% in group-A & 18.0% in group-B. So the common risk factors smoking and family history of CAD were significantly ($p<0.05$) higher in group-A patients; whereas hypertension, DM, & dyslipidemia were significantly ($P <0.05$) higher in group-B patients. It has been reported more prevalence of smoking, family history, hypertriglyceridemia and low level of HDL ($P<0.01$) in younger groups. DM & hypertension more common in older groups.

Study mention that smoking, family history of CAD, dyslipidemia are important cardiovascular risk factors in young patients. Smoking 47.48% in group-A & 40.09% in group-B. DM 13.24% in group-A & 30.73% in group-B. Systemic hypertension 31.50% in group-A & 46.96% in group-B. Positive family history of premature CAD 18.26% in group-A & 10.6% in group-B

In this present study it was observed that mean blood sugar was 119.5(SD103.7) mg/dl in group-A and 172.5(SD70.4) mg/dl in group group-B. The mean total cholesterol (TC) was 184.9(SD31.4) in group-A & 234.9(SD58.5) mg/dl in group-B. The mean HDL cholesterol was 36.0(6.12) mg/dl in group-A & 42.1(SD7.37) mg/dl in group-B. The mean LDL cholesterol was 136.08(SD26.0) mg/dl in group-A & 151.6(SD27.2) mg/dl in group-B. The mean TG was 192.8(SD41.6) in group-A & 172.9(SD25.5) mg/dl in group-B. The mean blood sugar was significantly ($p<0.05$) higher in group-B. However the mean TG was significantly higher ($P<0.05$) in group-A; but TC, LDL and HDL cholesterol were significantly higher in group- B. As regards to the lipid profile, a number of investigators

Regarding the involvement of major vessels it was observed that LAD involved 48.0% and 68.0% patient in group-A and group-B respectively. LCX involved was found 20.0% in group-A and 56.0% in group-B. RCA involved was 40.0% in group-A and 54.0% in group-B. LM involved was 4.0% in group-A and 4.0% found in group-B. Normal findings were found 24.0% in group-A & 8.0% in group-B. LAD & LCX were significantly higher ($P<0.05$) in group-B patients. Normal coronary arteries were significantly ($P<0.05$) higher in group-A patients.

It is mentioned that LAD was the most commonly involved vessels followed by right coronary artery (RCA) and left circumflex artery (LCX) in both group. All these investigators have observed almost similar results with the current study.

In the present study coronary angiogram of the study population showed that normal vessels was 24.0% in group-A & 8.0% in group-B. Single vessel disease 66.0% in group-A and 26.0% in group-B. Double vessels (DVD) disease 6.0% in group-A and 34.0% in group-B. Triple vessels disease 4.0% in group-A and 32.0% in group-B. Single vessel disease was significantly ($p=0.001$) higher in group- A and double vessel (DVD) & triple vessels disease (TVD) ($p=0.001$) in group-B. Single vessel (SVD) disease was significantly higher & 61.15% in group- I & 45.56% in group- II. DVD 23.28% in group-I & 32.35% in group-II and TVD 14.61% in group-1 & 21.32% in group-II. It is mentioned that coronary angiography of patients in group-I revealed normal coronaries 36.0%, single vessel disease (SVD) 29.0% and multi-vessels disease was 34.0%. However, 53 (82.0%) patients in group- II were found to have MVD, 9 of them with significant LMCA involve. SVD, DVD and TVD 34.4% 31.3% and 12.5% in group-I respectively. In group-II SVD 31.2%, DVD 31.3% and TVD 21.8%. Normal vessel was found 21.9% in group-I and 12.5% in group-II.

In conclusion Young patients have a different risk factors profile in comparison with older patients. Smoking is a strong and quite common coronary risk factor in the young patients. The majority of young patients have normal coronaries and single vessels disease. Angiographic pattern is less severe and most frequent involved vessel is LAD in younger group.

References

1. Thygesen K, Alpert JS and White HD. Universal definition of myocardial infarction. European Heart J 2007; 28: 2525-38.
2. Kim MC, Kini AS and Fuster V. Definition of Acute Coronary Syndromes. In: Fuster V, Walsh RA, O"Rourke RA (eds), Hurst The Heart, 12th edn. 2008, Mc Graw Hill Medical New York USA, p. 1311-8.
3. Yusuf S, and Ounpuu S. Tracking the growing epidemic of cardiovascular disease in South Asia. J Am College Cardiol 2001;38: 688-9.
4. Majumder, AAS, Ali, MA and Shaha. GK. Comparison of risk factors prevalence and complications between early onset and late onset acute myocardial infarction, Bangladesh heart J 2000; 15:77-80
5. Cole JH, Miller III JI and Sperling LS. Long term follow-up of Coronary Artery Disease Presenting in Young Adults, J Am College Cardiol 2003; 41:521-5
6. Jalowiec DA and Hill JA. Myocardial infarction in the young and in women, Cardiovascular J 1989; 20:197–206.
7. Meenakshi S and Nirmal K. Premature Coronary Artery Disease in Indians and its Associated Risk Factors, Vascular Health Risk Management 2005; vol. 1, no. 3, pp. 217–25.
8. Tungsubutra W, et al., Acute Coronary Syndrome in young adults. Thai ACS Registry. J Med Assoc Thailand 2007; 90: 81-90. suppl. 1.
9. Islam MN, Ali MA and Ali M. Spectrum of cardiovascular disease: the current scenario in Bangladesh. Bangladesh Heart J 2004;19: 1-7.
10. Patwary MSR et al., Risk Factors and Pattern of Coronary Artery Disease in Young Patients with Acute Myocardial Infarction. University heart J 2008;4:20–3.
11. Chen L, Chester M and Kaski JC. Clinical Factors and Angiographic Features associated with Premature Coronary Artery Disease., Chest 1995; 108: 364-9.
12. Rahman MT et al., Angiographic Pattern of Patients with Myocardial infarction before 30 Years. Bangladesh Heart J 2009; 24:11-9.
13. Benfante RJ et al., Risk factors in middle age that predict early and late onset of coronary heart disease. J Clin Epidemiol 1989; 42:95-104.
14. Tresh DD, Brady WJ and Aufderheide TP. Comparison of Elderly and Younger Patients with out of Hospital Chest Pain. Arch Internal Med 1996; 156:1089-90
15. Mehta RH, Rathore SS and Radford MJ. Acute Myocardial Infarction in the Elderly: Difference by Age. J Am College Cardiol 2001; 38:. 736-9.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Comparative study of primary failure of arterio-venous fistula in patient starting hemodialysis with central venous catheter and without central venous catheter

Mohd. Abdus Samad Azad[1], Khusheda Hussain[2], Samiran Chandra Nath[3], Mohammad Alamgir Ahmed Chowdhury[4], Tahmid Bhuiyan[5]

Abstract

Arterio-venous fistula (AVF) represents the gold standard of hemodialysis (HD) vascular access since it is associated with less infection and offer greater dialysis adequacy. Primary AVF failure means an AVF that is never usable or fails within the first three months of its use, which is a major problem. Thrombus and infection are considered two major causes of primary failure of AVF. Most patients come to create an Arterio-venous fistula after starting HD by Central Venous Catheter (CVC), which is also considered a permanent source of infection. This study was conducted in order to identify and compare the primary failure of AVF in patients starting HD with CVC and new patients creating AVF without a CVC. A prospective Study was conducted on 65 patients from January 2014 to December 2014 who undergone AVF operation for vascular access for HD in different centers of Sylhet and fulfilling the inclusion criteria. Patients were divided in two groups. Group A contains 45 patients who came for AVF after starting HD with CVC and group B contains 20 patients who came for creating new AVF. Mean age of patient in group A was 41±12.9 and group B 34±8.9. Male are predominant in both group (3:2). Among risk factors Diabetes was present in 38 patients in group A and 13 patients in group B. All patients of both groups are found hypertensive. In group A 15(33.3%) patients came with Jugular Catheter and 30(66.7%) patients with Femoral Catheter. Primary failure of AVF in group A was 18 (40%) which is statistically significantly higher (P<0.001) than group B which was 3(15%). Common complication was infection which is 15(33.3%) in group A which also significantly higher than 2(10%) in group B. So, in conclusion, infection is considered the major cause of primary failure of AVF as patient with a central venous catheter frequently associated with bacteremia and severe infection.

[OMTAJ 2016; 15(2)]

Introduction

An arterio-venous fistula (AVF) is the connection of a vein and an artery, usually in the forearm, to allow access to the vascular system for hemodialysis, a procedure that performs the functions of the kidneys in people whose kidneys have failed. Connecting the vein and artery is a surgical procedure. Chronic kidney disease (CKD) isa condition that causes reduced kidney function over a period of time. CKD is present when a patient's glomerular filtration rate remains below 60 milliliters per minute for more than 3 months or when a patient's urine albumin-to-creatinine ratio is over 30 milligrams (mg) of albumin for each gram (g) of creatinine (30 mg/g).[1] End-stage renal disease (ESRD) means total and permanent kidney failure. When the kidneys fail, the body retains fluid. Harmful wastes build up. A person with ESRD needs treatment to replace the work of the failed kidneys by hemodialysis or kidney transplantation.[2]

The surgical creation of an AV fistula provides a long-lasting site through which blood can be removed and returned during hemodialysis. The fistula, which allows the person to be connected to a dialysis machine, must be prepared by a surgeon weeks or months before dialysis is started.[1]

The prevalence of Chronic Kidney Disease (CKD) is rapidly increasing worldwide. Population based studies shows that it is about 19% in Bangladesh.[2] According to the National Kidney Foundation (USA) One in 10 American adults, more than 20 million have some level of CKD,and three million people will be

1. Assistant professor of Surgery, Sylhet M. A. G Osmani Medical College Hospital, Sylhet.
2. Assistant Professor of Pediatrics, Park View Medical College Hospital, Sylhet.
3. Assistant Registrar, Burn and Plastic Surgery, Sylhet M. A. G Osmani Medical College Hospital.
4. Associate Professor of Nephrology, M. A. G Osmani Medical College, Sylhet.
5. Intern Doctor, Sylhet M. A. G Osmani Medical College Hospital, Sylhet.

expected to have what is known as end-stage renal (kidney) disease and most will require dialysis.[3]

Many advances in the treatment of kidney failure have been seen since the first attempts at dialysis treatments were made in the 1920s. Since then, the development of the AV fistula has marked another important advance, allowing effective treatment for longer periods of time.[4]

Hemodialysis circulates blood through a dialysis machine that contains a filter membrane. The blood is slowly pumped out of the body and into the machine for filtering. After being filtered, the blood is returned to the body through the same vascular access. About one cup of blood is outside the body at any given moment during the continuous circulation process. [5]

An access or entry to the vascular system is needed to perform the blood-cleansing role of the kidneys through hemodialysis. There are three types of vascular access:Arteriovenous fistula, Grafts andCentral Venous Catheters.[1]

An AV fistula has proven to be the best kind of vascular access for people whose veins are large enough. If the veins are not large enough, or there is no time to wait for a fistula to develop, a graft or a catheter can be used. [5]

Primary AVF failure means an AVF that is never usable or fails within the first three months of its use, which is a major problem. Thrombus and infection are considered two major causes of primary failure of AVF.[6]

Most patients come for AVF who started hemodialysis by central venous catheter which is also considered a permanent source of infection.[7]

This study was conducted in order to identify and compare the primary failure of AVF in patient starting hemodialysis with central venous catheters and new patients creating AVF without central venous catheters.

