KuMCJ

Kushtia Medical College Journal

Volume 1 **Number 1** **June 2017**

An Official Organ of Kushtia Medical College

E-mail: kumcjournal.bd@gmail.com

KUSHTIA MEDICAL COLLEGE JOURNAL (KuMCJ)

Vol. 1, No. 1, June 2017

OFFICIAL ORGAN OF KUSHTIA MEDICAL COLLEGE, KUSHTIA

Tel. +8807163071

E-mail. Kumcjournal.bd@gmail.com

EDITORIAL BOARD

ADVISORY BOARD

KUSHTIA MEDICAL COLLEGE JOURNAL

Volume 1, Number 1, June 2017

Published By
Professor. Dr. Md. Saad Ahamed
On behalf of Kushtia Medical College, Kushtia
Bangladesh

Address of Correspondence
Editor in Chief
Kushtia Medical College Journal
Kushtia Medical College, Kushtia
Tel: +88071-63071
Email: kumcjournal.bd@gmail.com

Sponsored by
BRB Hospitals Ltd, Dhaka, Bangladesh as part of their continuing educational support.

Confusions in Biochemistry Laboratory Reports

Biochemical investigation reports frequently create confusion and sometimes we are confused to such an extent that decision becomes a great puzzle to solve. Biochemical investigations are inseparable part of our medical practice. But very often we the medical personnel are to face confusing situations due to mismatch of biochemical reports with patients' clinical status. We become embarrassed and sometimes we repeat the investigations in same or different labs; and again, variation of report of a same patient's sample in same/different laboratories make us puzzled. In fact those laboratory reports are very often not actually mismatched with patients' clinical status; rather, those are some extreme normal values. But we are to face confusion. To solve the problem, we should have some idea about (a) reference range, (b) allowable error recommendations for different laboratory procedures and (c) Sources of errors in collecting specimen and sending for analysis.

(a) Reference Range[1] - Reference Range does not represent 100% of the healthy population! Many quantitative measurements in populations exhibit Gaussian frequency distribution (bell-shaped curve); this is called 'normal distribution' (normal curve) and is characteristic of biological variables. A normal distribution can be described by a mean value (which is placed at the centre of the bell-shaped curve on the x axis) and a standard deviation (SD, which describes the width of the bell-shaped curve). Mean ± 1 SD represents about 68% of population, Mean ± 2 SD represents about 95% of population and Mean ± 3 SD represents about 99.5% of population. By convention, the 'normal range' is defined as those values which encompass the central 95% of the population, i.e. the values within 2 SDs above and 2SDs below the mean. So, 2.5% of the normal population will have their values above and 2.5% will have their values below the normal range. But this 5% is not belongs to anything abnormal or any diseased entity. This is why, it is more appropriate to say 'reference range' rather than 'normal ranges'. Abnormal results, i.e. the results those lying beyond 2 SDs from the mean, may occur either due to the person is one who lies within that 5% of the normal population whose test result is outside the reference range, or due to he or she is suffering from a disease characterized by a test result different from the reference range. Test results in 'abnormal population' (disease) also have a bell-shaped distribution with a different mean and different SD which even may overlap with reference range.

(b) Our next issue is allowable error recommendations for different laboratory procedures.[2] i.e. Laboratory Procedure related error-limits approved by **C**linical **L**aboratory **I**mprovement **A**mendments 19**88 (CLIA'88).** Here, in this clause, we are allowing some procedure related error as acceptable. There are several chemical methods to measure a substance. Procedure related error in the measurement of any substance varies among different methods. In the estimation of different substances, Clinical Laboratory Improvement Amendments of 1988 **(CLIA '88)** fixed up the limits of "Method related errors" for all biochemical parameter. If the error of any method crosses the limit, the method is declared as obsolete and is discarded.[2] Ultimately CLIA '88 has accepted some method related errors as usual phenomenon. Methods with error-limits beyond the acceptable range are already declared as obsolete. But we have to know the error limits of the running methods. Otherwise, borderline reports will confuse us at any time. When any borderline report creates confusion, we are to add or detract the allowable error and then again cross-check the value with patient's clinical status – confusion may be resolved.