Materials and Methods

A prospective study was conducted on 65 patients from January 2014 to December 2014 who undergone AVF operation for vascular access for hemodialysis in different centers of Sylhet. Patients of 15 to 60 years of both sexes suffering from End stage renal disease (ESRD) were included in the study. Patients above 60 years, Re-do cases and poor cardiac function (EF <35%) were excluded from the study.

Patients were divided into two groups.

Group A contains 45 patients who came for AVF surgery after starting hemodialysis with central venous catheters.

Group B contains 20 patients who came for creating new AVF without central venous catheters.

The following parameters were considered- Age, sex, co-morbid conditions (DM, HTN), site of central venous catheters in group A patients, site of AVF created in both groups of patients, common complications following AVF, cause and number of primary failure were recorded.

RESULTS

The age of the patients ranged from 15 to 60 years(mean 36.4±8 SD).In Group A, the age of patients ranged from 24 to 60 years with mean age 41±12 SD; while in Group B, patients age ranged from 15 to 45 years with mean age 34±8.9 SD . The mean age of Group A is higher than group B. 30 patients were male and 15 were female in Group A and 12 patients were male and 8 were female in Group B. Male female ratio in both groups is about 3:2. Regarding co-morbid conditions, diabetes mellitus (DM) was present in 38 patients (84.44%) and 13 patients (65 %) of Group B. All the patients of both groups were found hypertensive. In group A patients, the site of central venous catheter was 30 (66.67%) in femoral vein and 15 (33.33%) in internal jugular vein. The site of AVF between left radial artery and cephalic vein was 31 (68.8%) in Group A and 18 (90%) in Group B patients while between left brachial artery and cephalic vein it was 14 (31.2%) in Group A and 02 (10%) in Group B patients. The cause of primary failure of AVF is due to infection in 15 (33.33%) patients in Group A and 02 (10%) in Group B and due to thrombosis in 03 (8%) patients in Group A and 01 (5%) in Group B. The number of primary failure in Group A was 18 (40%) and in Group B it was 03 (15%). So primary failure rate of AVF in Group A is significantly higher than Group B, P<0.001.

Table I: Co-morbid conditions

Risk Factors	Group A (45)	Group B (20)
Diabetes mellitus	38 (84.44%)	13 (65%)
Hypertension	45 (100%)	20 (100%)

Table II: The site of CVC in group A patients

Internal Jugular Vein	Femoral Vein
15 (33.33%)	30 (66.67%)

Table III: Site of AVF

Site of AVF	Group A (45)	Group B (20)
Left Radial artery & Cephalic vein	31 (68.8%)	18 (90%)
Left Brachial artery & Cephalic vein	14 (31.2%)	02 (10%)

Table IV: Primary failure rate of AVF

Complications	Group A (45)	Group B (20)	
Infection	15 (33.33%)	02 (10%)	P<0.001
Thrombosis	03 (8%)	01 (5%)	

Discussion

In this study, the age of patients ranged from 15 to 60 years (mean36.4±8 SD).In Group A mean age of patient was 41±12 SD while in Group B mean age was 34±8 SD. The mean age of Group A is higher than Group B. Guessan et al (2008) [9] found the mean age of AVF patient 40±11 SD.

Regarding sex distribution in both Group, it is almost equal, that is 3:2 (male: female).But in Africa (Guessan et al 2008) it is about 4:1.[9]

About co-morbid conditions, 38(84%) of Group A& 15(65%) of Group B patients were found diabetic .All patients of both the Groups were hypertensive.

15(33.3%) of Group Apatients came with a CVC in IJV & 30(66.7%) in Femoral vein. But in USA (Wasse et al 2007)[5] 63.64% of CVC was inserted in IJV, 4.6% in Subclavian vein, 31.79% in femoral vein. The reverse picture in our city is probably due to patient's compliance, fear and also may be lack of expertise.

In 31(68.8%) patients of Group A and 18(90%) of Group B, AVF was created in Left Radial artery & Cephalic vein. 14 (31.1%) of Group A& 02(10%) of Group B was in Left Brachial Artery & Cephalic Vein. In Ivory Coast 82.89% of AVF was constructed in Left Radial artery.[9]

In our study about 69% patients came with CVC for creating an AVF. Where in Africa it is about 92%.It is due to late referral as well as patient's fear from surgical interventions.

In Group A,15(33.3%) patients developed infection comparing with 02(10%) patients of Group B, which is significantly higher in Group A($p<0.001$).Infection rate of AVF in Africa is about 25%[9] and in USA it is about 15%.[5]

Primary failure rate of Group A patient was 40 % which is significantly higher than 15 % of Group B ($p<0.001$). So, catheter related infections are considered as the major factor for high primary failure rate of AVF. In USA it is about 39.9%[5] and in Africa (Ivory Coast) it is about 50%[9] in patient with CVC.

In conclusion, infection is considered as the major cause of primary failure of AVF in patients with a central venous catheter (CVC), as CVC is frequently associated with bacteremia and severe infection. So, before starting hemodialysis by central venous catheter, it is better to create an AVF in patient of Chronic Kidney Disease (CKD) who may need hemodialysis. Early patient referral as well as patient education and counseling are equally important for reducing the failure rate of first AVF in CKD patient.

References

1. Culvert, L.lee. Arteriovenous fistula, Gale Encyclopedia of Surgery: A Guide for Patients and caregivers. 2004.
2. Hasan M J, Kasem M A, Rahman M H, et al.Prevalence of Chronic Kidney Disease (CKD) and Identification of Associated Risk Factors among Rural Population by Mass Screening. Com Based Med J 2012;1:30-33.
3. Eknoyang G. Lavin NW, Eschbach JW, et al. Continuous quality improvement: DOQI becomes K/DOQI and is updated. National Kidney Foundation Dialysis Outcome Quality Initiative. Am J Kidney Dis 2001:37:179-94.
4. Saleh H, Fazeizadeh A, Mehdizadeh A, et al. Complication of arteriovenous fistula in Dialysis Patients. Transplantation proceedings, June 2006;38:1261-4.
5. Wasse H. Speckman RA, Frankelfield DL Rocco MV, Mcclellan WM. Predictors of delayed transition from central venous catheter use to permanent vascular access among ESRD patients. Am J Kidney Dis 2007;49:276-83.
6. Kairaitis LK,Gottleib T.Outcome and complications of temporary hemodialysis catheters. Nephrol Dial Transplant 1999;14:1710-4.
7. Arogundade FA, Sanusi AA, Badmus TA, Ibarahim A, Akisola A. Internal Jugular and subclavian catheterization: Indication, problems and prospects in a Nigerian dialysis centre. Niger post grad Med J 2006;13:26-30.
8. Di Benedetto A, Basci A, Casare S, Marselli D, ponce P, Richards N. Increased use of catheters as vascular access: Is it justified by patient's clinical condition? J Vasc Access 2007;8:21-7.
9. Guessan C A, Gnionsahe A, Guei M, Sayegh M, Kossi S, Melanie TIA, Lagou D, Sissoko H. High Failure Rate of First Arterio-Venous Fistula in patients Starting Hemodialysis Treatment: A Report from the Ivory Coast. Saudi J Kidney Dis Transpl 2008;19:384-8.

ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 15 Number 2

Pattern of dermatophytes in patients attending in out-patient department of a tertiary care hospital.

Md. Arifur Rahman[1], Osul Ahmed Chowdhury[2], Md. Moynul Haque[3], Shantanu Das[4], Premananda Das[4], Sultana Mehnaz Rahman Chowdhury[1]

Abstract

Dermatophytes are one of the most common sources of human fungal infections. Hot and humid climate in the tropical and subtropical countries like Bangladesh make dermatophytosis a very common fungal infection. This is an attempt to observe the spectrum of dermatophytes among the clinically suspected cases of dermatophytosis attending the OPD of Dermatology Department, Sylhet MAG Osmani Medical College Hospital, Sylhet. Eighty-four (84) clinically suspected cases of dermatophytosis were subjected to mycological studies. The percentage of samples positive by microscopy was 35.7%, whereas the rate of Dermatophytosis was more common in in the age group of 21-30 years and in males (53.6%). *Trichophyton rubrum*(62.5%) was the commonest etiological agent followed by *Trichophyton mentagrophytes* (27.5%), *Trichophyton tonsurans*(5%), *Microsporum gypseum* (2.5%) and *Microsporum canis*(2.5%). Isolation and species identification as the etiological agent of superficial mycoses have significant impact on effective management of superficial mycoses.

[OMTAJ 2016; 15(2)]

Introduction

The incidence of skin diseases has been reported by various authors from different parts of the world. A review of literature on the incidence of skin diseases in different countries revealed that among the fungal infections, dermatophytosis was the most predominant and *Trichophyton rubrum* was the most prevalent dermatophyte species[1,2,3]. The relative occurrence of the etiologic agents of these infections varies from country to country and from one climatic region to another[4]. In tropical countries warm and humid climate, crowded living and poor sanitary conditions promote the spread of these infections[5].

Dermatophytes are a group of closely related fungi that have the capacity of invading the keratinized tissue (skin, hair or nail) of human and other animals to produce infections known as dermatophytosis which are commonly referred to as ringworm[6,7]. They are assuming greater significance both in developed and developing countries particularly due to advent of immunosuppressive drugs and diseases[8]. The etiological agents of the dermatophytic infections include the three genera of dermatophytes- Trichophyton, Microsporum and Epidermophyton[9,10,11].

Unlike other fungi, dermatophytes are communicable and cause infections in healthy immunocompetent individuals as well as in those with immune dysfunction. Estimates suggest that 30 to 70% of adults are asymptomatic carriers of these fungi[12, 13].

Dense populations, over-crowding, climate, poverty, malnutrition, lack of health education are some crucial factors that make Bangladeshi population particularly vulnerable to fungal infection. Season changes in Bangladesh showed an impact on etiological agents causing dermatophytosis[14]. Only a few research studies are available on dermatophytes in Bangladesh[15]. The present study was done to see the spectrum of dermatophyte causing skin and nail infections and to find the most common dermatophyte species.

Material and methods

Samples were collected from 84 clinically suspected cases of ringworm infection from July 2014 to June 2015, attending OPD of Dermatology Department, Sylhet MAG Osmani Medical College Hospital, Sylhet, Bangladesh. Suspected lesions were cleaned with 70%

1. Thesis part student, Department of Microbiology,Sylhet MAG Osmani Medical College.
2. Professor of Microbiology, Parkview Medical College, Sylhet
3. Associate Professor, Department of Microbiology, Sylhet MAG Osmani Medical College.
4. Lecturer, Department of Microbiology, Sylhet MAG Osmani Medical College.

alcohol to remove the dirt and contaminating bacteria. Samples were collected in sterile paper, folded, labeled and brought to the laboratory for further processing. For direct microscopy the sample collected was screened for the presence of fungal elements by KOH mount. A drop of 20% KOH was kept on a clean, grease free glass slide. The sample (skin, hair or nail clipping) was placed in the KOH drop and slide was passed through a burner flame to hasten keratolysis. When keratolysis softened the sample, a clean glass cover slip was kept in KOH for 5-15 minutes. Each slide was thoroughly examined for the presence of filamentous, septae, branched hyphae with or without arthrospores.

For the detection of dermatophytes following media were used:

a. Sabouraud's Dextrose Agar (SDA) (Himedia)
b. Dermatophytes Test Medium (DTM) (Himedia)
c. Sabouraud's Cycloheximide Chloramphenicol Agar (SCCA) (Himedia)

Isolation of dermatophytes was confirmed by gross morphology and typical microscopic characteristics. The species were detected by wet mount test using lactophenol cotton blue, urea agar test and hair perforation test.

Result:

The age of study population ranged from 1 to 70 years with the mean age of 26.15 (SD±16.69) years. Most common age group affected was 21-30 years with 28 cases (33.3%) followed by 11-20 years with 16 cases (19%). Out of 84 cases male were 45 (53.6%) and female were 39 (46.4%) with a male-female ratio 1.15:1 [Table I].

Table I: Age and sex distribution of the study population (n=84)

Age	Male	Female	Total	Percentage	*P-Value
1 – 10	6	9	15	17.9%	
11 – 20	8	8	16	19.0%	
21 – 30	21	7	28	33.3%	
31 – 40	4	7	11	13.1%	
41 – 50	0	5	5	6.0%	
51 – 60	3	2	5	6.0%	0.027
> 60	3	1	4	4.8%	
Total	45 (53.6%)	39 (46.4%)	84	100.0%	

*X^2 (Chi-square) test was employed to analyze the data.

In this study 42 (50%) patients were in poor class and 36 (42.9%) patients were in the lower middle class of socioeconomic status while only 6 (7.1%) patients were in the upper middle class. The following bar diagram reveals the fact-

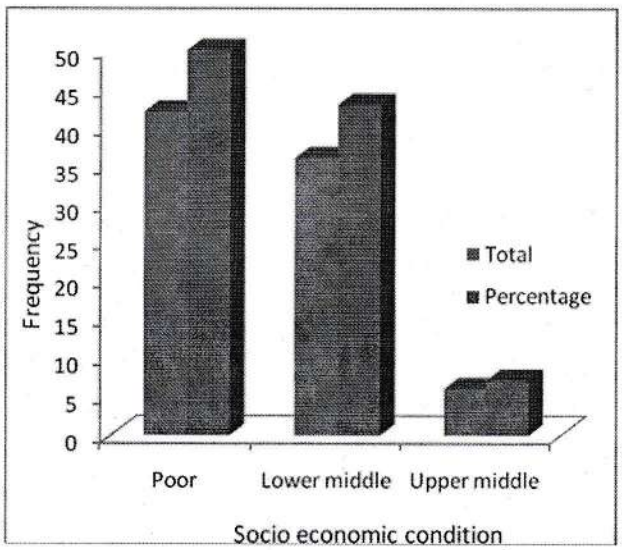


Figure 1: Bar diagram showing distribution of socioeconomic status of the patients

Out of 84 clinically suspected cases of dermatophytosis, dermatophytes were demonstrated in 30 (35.7%) cases by direct microscopy and 40 (47.6%) cases were positive by culture. 27 (32.1%) cases were positive by both microscopy and culture. Thirteen (15.5%) cases were negative for dermatophytes by direct microscopy but yielded growth on culture. Forty-one (48.8%) cases were negative by both techniques (Table II).

TableII:Comparison of results of microscopy andculture.

Culture	Microscopy +ve	Microscopy -ve	Total
+ve	27 (32.1%)	13 (15.5%)	40 (47.6%)
–ve	03 (3.6%)	41 (48.8%)	44 (52.4%)
Total	30 (35.7%)	54 (64.3%)	84 (100%)

Tinea corporis formed the major group (61.9%) followed by tinea cruris(17.9%), tinea manuum(6%), tinea unguium(4.8%), tineafacie (4.8%) and tineapedis(3.6%). Tinea capitis(1.2%) was the least common type (Table III).

Table III: Number and percentage of various tinea infections.

Clinical diagnosis	Cases	Percentage
Tinea corporis	52	61.9%
Tinea cruris	15	17.9%
Tinea manuum	5	6.0%
Tinea unguium	4	4.8%
Tinea facie	4	4.8%
Tinea pedis	3	3.6%
Tinea capitis	1	1.2%
Total	84	100.0%

Majority of the isolates were *Trichophyton rubrum* (62.5%) followed by *Trichophyton mentagrophytes* (27.5%), *Trichophyton tonsurans* (5.0%); *Microsporum gypseum* and *Microsporum canis* were found in 2.5% cases each (Table IV).

Table IV: Isolated species of dermatophytes.

Species of dermatophytes	Number	Percentage (%)
Trichophyton rubrum	25	62.5
Trichophyton mentagrophytes	11	27.5
Trichophyton tonsurans	2	5.0
Microsporum gypseum	1	2.5
Microsporum canis	1	2.5
Total	40	100.0

The details of the clinical type and their relation to the etiological agents are shown in table V. Most of the tinea corporis cases were found to be due to *Trichophyton rubrum* and *Trichophyton mentagrophytes*. Other species were *Trichophyton tonsurans* and *Microsporum gypseum*. Tineacruris was caused by *Trichophyton rubrum* and *Microsporum canis*. Species isolated from tinea facie were *Trichophyton mentagrophytes* and *Trichophyton tonsurans* and from tineacapitis, tineapedis, tinea manuum and tinea unguium was *Trichophyton rubrum*.

Table V: Number of dermatophyte species isolated from patients with tinea infections.
*X^2 (Chi-square) test was employed toanalyze the data

Species	T. corporis	T.cruris	T.capitis	T.pedis	T.manuum	T.unguium	T.facie	Total	Percentage (%)	*P-Value
Trichophyton rubrum	11	7	1	1	2	3	0	25	62.50	0.554
Trichophyton mentagrophytes	9	0	0	0	0	0	2	11	27.50	
Trichophyton tonsurans	1	0	0	0	0	0	1	2	5.00	
Microsporum gypseum	1	0	0	0	0	0	0	1	2.5	
Microsporum canis	0	1	0	0	0	0	0	1	2.5	
Total	22	8	1	1	2	3	3	40	100	

Discussion

Dermatophytes are distinct group of fungi that infect the skin, hair and nail of human and animals producing a variety of cutaneous infections known as ringworm[15,16]. Gross appearance of lesion is that of an outer ring of active, progressing infection with central healing within the ring [17].

Majority of the study subject in our study were in the age group of 21-30 years (33.3%) which is consistence with the study done by many other researchers in this field[18,19,20,21] Jesudanam et al[22] reported exaggerated outdoor physical activity, excessive sweating and constantshoe wearing habit among this age group could be the some of the contributing factors for the increased prevalence in the 21-30 years age group. However, some authors found higher incidence of such occurrence in second decade of human life[23].

In the present study, out of 84 cases, males (53.6%) were more commonly affected than females (46.4%). Male to female ratio was 1.15:1, which was comparable with other studies[3,18-20,24-25]. The highest incidence in

male may be due to increased outdoor physical activity and increased sweating. Like other subcontinental countries, Bangladeshi female patients hesitate to consult any male doctor regarding their skin infection. Nevertheless they pass through these miserable conditions they do not get treatment. Sometimes females are reluctant to consult doctors just for distance. Perhaps, for this reason, the number of female patients was found less in our study. The picture might be different in other areas. A different scenario was reported in an Iranian study presented by Jahromi and Khaksari[26].

In the present study most of the patients were from the poor (50%) and lower middle class (42.9%). Similar observation was reported by Kamothi et al[9]. The reason behind this might be living condition, large family size and close contact, either directly or by sharing facilities, including combs and towel is common between family members in low socioeconomic people[9]. As the treatment in private hospital is expensive, the poor usually come to the government hospital. It might be one of the reasons of availability of poor patients in our study. So, this picture may not represent the actual situation.