Allowable error limits of some biochemical parameters are mentioned here:[2] For glucose at decision level 50 mg/dl to 200 mg/dl, error limit is 10%. For creatinine at decision level 1.0 mg/dl, error limit is 0.3 mg and at decision level 3.0 mg/dl, error limit is 15% with maximum total error being 0.45 mg. For BUN at decision level 27 mg/dl, error limit is 9% with maximum total error being 2.4 mg. For bilirubin at decision level 1.0 mg/dl, error limit is 0.4 mg and at decision level 20 mg/dl, error limit is 20% with maximum total error being 4 mg. For total protein at decision level 7.0 gm/dl, error limit is 10% with maximum total error being 0.7 gm. For albumin at decision level 3.5 gm/dl, error limit is 10% with maximum total error being

0.35 gm. For uric acid at decision level 6.0 mg/dl, error limit is 17% with maximum total error being 1.02 mg. For total cholesterol at decision level 200 mg/dl, error limit is 10% with maximum total error being 20 mg. For HDL cholesterol at decision level 35 mg/dl, error limit is 30% with maximum total error being 10.5 mg. For triglyceride at decision level 160 mg/dl, error limit is 25% with maximum total error being 40 mg. For ALT at decision level 50 U/L, error limit is 20% with maximum total error being 10 mg. For AST at decision level 30 U/L, error limit is 20% with maximum total error being 6 mg. For Alkaline Phosphatase at decision level 150 U/L, error limit is 30% with maximum total error being 45 mg. For Amylase at decision level 100 U/L, error limit is 30% with maximum total error being 30 mg. For LDH at decision level 100 U/L, error limit is 30% with maximum total error being 30 mg. For CKMB, maximum total error is 3 SD. For sodium at decision level 130-150 mmol/l, maximum total error being 4.0. For potassium at decision level 3.0-6.0 mmol/l, maximum total error being 0.5. For chloride at decision level 90-110 mmol/l, error limit is 5% with maximum total error being 4.5-5.5 mmol/l. For bicarbonate decision level 20-30 mmol/l, maximum total error is 5. For calcium at decision level 7.0-13 mg/dl, maximum total error is 1.

(c) Now, Let's have a view to few specimen collections: (1) Preconditions to be fulfilled for OGTT[3] – unrestricted usual standard diet for previous 3 days, overnight fasting, no exercise or smoking in the morning and must remain confined to seat for 2 hours between fasting sample and 2 hour sample. Delay in serum separation without anticoagulant sodium fluoride use may interfere with glucose level. (2) Preconditions to be fulfilled for estimation of serum creatinine level –exercise/or excessive heavy physical activity or muscle injury/crush in previous 24 hours, very low fluid intake resulting in oligurea, haemolysis during procedure will cause transient rise of creatinine which may be confused with renal functional impairment. (3) Precautions to be taken for estimation of serum bilirubin – avoid haemolysis during procedure, exposure of sample to light after specimen collection. (4) Precautions to be taken for serum (venous) electrolytes and arterial blood gas analysis – drawing blood gently with wide bore needle, pricking the needle in a piece of paraffin or soap, if possible bending the needle after blood drawing, maintaining the needle-side of the syringe up and sending specimen with the whole syringe to laboratory. – Like these four examples, many other biochemical investigations require some specific precaution of their own. If not maintained, the result will be erroneous.

Of course, "To err is human". And, personnel related to a biochemical lab may also make error at any time. But, as we observe, even when performance of the laboratory personnel and procedure is within acceptable limits, problems related to any one of the three points which have already been described above, is sufficient to make us confused (reference range, "allowable error recommendations" for different laboratory procedures and sources of errors in collecting specimen and sending for analysis). Now, let us think about what may happen if they play role in combination.

KuMCJ, June 2017; Vol. 1(1):1-2

Dr. Abu Hena Mostafa Kamal
MBBS, M.Phil. (Biochemistry)
Assistant Professor of Biochemistry
Kushtia Medical College, Kushtia

References

1. Cumming AD, Noble SIR. The Normal (or Reference) Range. In: Good medical practice. In: Walker BR, Colledge NR, Ralston SH & Penman ID, editors. Davidson's Principles and Practice of Medicine, 22nd edition. London: Churchill Livingstone ELSEVIER, 2014; pp-5.

2. Koch DD, Peters T. Evaluation of Methods – with an Introduction to Statistical Technique. In: Burtis CA, Ashwood ER & Bruns DE, editors. Tietz Fundamentals of Clinical Chemistry, 6th edition. London: Saunders, 2008; pp-212.