Tinea corporis was the most common clinical type encountered (61.9%) followed by tineacruris (17.9%), tinea manum unguium, tinea facie, tineapedis and tineacapitis. The above findings concurred with the reports of Singh and Beena[24] in which 58.84% tinea infection cases were tineacorporisfollowed by tineacruris. Differences in the incidence of clinical types were also observed in different studies. In the study of Rahim et al[27], tinea unguiumwas the most common clinical type.

Trichophyton rubrum (62.5%) was the common isolate followed by *Trichophyton mentagrophytes*(27.5%), *Trichophyton tonsurans*, *Microsporum gypseum* and *Microsporum canis* which was compatible to other studies[24,28,29]. However Nasimuddin et al in their study reported *Trichophyton mentagrophytes* as the predominant isolate[30]. In our study, *Trichophyton rubrum* was the most frequently isolated dermatophyte in almost all anatomical sites and recently numerous authors reported similar findings[7,26]. Specific biological characteristics and transmission dynamics which made *Trichophyton rubrum* responsible for dermatophytosis in almost all anatomical sites in our study remains to be elucidated.

In conclusion, dermatophytosis was the most predominant among superficial mycosis. The commonest clinical manifestation was tinea corporis. As the etiological agent, *Trichophyton rubrum* was the commonest species of dermatophyte isolated. The other etiological agent were *Trichophyton mentagrophytes* (27.5%), *Trichophyton tonsurans*(5%), *Microsporum gypseum* (2.5%), *Microsporum canis*(2.5%). Moreover, occurrence of dermatophytosis in the age group of 21-30 years and in males (53.6%), were very important findings of our study. Isolation and identification of these fungal species by culture and microscopic examination have significant impact on effective management of superficial mycoses. Such knowledge may help in the maintenance of skin hygiene duringhot and humid climate and thereby common irritating superficial mycoses could be avoided.

References:

1. Sarma S, Borthakur AK. A clinic-epidemiological study of dermatophytoses in Northeast India. Indian J Dermatol Venerol Leprol 2007; 73: 427-8.
2. Welsh O, Welsh E, Ocampo-Candiani J, Gomez M, Vera-Cabrera L. Dermatophytosis in monterrey, méxico. Mycoses 2006; 49: 119-23.
3. Prasad PVS, Priya K, Kaviarasan PK, Aanandhi C, Sarayu L. A study of chronic dermatophyte infection in a rural hospital. Indian J Dermatol Venerol Leprol 2005; 71: 129-130.
4. De Vrocy C. Epidemiology of Ringworm (Dermatophytes). Sem Dermato 1985; l4: 185-92.
5. Rahman SMA, Nahata MC. Treatment of Tinea capitis. Ann Pharmacother 1997; 31: 338-48
6. Gugnani HC. Non-dermatophytic filamentous keratophilic fungi and their role in human infection. In: Kushwaha RKS, Guarro J, editors. Biology of dermatophytes and other keratophilic fungi. Bilbao: Revista Iberoamerican de Microglia Apdo; 2000: 109-14.
7. Weizman I, Summerbell RC. Clinicall Microbiology Review. New York: American Society for Microbiology; 1995: 240-259.
8. Chandra J. A Textbook of Microbiology. New Delhi: Mehta publishers; 1996: 67-79.
9. Kamothi MN, Patel BP, Mehta SJ, Kikani KM, Pandya JM. Prevalence of dermatophyte infection in district Rajkot. Electronic J Pharma Therapy. 2010; 3: 1-3. Available from: URL: www.faqs.org /periodicals/201001/2075 [Accessed on 12 July 2015].

10. Nwadiro PO. Incidence of Dermatophyte infection amongst some occupational and select groups in JOS. African J Clin Expmicrobiol 2003; 4: 11-7.
11. Straten MRV, Hossain MA, Ghannoum MA. Cutaneous infections dermatophytosis, onychomycosis and tinea versicolor. Infect Dis Clin North Am 2003; 17: 87-112.
12. Woodfolk JA, Wheatley LM, Piyasena RV, Benjamin DC, Platts-Mills TA. Trichophyton antigens associated with IgE antibodies and delayed type hypersensitivity; sequence homology to two families of serine proteinases. J Biol Chem 1998; 273: 29489-96.
13. Eleski B. Tinea capitis. Dermat Clin 1996; 14: 23-31.
14. Islam KMS. A study on dermatophytes in Bangladesh. M.Phil. Thesis. 1983. University of Dhaka.
15. Neela FA, Alam MS. A survey on tinea disease in Rajshahi, Bangladesh. Univ J Zool Rajshahi Univ 2000; 19: 73-7.
16. Koneman EW. Koneman's colour atlas and textbook of diagnostic Microbiology. 6th ed. Philadelphia: JB Lippincott Company; 2006: 1151-1175.
17. Forbes BA, Sahm DF, Weissfeld AS, Bailey WR. Bailey and Scott's text book of diagnostic Microbiology. 12th ed. St. Luis: Mobsy Elsevier; 2007: 724-741.
18. Doddamani PV, Harshan KH, Kanta RC, Gangane R, Sunil KB. Isolation, identification and prevalence of dermatophytes in tertiary care hospital in Gulbarga district. People's J Scientific Res 2013; 6: 10-13.
19. Sumathi S, Mariraj J, Shafiyabi S, Ramesh R, Krishna S. Clinicomycological study of dermatophytes. Int J Pharm Biomed Res 2013; 4: 132-4.
20. Kamothi MN, Patel BP, Mehta SJ, Kikani KM, Pandya JM. Prevalence of dermatophyte infection in district Rajkot. Electronic J Pharma Therapy. 2010; 3: 1-3. Available from: URL: www.faqs.org /periodicals/201001/2075 [Accessed on 12 July 2015].
21. Mishra M, Mishra S, Singh PC, Mishra BC. Clinico-mycological profile of superficial mycoses. Indian J Dermatol Venerol Leprol 1998; 64: 283-5.
22. Jesudanam TM, Rao GR, Laksmi DJ, Kumari GR. Onychomycosis: A significant medical problem. Indian J Dermatol Venerol Leprol 2002; 68: 326-9.
23. Bindu V, Pavitran K. Clinico-mycological study of dermatophytosis in Culicut. Indian J Dermatol Veneriol Leprol 2002; 68: 259-61.
24. Singh S, Beena PM. Profile of dermatophyte infection in Baroda. Indian J Dermatol Venerol Leprol 2003; 69: 281-3.
25. Rahman AKM, Rahman MM, Haque MA. Dermatophytoses : A study in northern area of Bangladesh. Northern Med J 2000; 9: 91-95.
26. Jahromi SB, Khaksari AA. Epidemiological survey of dermatophytosis in Tehran, Iran, from 2000 to 2005. Indian J Dermatol Venerol Leprol 2009; 75: 142-7.
27. Rahim MR, Saleh AA, Miah MRA, Anwar S, Rahman MM. Pattern of dermatophytes in Bangabandhu Sheikh Mujib Medical University. Bangladesh J Microbiol 2012; 06: 11-4.
28. Kannan P, Janaki C, Selvi GS. Prevalence of dermatophytes and other fungal agents isolated from clinical samples. Indian J Med Microbiol 2006; 24: 212-5.
29. Ranganathan S, Menon T, Sentamil GS. Effect of socio-economic status on the prevalence of dermatophytosis in Madras. Indian J Dermatol Venerol Leprol 1995; 61: 16-8.
30. Nasimuddin S, Appalaraju B, Surendran P, Srinivas CR. Isolation, identification and comparative analysis of SDA and DTM for dermatophytes from clinical samples in a tertiary care hospital. J Dent Med Sci 2014; 13: 68-73.

ORIGINAL ARTICLE

The Osmani Medical
Teachers Association Journal
Volume 15 Number 2

Prospective Comparative Study Of Operative And Non-Operative Management Of Peptic Ulcer Perforation In Selected Cases

Muhammad Mofazzal Hossain[1], AKM Shafiul Alam[2], Mohammad Mahbubul Haque[3], Syed Abul Foez Showkat Uddin Ahmed[5], Abdullah Md. Abu Ayub Ansary[6], Ranjon Kumer Roy[7]

Abstract

This prospective clinical trial was carried out to compare the outcome between non-operative and operative management of perforated peptic ulcer. A total of 100 such cases were included in the study, out of them 49 received non-operative treatment and 51 operative treatment. The mean age 36.3 ± 12.1 years with male and female ratio being 24:1 in non-operative group and 6:1 in operative group. 25.5% of the patients in the non-operative group got admitted in less than 6 hours and 52.9% between 6 – 12 hours of onset of pain, while in operative group nearly 64% admitted in the hospital 12 hours after onset of pain. The difference between the groups with respect to time lapsed between onset of pain and admission was statistically significant ($p < 0.001$). About 60% the former group did not take anything orally after perforation. In the operative group majority (81.6%) received some amount of food and drinks and oral medications. The pulse, systolic and diastolic blood pressures and respiratory rate were significantly stable in the non-operative group than those in the operative group ($p<0.001$, $p<0.001$, $p=0.002$ and $p<0.001$ respectively). Nearly half (49%) of the non-operative group exhibited mild dehydration, 41.3% moderate dehydration and 7.8% severe dehydration. In contrast, 42.9% of the operative group had moderate dehydration and 57.1% severe dehydration. The difference between the groups in terms of urine output in 1st 6 hours of observation was statistically significant ($p < 0.001$). Hospital stay was significantly less in non-operative group than that in operative group (6.7 vs. 10.3 days, $p < 0.001$). Recovery does not depend on age of the patients ($p = 0.950$) and the sex also did not influence outcome (86.8% vs. 77.8%, $p = 0.809$). Uneventful recovery was significantly less in patients who delayed in getting admitted to the hospital compared to those who presented earlier ($p < 0.001$). Intake of anything orally after perforation was also observed as a barrier to uneventful recovery ($p < 0.001$).

[OMTAJ 2016; 15(2)]

1. Registrar, Department of Surgery, 300 Beded Hospital, Narayangonj.
2. Senior consultant, Department of Surgery, 300 Beded Hospital, Narayangonj.
3. Senior consultant, Department of Medicine, 300 Beded Hospital, Narayangonj.
4. Assistant Professor, Department of Hepatology, Sylhet MAG Osmani Medical College.
5. Junior Consultant (Surgery), UHC, South Surma, Sylhet.
6. Resident Surgeon, Shaheed Suhrawardy Medical College Hospital.
7. Assistant Professor, Department of Medicine, Sylhet MAG Osmani Medical College.

Introduction

Peptic ulcer disease (PUD) is a very common abdominal condition especially in middle aged male[1]. Because of changing pattern of life styles and dietary habits the incidence is increasing in this subcontinent. More over smoking and use of certain drugs such as NSAIDs, steroids also play role as aggravating factor.[2] Duodenal ulcer perforation is one of the gravest acute abdominal condition among its complications like peritonitis, septicaemia, gross water and electrolyte imbalance which increases the mortality and morbidity of the patient unless treated energetically.[3] Early diagnosis is important, as delayed diagnosis usually results in intraperitoneal sepsis.[4]

Despite dramatic improvements in peptic ulcer management in the last two decades the frequency of emergency surgery for perforated gastro-duodenal ulcer remains stable or little changed.[5] A number of opinions have been put forward in the management of duodenal perforation. Most authorities recommend surgical closure of the perforation.[6] But it has been well established that non-operative management often named as conservative management with nasogastric suction, circulatory support, anti-secretory drugs and antibiotics can be an effective treatment of perforated ulcer[7]; Non-surgical treatment of the patient with self-

sealing can be undertaken with the assurance that the seal will be secure and that the incidence of septic intra-abdominal complications will be very low. This option is particularly attractive in a case considered to be at high surgical risk because of age and/or associated disease. A surgical procedure performed to close an already-sealed perforation is unnecessary. Repeated clinical examinations to assure early progressive resolution of evidence of peritonitis are mandatory. If the physician is unable to conduct such examinations, non-surgical treatment is contraindicated[8]. Although each case must be individualized and not withstanding the foregoing, if a Gastrografin upper gastrointestinal series shows continuing free perforation despite the best conservative management, surgery may well become indicated.[9]

MATERIALS AND METHODS

This prospective clinical trial were conducted in surgery department of Sylhet MAG Osmani Medical College Hospital from August 2006 to December 2008. Age more than 18 years irrespective of sexes, early or late presentation but comparatively soft abdomen with minimum tenderness and distension and haemodynamically stable patients who responds to initial six hours treatment were included. Whereas similar illness following fever, suspected perforation with extreme shock, rigid abdomen and significant tenderness who did not respond to initial management, concurrent any other illness specially diabetes, any confusion in diagnosis, perforations due to trauma were excluded. Data presented on categorical scale were compared between groups with the help of Chi-square (χ^2) or Fisher's Exact Probability Test and quantitative data were compared between groups using Student t-Test.

RESULTS

Out of 100 cases of perforated peptic ulcers 51 received non-operative treatment and 49 operative treatment. There is no significant difference was observed between the groups in terms of age ($p = 0.698$). Majority of the patients in both groups (98%) informed that pain commenced in empty stomach ($p = 0.742$). In non-operative group over one-quarter (25.5%) of the patients admitted in less than 6 hours and 52.9% between 6 – 12 hours of onset of pain, while in operative group nearly 64% admitted in the hospital 12 hours after onset of pain and statistically significant ($p < 0.001$).

Table I. Comparison clinical characteristics between groups

Clinical presentation	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
Vomiting#	12(23.5)	7(14.3)	0.239
H/O taking NSAIDs*	2(3.9)	5(10.2)	0.202
Muscle guard/rigidity *	2(3.9)	5(10.2)	0.202
H/O peptic ulcer disease#	50(98.0)	41(83.7)	0.013

Data were analysed using **Chi-square (χ^2)**; data were analysed using **Fisher's Exact Test**
Figures in the parentheses denote corresponding **percentage.**

Table II. Comparison clinical characteristics between groups (n = 100)

Clinical characteristics	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
Distension			
Mild	29(56.9)	0(0.0)	
Moderate	21(41.2)	32(65.3)	< 0.001
Severe	1(2.0)	17(34.7)	
Fluid thrill	1(2.0)	6(12.2)	0.050
Liver dullness			
5th	16(31.4)	0(0.0)	
6th	32(62.7)	36(73.5)	
7th	3(5.9)	13(26.5)	< 0.001
Shifting dullness	20(39.2)	22(44.9)	0.565
Bowel sound	40(78.4)	28(57.1)	0.023

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

In the non-operative group about 60% of the patients did not take anything orally after perforation of peptic ulcer, 37.2% took limited amount of food and drinks and 5.9% took everything liberally. In the operative group majority (81.6%) received limited amount of food and drinks, 16.7% received food and drinks without restriction and only 2% did take nothing. As muscle guard/rigidity was compared between groups, it was observed that in 80.6% of the cases of non-

operative group it was restricted to upper abdomen and subsided with time, while in operative group majority (77.6%) of the cases exhibited generalized rigidity at initial presentation. In 19.4% of cases of non-operative group exhibited generalized rigidity but that gradually subsided and restricted to upper abdomen within two hours of initial management. On the other hand 22.4% of the cases of operative group muscle guard/rigidity was restricted to upper abdomen but they were haemodynamically unstable so that they were allocated to operative group. Comparison of haemodynamic state shows that pulse, systolic and diastolic blood pressures and respiratory rate were significantly stable in the non-operative group than those in the operative group ($p < 0.001$, $p < 0.001$, $p = 0.002$ and $p < 0.001$ respectively). Nearly half (49%) of the non-operative group exhibited mild dehydration, 41.3% moderate dehydration and 7.8% severe dehydration. In contrast, 42.9% of operative group had moderate dehydration and 57.1% severe dehydration.

Table III. Comparison urine output 6 hours between groups (n = 100)

Urine output in 6 hours	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
≤ 300 ml	1(2.0)	16(32.7)	
301 – 600 ml	41(80.4)	33(67.3)	< 0.001
> 600 ml	9(17.6)	0(0.0)	

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

Plain X-ray of abdomen in erect posture shows that over half (51%) of the non-operative group had small amount of free gas under diaphragm, while majority (73.5%) of the operative group exhibited moderate and 26.5% huge amount of gas under diaphragm ($p < 0.001$).

Table IV. Comparison of outcome between groups

Outcome	Group		p-value
	Non-operative (n = 51)	Operative (n = 49)	
Cured	45(88.2)	41(83.7)	0.511
Complications developed	6(11.8)	8(16.3)	

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

Table V. Comparison of type of complications between groups

Type of complications	Group		p-value
	Non-operative (n = 6)	Operative (n = 8)	
Residual abscess	6(100.0)	2(25.0)	0.009
Wound infection	0(0.0)	6(75.0)	

Data were analysed using **Fisher's Exact Test.**
Figures in the parentheses denote corresponding **percentage.**

Hospital stay was significantly less in non-operative group than that in operative group (6.7 vs. 10.3 days, $p < 0.001$)

DISCUSSION

Perforation is the second most important and dramatic complication of PUD that deserves immediate treatment[10]. Once the diagnosis of perforation has been made, it is generally agreed that emergency surgery should be performed[11]. Conservative treatment is reserved for patients considered to be too ill to stand the stress of surgery[12]. However, controversies surround the issue and surgeons sometimes confuse to take decision which treatment options would be correct in an individual patient. The main reason that confuses a surgeon is that in many cases when abdomen is opened for surgical repair and peritoneal toileting, the perforation has already been sealed. This phenomenon convinced us to undertake the present study to see whether there is any difference in outcome between conservative and operative management and, if so, what factors influence the outcome.

The present study did not find any difference in outcome of treatment between the two groups because non-operative treatment was continued if patient is adult>18 years, symptoms are mild and when patients were responds favorably. Majority of the subjects in either group experienced uneventful recovery and most of them were followed up to three months. Only 14 cases developed complications, and factors like delayed presentation (>12 hours of time

lapsed between onset of pain and admission) and liberal intake of foods and drinks after perfo ration were found to influence the outcome irrespective of mode of treatment. The age and sex of the patients were not observed to be the determinants of outcome.

However, a retrospective study conducted in the Department of Surgery of Khulna Medical College Hospital, Bangladesh on 491 patients of perforated peptic ulcer from July 1992 to November 2002 put different experience. In that study two options of treatment were carried out: simple closure and peritoneal lavage in 364 cases, and 127 patients managed by non-surgical methods. The outcome evaluation showed that mortality in the surgical group was significantly higher (6.8%) than that in the non-surgical group (0.02%) ($p< 0.001$). This results go in favor of non-operative management of perforated peptic ulcer and in case if the patient does not respond to conservative treatment and deteriorates, surgical treatment can be tried.[13]

Although it was documented as early as 1843 that gastroduodenal ulcers tend to seal spontaneously,[14] it was not until 1935 that Wangensteen[15] reported the success of non-operative management of patients of perforated peptic ulcer. A review of cases in 1963 demonstrated that up to 80% of patients could be managed nonoperatively.[16] Despite numerous reports of success with non-operative management, surgical intervention became the treatment of choice for perforated peptic ulcer disease because prognosis is relatively certain.[17]

Since that time, there have been 2 prospective randomized controlled trials and 3 prospective validation studies on the utility of non-operative management of perforated peptic ulcer disease. A prospective randomized controlled trial by Cocks et al[18] in 1989 demonstrated successful non-operative management in 79 (68%) of 116 patients with perforated peptic ulcer disease. This was further substantiated by a prospective randomized controlled trial by Croft et al[19] of 83 patients in which 72% in the non-operative group were successfully managed without surgery and had equivalent morbidity and mortality rates compared with the operative group. These studies have been validated in 3 further prospective uncontrolled trials by Marshall et al,[20] Gul et al,[21] and Songne et al.[22]

In our study all the patients of non-operative group were successfully managed. No one required surgical intervention. The reason of such a successful outcome may be that the patients of this group had more stable haemodynamic state than that of operative group. Hydration status was also comparatively good and urine output in first 6 hours of observation was adequate. Moreover, majority of the patients in the non-operative group were admitted in hospital within 12 hours and without generalized peritonitis, they took nothing or limited amount of food or drinks after onset of perforation.