3. Pearson ER, McCrimmon RJ. Diabetes mellitus. In: Walker BR, Colledge NR, Ralston SH & Penman ID, editors. Davidson's Principle and Practice of Medicine, 22nd edition. London: Churchill Livingstone ELSEVIER, 2014; pp-809.

Correlation of Splenic Size and Portal Vein Diameter with Endoscopic Findings of Oesophageal Varices in Patients with Cirrhosis of Liver

Moniruzzaman M[1], Parvin SS[2], Mondal D[3], Akramuzzaman M[4], Rahman A[5], Ahamed MS[6]

Abstract

Liver cirrhosis is prevalent in both developed and developing countries with variation in underlying causes. This descriptive cross sectional study was carried out to assess the correlation of splenic size and portal vein diameter with the severity of oesophageal varices in 110 purposively selected patients of liver cirrhosis with suspected oesophageal varices attended at Radiology and Imaging department, Bangabandhu Sheikh Mujib Medical University (BSMMU) during September 2014 to August 2016. Upper GIT endoscopy was performed to identify as well as for grading of oesophageal varices. Ultrasonography was carried out to measure size of spleen and portal vein diameter. Out of 110, 94(85.5%) patients had oesophageal varices. Of them, 36(38.2%) patients had grade III, while 29(30.9%) each had grade II and grade I oesophageal varices. The mean portal vein diameter was 13.8 mm with a standard deviation of 2.8 mm, whereas mean splenic size was 13.7 cm with a standard deviation of 2.4 cm. The mean portal vein diameter was found 13.0±2.4 mm in grade I, while 13.9±1.5 mm in grade II and 15.3±3.0 mm in grade III varices with a statistically significant ($p<0.05$) difference. There was a statically significant moderately positive correlation ($r=0.520$; $p=0.001$) between oesophageal varices and portal vein diameter. The mean splenic size was found 13.5±2.2 cm in grade I, while 14.0±2.0 cm in grade II and 14.6±2.4 cm in grade III varices which was statistically significant ($p<0.05$). There was a moderately positive but statistical insignificant correlation ($r=0.348$; $p=0.110$) between oesophageal varices and splenic size. Transabdominal ultrasonography is useful diagnostic modality in the evaluation of severity of oesophageal varices by measuring portal vein diameter and spleen size. It can be used as a reliable and noninvasive screening tool in patients with suspected portal hypertension due to cirrhosis that help in appropriate management in majority of cases.

Key Words: Oesophageal varices, Cirrhosis, Portal Vain Diameter, Splenic size.

KuMCJ, June 2017; Vol. 1 (1): 03-07

Introduction

In underdeveloped countries liver cirrhosis is a major cause of morbidity and mortality due to unawareness of the patients, inadequate facilities and financial implications associated with the disease. In western world, chronic alcohol consumption accounts for majority of the cases, however in developing countries, infection by hepatotropic viruses like Hepatitis B and Hepatitis C are most likely responsible. Liver cirrhosis often is an indolent disease, most patient remains asymptomatic until the occurrence of decompensation, characterized by ascites, spontaneous bacterial peritonitis, hepatic encephalopathy or variceal bleeding due to portal hypertension[1]. The prevalence of varices in patients with cirrhosis is approximately 60 to 70 percent, while the risk of bleeding is 25 to 35 percent. The incidence of oesophageal varices increases by nearly 5 percent per year, and the rate of progression from small to large varices is approximately 5 to 10 percent per year[2].

Approximately one third of patients with oesophageal varices experience variceal bleeding, which in up to

Authors' Affiliations:

1..Dr. Md. Moniruzzaman, MD (Radiology & Imaging), Junior Consultant, Radiology & Imaging, 250 Bedded General Hospital, Kushtia.
2. Dr. Salma Shahnewaz Parvin, MD (Radiology & Imaging), Assistant Professor (Radiology & Imaging), Faridpur Medical College, Faridpur.
3. Dr. Dhrubo Mondal, , MD (Radiology & Imaging), Medical Officer (Radiology & Imaging), Kushtia Medical College, Kushtia.
4. Dr. Md. Akramuzzaman, MD (Internal Medicine), Assistant Professor (Medincine), Kushtia Medical College, Kushtia.
5. Dr. Ashadur Rahman. MD(Neurology), Lecturer, (Community Medicine), Kushtia Medical College, Kushtia.
6. Professor Dr. Md. Saad Ahamed. M.Phil. (Radiology & Imaging), Professor (Radiology & Imaging), Kushtia Medical College, Kushtia.