Thus the success of non-operative management of perforated peptic ulcer lies in careful selection of patients. As pulse, blood pressure, hydration, urine output, extent of peritonitis and early presentation (<12 hours) are very much influenced by the severity of illness, they act as prognostic signs in decision making. Besides these, there are several other risk factors for failure of non-operative management like intake of food and fluid following incidence of perforation increase the extent of peritonitis. Here the role of primary health care personnel is important in the sense that they can give them proper advice not to take anything by mouth and refer immediately to nearby hospital where facilities for proper management exists. Thus we can avoid the operative assault of the patient, minimize the workload of the hospital, hospital stay and cost of the patient for treatment.

In conclusion, from the findings of the study and discussion it could be concluded that there is no significant difference in the outcome of treatment between non-operative and operative management of perforated peptic ulcers in selected cases. The findings of the study suggest that non-operative treatment should be started in almost all adult patients who are

haemodynamically stable and present early in hospital with minimal signs and symptoms. If the condition deteriorates or not responds to non-operative management in few hours of observation, immediate surgical intervention should be considered.

The study has got some limitation including non randomized trial and limited size of sample. A fare and randomized trial with a bigger sample size to recommend the criteria and formulate a scoring system for the management of the patients suffering from peptic ulcer perforation is suggested.

References

1. John NP. Stomach and Duodenum. In: R.C.G. Russell, Norman S. Williams & Christopher J.K. Bulstrode, editors. Bailey & Love's Short Practice of Surgery. 24th Edition. London: ARNOLD; 2004. p. 1026-61.
2. Gupta PKS and Azad MR. Trends of clinical presentation of peptic ulcer disease at DNMIH in recent years. Med J 1996; 32:10-4.
3. Miah MAG, Selim S, Islam S. Intestinal perforation – a clinical study in district hospital. Mymensingh Med J 1999; 8:18-21.
4. Cheefook C, Lawrence J. Conservative management of duodenal perforation following endoscopic sphincterotomy Digestive Endoscopy 2005;17:168–71.
5. Bucher P, Wassila O, Philippe M, Frederic R, Olivier H. Results of conservative treatment for perforated gastroduodenal ulcer in patients not eligible for surgical repair. Swiss Med WKLY 2007;137:337-40.
6. Graham RR. The treatment of perforated duodenal ulcers. Surg Gynecol Obstet.1937;64:235-8.
7. Crofts TJ, Park KG, Steele RJ, Chung SS, Li AK. A randomized trial of nonoperative treatment for perforated peptic ulcer. N Engl J Med 1989; 321:1050-1.
8. Arthur JD, Thomas VB, John AD. Perforated Duodenal Ulcer: An Alternative Therapeutic Plan. Arch Surg 1988;33:133-7.
9. Jamieson GG. Current Status of Indications for Surgery in Peptic Ulcer Disease. World J Surg 2000; 24:256–8.
10. Matthias H. Seelig, Stefanie KS, Christian Behr and Klaus Schonleben Comparison Between Open and Laparoscopic Technique in the Management of Perforated Gastroduodenal Ulcers. J Clin Gastroenterol 2003;37:226–9.
11. Tanphiphat C, Tanprayoon T. Surgical Treatment of perforated duodenal ulcer: a prospective trial between simple closure and definitive surgery. Br J Surg 1985; 72:70-2.
12. Keane E, Dillon B. Conservative management of perforated duodenal ulcer. Br J Surg 1988; 75:583-4.
13. Rahuman MM, Saha AK, Rahim A. Experience of peptic ulcer perforation over a decade in a teaching hospital of southern Bangladesh. Ceylon Med J 2003;48:53-5.
14. Crisp E. Cases of perforation of the stomach with deductions there from relative to the character and treatment of that lesion. Lancet 2009; 2:639.
15. Wangensteen OH. Nonoperative treatment of localized perforations of the duodenum. Minn Med 1935;18:477-80.
16. Fontaine R, Hiebel G, Lang G, Barth G. Continuous aspiration by Taylor's method for gastroduodenal ulcer perforated into the free peritoneum: complementary statistics from July, 1953 to the end of 1962 [in French]. Lyon Chir. 1963;59:682-95.
17. Lau WY, Leow CK. History of perforated duodenal and gastric ulcers. World J Surg 1997;21:890-6.

18. Cocks JR, Kernutt RH, Sinclair GW, Dawson JH, Hong BH. Perforated peptic ulcer: a deliberative approach. Aust N Z J Surg. 1989;59:379-85.
19. Crofts TJ, Park KG, Steele RJ, Chung SS, Li AK. A randomized trial of non-operative treatment for perforated peptic ulcer. N Engl J Med 1989;320:970-3.
20. Marshall C, Ramaswamy P, Bergi FG, Rosenberg IL, Leaper DJ. Evaluation of a protocol for the non-operative management of perforated peptic ulcer. Br J Surg. 1999; 86:131-4.
21. Gul YA, Shine MF, Lennon F. Non-operative management of perforated duodenal ulcer. Ir J Med Sci. 1999;168:254-6.
22. Songne B, Jean F, Foulatier O, Khalil H, Scotte M. Non-operative treatment for perforated peptic ulcer: results of a prospective study. Ann Chir 2004;129:578-82.

ORIGINAL ARTICLE

The Osmani Medical
Teachers Association Journal
Volume 15 Number 2

Evaluation of Orbital Masses in Adult Patient by Computed Tomography- a study of 50 cases

Nahar-E-Zannat[1], Md. Jalal Hossain [2], Swajal Chandra Das[1], Sudipta Gope[1], Farhana Chowdhury[3], Mostaque Ahmed Bhuiyan[1] Nila Kantha Paul[4], Md. Ashikur Rahman Mazumder[4]

Abstract

The orbit is a bony cavity within the skull that is composed of many structures which may undergo neoplastic transformation. CT-Scan for the detection of orbital mass is a very important noninvasive radiological modality. The aim of this study was to evaluate the orbital masses in adult patient by computed tomography.

This cross sectional study was conducted in the Department of Radiology & Imaging, BSMMU, Dhaka in collaboration with the National Institute of Ophthalmology, Dhaka during the period from September 2012 to February 2013. Fifty patients with suspected orbital masses aged 18 years or above were selected. CT of the orbit was done in each patient.

The mean age was 42.56 ± 12.72 years majorities were in fifth and sixth decade with female preponderance. The mean duration of symptoms was 7.7±1.2 months The most common clinical presentations were proptosis of left eye (32.0%), proptosis of right eye (20.0%) and swelling of right eye (16.0%). Majority of orbital masses were cavernous hemangiomas (28.0%) with followed by pseudotumors (20.0%) and mucocele infective (12.0%). CT scan may be considered as a single non-invasive diagnostic tool in evaluation of orbital masses which is not only characterizing the lesion but also extent of the disease process and further management of the patients.

[OMTAJ 2016; 15(2)]

1. Assistant professor, Department of Radiology & Imaging, Sylhet MAG Osmani Medical College, Sylhet
2. UH&FPO, Biswanath Health Complex, Biswanath,Sylhet.
3. Radiologist, Department of Radiology & Imaging, Sylhet MAG Osmani Medical College, Sylhet.
4. Associate professor, Department of Radiology & Imaging, Sylhet MAG Osmani Medical College, Sylhet

Introduction

The orbit is a pyramidal shaped cavity within the skull which is made up of seven bones and composed of structures such as the eyeball, the optic nerve, extraocular muscles, lacrimal gland, vessels and nerves, all of which are surrounded by orbital fat.[1] In addition to tumours arising from surrounding structures like paranasal sinuses and brain, any of these orbital contents may undergo neoplastic change. Metastasis from distant malignancies may also be deposited in the orbit.[2]

Tumors of the orbit are rare diseases constituted 3.5-4% of ophthalmic pathology.[3,4] They are a great challenge for the ophthalmologist since they present with a variety of signs and symptoms and they are often difficult to diagnose at the initial stage.[5] The major presenting symptom is proptosis, resulting from the mass effect. Changes in visual acuity or field, diplopia, extraocular motility disturbances, or pupillary abnormalities can result from invasion or compression of intraorbital contents secondary to tumor.[4]

An ophthalmologic examination and an imaging study of the orbit usually confirm the presence of a lesion and suggest a differential diagnosis. [6] Earlier modalities, such as plain radiography had limited tumour detection rates and could not reliably differentiate between benign and malignant tumours.[7] Magnetic Resonance Imaging (MRI) has been extensively explored, it provides critical anatomic information about ocular structures involved, perineural spread, and intracranial extension,[8] it is however not widely available. Ultrasonography (US) on the other hand is limited in the detection of these tumours and in determining intracranial extension. Though CT has virtually been accepted as the radio-diagnostic tool of choice for the evaluation of orbital tumours, there are scarcity of literature in our environment. This study was undertaken to evaluate orbital masses in adult patient by computed tomography.

Materials and Methods

This was a cross study conducted in the Department of Radiology & Imaging, BSMMU and National Institute of Ophthalmology, Dhaka during the study period from September 2012 to February 2013. Fifty adult patients with orbital masses aged above 18 years were included in this study.

Inclusion criteria were (1) Unilateral exopthalmos with a suspicion of posterior orbital tumor (POT), (2) POT with involvement of the 2nd, 3rd, 4th or 6th cranial nerves, (3) A lower degree of suspicion for the exclusion of POT (4) Anterior orbital tumor, (5) Trauma (6) Impairment of the 2nd, 3rd, 4th or 6th cranial nerves solely. Patients who had bony lesions or foreign body in eye were excluded.

Fifty cases of orbital masses was included using convenient sampling. Provisional diagnosis was made on the basis of clinical features and standard biochemical tests.

Procedure of CT scan of orbit: CT of the orbit was done in all cases. The patient's head is slightly reclined since the X-ray beam must be projected parallel to the orbital floor. Computed tomograms are made at an angle of approximately 20 from the orbitomeatal line which is the usual baseline for the examination of cerebral diseases. The sections are parallel to Reid's baseline. Furthermore, the head of the patient has to be inserted deeper into the water box so that the rubber head bag extends down over the eyes. Although perfect fitting of the head bag over the eyes (as is possible on the neurocranium) cannot be achieved, the scans are excellent unless the patient moves. CT of the orbit also requires a different operation technique than does CT of the brain (examinations of the orbit: 13 mm or 8 mm slices, 140 kV, .28 mAs).

After positioning the patient as described above, the examination was performed and included plain scans as well as postcontrast scans after the intravenous injection of 1 ml/kg of a 60% or 66% contrast medium. Following this contrast injection, the pathological tissue often manifests an increase of density which facilitates distinction between normal and pathological tissue.
The cross sections made at different levels include the entire orbit. Not only are the bony structures of the orbit and paranasal sinuses seen but also the bulb, the optic nerve and the eye muscles. The lens and sclera of the eye ball are clearly recognized. The CT image shows a dark area (low density) behind the bulb that corresponds to intraorbital fatty tissue. Since tumors of this region exhibit a significantly higher density than the surrounding fat, diagnosis of intraorbital mass lesions cause no difficulties.

Main outcome variables were age, sex, clinical presentation and diagnosis of orbital masses by CT.

Statistical analysis: Statistical analyses of the results were obtained by using window based computer software devised with statistical packages for Social Sciences (SPSS-16). Quantitative data were summarized as mean and standard deviation, while qualitative data were as frequency and percentage.

Ethical issue: Informed written consent was taken from each patient. Ethical clearance was taken from the local Ethical Committee.

Result

The mean age was found 42.56 ± 12.72 years (range, 18 to 60 years), majorities were in fifth and sixth decade (36%); female (58%) preponderance over male (42%) with a ration of female to male of 1.38:1. The mean duration of symptoms was 7.7±1.2 (range, 1 to 72) months, maximum 33 (66.0%) patients had symptoms 1-6 months (Table-I).
Regarding clinical presentation of the study patients, most common clinical presentation were seen in proptosis of left eye (32.0%), proptosis of right eye (20.0%) and swelling of right eye (16.0%) (Table-II).
The common orbit masses evaluated by CT were cavernous hemangiomas (28.0%) tumour, pseudotumors (20.00%) and infective mucocele (12.0%) (Table-III).

Table I. Distribution of the patients on baseline characteristics (n=50)

Baseline characteristics	Frequency	Percentage
Age		
Mean ± SD	42.56 ± 12.72	
≤30 years	10	20.0
31-40 years	13	26.0
41-50 years	9	18.0
51-60 years	18	36.0
Sex		
Male	21	42.0
Female	29	58.0
Duration (months)		
Mean ± SEM	7.7±1.2	
1 – 6 months	33	66.0
7 – 12 months	9	18.0
13 – 24 months	5	10.0
> 24 months	3	6.0

Table II. Distribution of the study patients according to clinical presentation (n=50)

Clinical presentation	Number of patients	Percentage
Dimness of vision in left eye	2	4.0
Dimness of vision on right eye	1	2.0
Intermittent redness and pain of left eye following Operation (due to trauma on left eye)	2	4.0
Loss of vision of right eye	3	6.0
Pain in both eye	6	12.0
Pain in the right eye	4	8.0
Proptosis of both eye	5	10.0
Proptosis of left eye	16	32.0
Proptosis of right eye	10	20.0
Redness of right eye	2	4.0
Swelling of both globe	1	2.0
Swelling of left eye	4	8.0
Swelling of left upper eyelid	3	6.0
Swelling of right eye	8	16.0
Swelling on superomedial aspect of right eye	2	4.0
Watering from both eye	1	2.0
Watering of right eye	3	6.0

Table III. Distribution of the study patients according to lesions of the orbit evaluated by CT (n=50)

Lesions of the orbit evaluated by CT	Number of patients	Percentage
Benign tumour		
Cavernous hemangiomas	14	28.0
Meningiomas of the optic sheath	4	8.0
Neurofibroma	2	4.0
Lipomas	2	4.0
Tumor of the lacrimal gland	4	8.0
Dermoids	2	4.0
Lipodermoid	2	4.0
Pseudotumors	10	20.0
Infective		
Cellulitis	2	4.0
Mucocele	6	12.0
Endocrine Exophthalmos	2	4.0

Discussion

Tumor demonstration by means of CT is quite easy as the density of tumors is greater than that of the surrounding fatty tissue. Thus, the normal orbit represents an ideal background for the detection of orbital lesions in the CT scan. In this current study it was observed that the mean age was found 42.56 ± 12.72 years with range from 18 to 60 years and majority of the patients having orbital masses were in fifth and sixth decade. Parashkevova and Stateva,[9] mentioned that the greater (61.0%) number being in the sixth and the seventh decade. On the other hand, Demirci et al.[10] observed lower mean age in their patients having orbital masses, which was 24.6 ± 21.4 years and Cangiarrela et al.[6] showed age belonged to 3.5 - 87 years, which are differ with the current study. They have stated that the higher age range maybe due to increased life expectancy and geographical influences may have significant impacts on orbital masses.

Regarding the sex distribution orbital mass was more common in female subject (58.0%) and male to female ratio was 1:1.38. Similarly, Cangiarrela et al.[6] showed male to female ratio was almost 1:2. However, Parashkevova and Stateva,[9] and Poso et al.[11] showed orbital mass was more frequent in male subject, where the authors obtained 60.7% and 60.0% respectively patients were male.

In this current series it was observed that more than one fourth (66.0%) patients had symptoms 1-6 months and the mean duration of symptoms was 7.7±1.2 months varied from 0.7 to 72 months. Demirci et al.[10] showed the mean duration of symptoms was 11 months before referral, which is higher with the current study.

The present series revealed that most common clinical presentation were proptosis of left eye (32.0%), right eye 20.0% and proptosis of both eye 10.0%. O'Donovan et al.[12] mentioned that one of the most common symptoms is unilateral painless proptosis, which can be accompanied by visual disturbance, palpable orbital mass, ocular mobility disturbance, hyperglobus or blepharoptosis from the secondary mass effect in the orbit. Rodman and Font,[13] and Knapp et al.[14] reported that unilateral proptosis is a well-recognized but somewhat rare presentation of multiple myeloma in ophthalmic practice. Bilateral proptosis with multiple soft tissue masses is an extremely rare presentation of multiple myeloma obtained by Kwartz and Jackson,[15] In another study

Ajithkumar et al.[16] documented that orbital myeloma most commonly presents as a unilateral solitary soft tissue intraorbital tumor which is an extension of bony deposit and is associated with bone destruction. Mass effect on the orbits can lead to clinical presentations such as proptosis, orbital pain, and diplopia. In most cases, the effect is caused by soft-tissue lesions. However, vascular lesions, either within the orbits or extraorbital, can alter the normal orbital blood flow pattern and lead to vascular engorgement and an orbital mass effect.[17,18]

In this current series it was observed that swelling of right eye 16.0%, swelling of left eye 8.0%, swelling of both globe 2.0%, and swelling of left upper eyelid 6.0%. Pain was in both eye in 10.0%, pain in the right eye 6.0% and pain and watering from right eye 2.0%. Watering of right eye and both eye were 4.0% and 2.0% respectively. Intermittent redness and pain of left eye following operation (due to trauma on left eye) in 4.0%, dimness of vision in left eye 4.0%, dimness of vision on right eye 2.0%, loss of vision of right eye 6.0%, swelling on superomedial aspect of right eye 4.0%, redness of right eye 4.0%. On symptomatology, optic nerve sheath meningioma, as it gradually increases in size, usually causes a disturbance in the blood flow to the adjacent area of the optic nerve obtained by Miller.[19] For this reason, visual symptoms and signs including impairment of visual field and acuity, disturbance in color vision and development of diplopia tend to occur earlier than proptosis. The majority of patients with orbital solitary fibrous tumor, however, present with unilateral painless proptosis as the initial clinical feature because of the mass effect compressing both eyeball and optic nerve.[19]

CT scan of orbit revealed that that majority (28.0%) patients with orbital masses had cavernous hemangiomas tumour, meningiomas of the optic sheath in 8.0% cases, neurofibroma 4.0%, lipomas 4.0%, tumor of the lacrimal gland 8.0%, dermoids 4.0%, lipodermoid 4.0%, pseudotumors in 20.00% patients, Cellulitis in 4.0%, mucocele in 12.0% and endocrine Exophthalmos 4.0%. In another study, Gyldensted, Lester and Fledelius (1977) found meningiomas in 14.9%, hemangiomas 9.0% and lymphomas 9.0%. Wende et al.[20] showed cavernous hemangiomas in 34.0% cases, meningiomas of the optic sheath 5.7%, neurinomas 9.4%, lipomas 3.8%, dermiode 3.8% and pseudotumors 2.39%. Cavernous hemangioma was found 8.0% in the study of Derirci et al.[10] The findings of the above authors regarding the lesions of the orbit evaluated by CT are comparable with the current study.

Limitations of the study were (1) The present study was conducted at a very short period due to time constrain and fund limitation, (2) Small sample size was also a limitation of the present study, (2) diagnosis was not confirmed by resection and histolpathology of the orbital lesion.

In conclusion, CT scan may be considered as a single non-invasive diagnostic tool in evaluation of orbital masses which is not only characterizing the lesion but also extent of the disease process and further management of the patients. However, further study involving multicentre and large sample is warranted.

References

1. Weisman RA. Surgical anatomy of the orbit. Otolaryngol Clin North Am 1999; 12:279-91.
2. Novitskaya E and Rene C. Enophthalmos as a sign of metastatic breast cancer. CMAJ 2013;185:1159.
3. Balabanov CH, Parashkevova B, Murgova S, Iordanov I. Tumors of the eye and adnexa. Bulgarian Rev Ophthalmol 2005;49:12-20.
4. Goldberg SH, Cantore WA. Tumors of the orbit, Curr Opin Ophthalmol 1997; 8:51-6.
5. Weisman RA, Kikkawa D, Moe KS, Osguthorpe JD. Orbital tumors. Otolaryngol Clin North Am 2001;34:1157-74.
6. Cangiarella JF, Cajigas A, Savala E, Elgert P, Slamovits TL, Suhrland MJ. Fine needle aspiration cytology of orbital masses. Acta Cytol 1996;40:1205-11.
7. Khan SN, Sepahdari AR. Orbital masses: CT and MRI of common vascular lesions, benign tumours and malignancies. Saudi J Ophthalmol 2012;26: 373-83.
8. Tailor TD, Gupta D, Dalley RW, Keene CD, Anzai Y. Orbital neoplasms in adults: clinical, radiologic, and pathologic review. Radiographics 2013; 33:1739-58.
9. Parashkevova B, Balabanov CH, Stateva D. Orbital Tumors-Clinical Cases Presentation. J of IMAB 2007;13:47-50.
10. Demirci H, Shields CL, Shields JA, Honavar SG, Mercado GJ, Tovilla JC. Orbital tumors in the older adult population. Ophthalmol 2002; 109: 243-8.

11. Poso MY, Mwanza JC, Kayembe DL. Malignant tumors of the eye and adnexa in Congo-Kinshasa. J Fr Ophtalmol 2000;23:327-32.
12. O'Donovan DA, Bilbao JM, Fazl M, Antonyshyn OM. Solitary fibrous tumor of the orbit. J Craniofac Surg 2002; 13:641-4.
13. Rodman HI, Font RL. Orbital involvement in multiple myeloma. Review of the literature and report of three cases. Arch Ophthalmol 1972;87:30-5.
14. Knapp AJ, Gartner S, Henkind P. Multiple myeloma and its ocular manifestations. Surv Ophthalmol 1987;31:343-51.
15. Kwartz J, Jackson A, Noble JL. Bilateral proptosis and corneal crystals in multiple myeloma. Eye 1993;7:191-3.
16. Ajithkumar TV, Sivasankar C, Ramachandran K. Orbital multiple myeloma: Case report and review of computed tomography features. Australasian Radiol 2002; 46: 119–20.
17. Rootman J. Vascular malformations of the orbit: hemodynamic concepts. Orbit 2003;22:103-20.
18. Stiebel-Kalish H, Setton A, Nimii Y, Kalish Y, Hartman J, Huna Bar-On R, et al. Cavernous sinus dural arteriovenous malformations: patterns of venous drainage are related to clinical signs and symptoms. Ophthalmol 2002;109:1685-91.
19. Miller NR. New concepts in the diagnosis and management of optic nerve sheath meningioma. J Neuroophthalmol 2006;26:200-8.
20. Wende S, Aulich A, Lange S, Lanksch W, Schmitt EJ. Computerized tomography in diseases of the orbital region. In: Ed.: Lanksch AW, Kazner E, eds. Cranial Computerized Tomography. New York: Springer. 1977; pp. 207-11.

INFORMATION FOR THE CONTRIBUTORS

THE OSMANI MEDICAL TEACHERS ASSOCIATION JOURNAL **(OMTAJ)** IS THE OFFICIAL ORGAN OF THE TEACHERS ASSOCIATION OF SYLHET M A G OSMANI MEDICAL COLLEGE AND IS PUBLISHED BI-ANNUALLY *(JANUARY AND JULY EACH YEAR)*

The guidelines are in accordance with the "Recommendation for the conduct, Reporting, Editing and Publication of Scholarly work in Medical Journals (ICMJE Recommendations)".[1]

Subscription

The annual subscription rate for the non-members: medical students Taka 100/- and doctors Taka 200/- only.

Submission of manuscripts

The OMTAJ considers manuscripts for publication reporting original clinical or laboratory studies, reviews, case reports, medical progress and brief communications. Manuscript must not be longer than 2700 words. Please provide a word count excluding abstract and references.

Each manuscript must be accompanied by a covering letter from the corresponding author with a statement that the manuscript has been seen and approved by all authors and the material has not been previously submitted to or published elsewhere wholly or partially. A manuscript in duplicate together with tables and illustrations along with a copy in word 97/2000/word XP format in a 3.5" diskette/ CD should be sent to the Editor.

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement) provided they do not contain any material that has been submitted or published elsewhere.

Please note the following: *Your letter must be typewritten and triple spaced; *Its text, not including references, must not exceed 250 words, if it is in reference of a recent journal article, or 400 words in all other cases (please provide a word count). *It must have no more than five references and one figure or table. *It must not be signed by any more than three authors. *Please include your full address, office-time telephone (and/or mobile) number, and e-mail address.

Preparation of the manuscript[2]

All papers must be written in English. All sections of the manuscript should be typed double-spaced, with left alignment in MS Word documents and on one side of good quality bond papers of A4 size (21x 29.7 cm) with margins of at least 2.5 cm., beginning each of the following sections on separate pages: title page, abstract, text, acknowledgments, references, individual tables, and legends for illustrations. Number pages consecutively, beginning with the title page.

Title page

The title page should contain: (1) the title of the article; (2) a short running head of fewer than 40 letter spaces; (3) name of the author (s); (4) institutional affiliation of each author; (5) name and address of the corresponding author.

Abstract

The second page should carry an unstructured abstract of not more than 150 words.

Text

The text of observational and experimental articles should be divided into sections with headings: Introduction, Materials & Methods, Results, and Discussion.

Acknowledgments

All acknowledgments including financial supports should be mentioned under the heading 'Acknowledgments' and not as a footnote on the first page or in the text.

References

Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legends by Arabic numerals (1, 2, 3....). Follow the form of references used in the *Index Medicus,* including the style of abbreviations. Try to avoid using abstracts as references: 'unpublised observations' and 'personal communication' may be inserted in the text.

Information supplied in the references section of any manuscript is not usually checked by the editorial staff, and hence, the concerned author(s) bear total responsibilities of the references.

Following are few examples of references:

1. Standard Journal Article: (List all authors when six or less; when seven or more, list only first three and add *et al).* Akhter A, Haque R, Kholil M, Sultana Z, Fakir MAH. Effects of Oral Garlic on Testicular Micro-architecture of Adult Rat. Osmani Med Teachers Assoc J 2002; 1(1): 1-3.

2. Corporate Author in Journal: Committee for Computer Application in Clinical Microbiology. Bacterial Antimicrobial Susceptibility Pattern, 1988. J Infect Dis Antimicrob Agents 1991; 8: 25-39.

3. Letter to Editor: Yagupsky P, MA Menegu. Intraluminal colonization as a source of catheter-related infection. Antimicrob Agents Chemother 1989; 33: 2025. (Letter)

4. Corporate Author in Book: World Health Organization. On being in charge: a guide to management in primary health care, 2nd ed. England: World Health Organization 1992.

5. Chapter in Book: Wenzel RP. Organization for infection control. In: Mandell GL, Douglas RG, Bennett JE, eds. Principles and practice of Infectious Disease, 3rd ed. USA: Churchill Livingstone Inc 1990: pp. 2176-80.

6. Thesis/ Dissertation: Kaplan SJ. Post-hospital home health care: the elderly's access and utilization [dissertation]. St. Louis (MO): Washington University 1995.

7. Formally published abstracts: Geesy GG, Costerton JW. Bacterial population adherent to submerged surfaces in a pristine mountain stream. Abstracts of the Annual Meeting of the American Society for Microbiology 1977: 235.

8. Articles from symposium volumes: Hamilton LD. Immunogenic polynucleotides. In: Beers RF Jr (ed). Biological effects of polynucleotides: Proceedings of the symposium on molecular biology. New York, Heidelberg, Berlin: Springer Verlag 1971: 107-28.

9. Insert from commercial product: Zyvox (linezolid). Peapack NJ: Pharmacia & Upjohn 2000 (package insert).

10. Web site: Division of tuberculosis elimination. Surveillance reports: reported tuberculosis the United States, 2000. Atlanta: Centres for Disease Control and Prevention, 2001. (Accessed June 27, 2001, at http:/ www.cdc.gov /hchstp/ tb/surv/surv2000.)

11. On-line only Journal: Scientist JQ. 2 October 1998, Posting date. History of virology. Am Virol J 1998; 30-150. *(Page numbers may not be available)* [Online.] http://cbxiou.pgr (last accessed October 10, 1998)

12. Online version of print journal: Scientist JQ. History of clinical microbiology. Clin Microbiol 1999; 100: 123-345. [Online]

13. Online version of print books: Scientist JQ. 4 October 1998, Posting date. Culturing methods, 750-800 In: Gavier (ed). Practical procedures for Laboratory, 5th ed. [Online.] DEF Publishing Co. Boston, Mass Http://cbxiou.pgr. (last accessed October 10, 1998).

Abbreviations

Except for units of measurements, abbreviations are discouraged. The first time an abbreviation appears, it should be preceded by the words for which is stands.

Drug name

Generic names should generally be used. When proprietary brands are used in research, include the brand name in parentheses in the methods section.

Permissions

Materials taken from other sources must be accompanied by a written statement from both author and publisher to the OMTAJ for reproduction.

Review and Action

Manuscripts are examined by editorial staff and sent to reviewers.

Rejected manuscripts will only be returned if accompanied by stamped and self-addressed envelope.

Letters about potentially acceptable manuscripts will be sent to the corresponding author after the review process is complete.

---

[1] Recommendation for the conduct, Reporting, Editing and Publication of Scholarly work in Medical Journals (ICMJE Recommendations). International Committee of Medical Journal Editors. Available at www.icmje.org/icmje-recommendations

[2] Additional information regarding manuscript reparation and relevant editorial policy is available in the editorial office

Covering letter to the Editor for submission of manuscripts

To
The Editor
The OMTAJ
Sylhet MAG Osmani Medical College,
Sylhet-3100

Subject: Submission of manuscript

Dear Sir,

I/we am/ are submitting along with a manuscript for Original Article/ Case Report/ Review Article/ Medical Progress/ Occasional Note/ Others, having title:----------
---

---------- for publication in the OMTAJ.

I/we mention that the manuscript had not been **submitted to,** or **accepted for publication,** or **published** in any form, in any other journal partially, or completely.

We also agree with the following orders of authorship to the manuscript, and we also certify that the authorship will not be contested by anyone whose name is not listed here.

Authors chronology Signature

1.

2.

3.

4.

5.

Corresponding Author: ----------Signature:----------

Address:----------