Address of correspondence: Dr. Md. Moniruzzaman, Junior Consultant, Radiology & Imaging, 250 Bedded General Hospital, Kushtia, Mobile no.- 01715143875, E-mail: dr.mmzaman@gmail.com

70 percent of the survivours is followed by repeated bleeding episodes[3]. Due to this reason, a screening endoscopy is indicated in all patients with newly diagnosed cirrhosis. It has also been suggested to repeat endoscopy at every 2 to 3 years interval in patients without varices, whereas at 1 to 2 years interval in patients with small varices to evaluate the progression[4]. Endoscopic examination is an invasive as well as an expensive procedure used for screening oesophageal varices in all patients with chronic liver disease[5]. Consequently, mass screening using endoscopy will significantly increase the cost. Factors like platelet count, coagulation profile, splenic size and portal vein diameter correlate with the risk of variceal bleeding. Alternatively, these less invasive procedures can be used for assessing patients with cirrhosis. Due to cost effectiveness and to limit the number of endoscopic procedures, these parameters become more significant and carry paramount importance to avoid unnecessary intervention[6].

Portal hypertension results from increased resistance to portal blood flow, and has the potential complications of variceal bleeding and ascites. The splenoportal veins increase in caliber with worsening portal hypertension and partially decompress by opening a shunt with systemic circulation like a varix. In the event of portosystemic shunting, there is a differential decompression across the portal vein and splenic vein, with a resultant decrease in the ratio of portal vein diameter to that of splenic vein[7]. So, this study was designed to measure spleen size and portal vein diameter by transabdominal ultrasonography to assess their correlation with the severity of oesophageal varices.

Materials and methods

This descriptive, cross-sectional type of observational study was carried out in the Department of Radiology and Imaging, BSMMU, Dhaka in collaboration with Department of Hepatology, BSMMU, Dhaka during the period of September 2014 to August 2016. A total of 110 patients of liver cirrhosis with suspected oesophageal varices with age between 17 to 87 years were purposively selected for the study. Patients with non-cirrhotic portal hypertension, hepatic failure and who refused to undergo endoscopic examination were excluded from the study. Ultrasonography was conducted to measure portal vein diameter and splenic size. Oesophago-gastro-duodenoscopy report was recorded for the detection of the oesophageal varices and portal vein diameter with a high level of significance (p=0.001). The mean splenic size was found 13.5±2.2 cm in oesophageal varices grade I, 14.0±2.0 cm in grade II and 14.6±2.4 cm in grade III with a statistically significant difference ($p<0.05$).

and was assessed for correlation with the ultrasonography findings. In addition, socio-demographic variables were recorded using case record form. Data were cross-checked for completeness, consistency and relevancy. Data were analyzed using the software SPSS. Based on nature, data were expressed in mean, standard deviation and proportion, and presented in tables and figures as appropriate. Statistical tests like Pearson's correlation, ANOVA were carried out to measure the statistical significance.

Results

This descriptive, cross-sectional study was carried out in the Department of Radiology and Imaging, Bangabandhu Sheikh Mujib Medical University, Dhaka during September 2014 to August 2016 among 110 purposively selected liver cirrhosis patients with suspected oesophageal varices to measure the diameter of portal vein and splenic size by ultrasonography, and to conduct endoscopy for detection of oesophageal varices. Moreover, ultrasonography and endoscopic findings were correlated for the evaluation of severity of oesophageal varices.

The mean age was found 47.0 years and a standard deviation of 14.3 years with range from 20 to 80 years. Majority 88(80.0%) patients were males, while and 22(20.0%) were females with a male-female ratio of 4:1. More than one third 42 (38.2%) patients were farmers. More than two-thirds 75(68.2%) patients had HBV related cirrhosis, 20(18.2%) had HCV, 12(10.9%) had NBNC, 1(0.9%) had NASH, 1(0.9%) had haemochromatosis and 1(0.9%) had Budd-Chiari syndrome.

The mean portal vein diameter was 13.8 mm with a standard deviation of 2.8 mm, whereas mean splenic size was 13.7 cm with a standard deviation of 2.4 cm. Out of 110 patients, 94(85.5%) had oesophageal varices, while the rest 16(14.5%) did not have oesophageal varices. Of 94, 36(38.2%) had grade III oesophageal varices, while 29(30.9%) each had grade I and II oesophageal varices (Fig.1). The mean portal vein diameter was found 13.0±2.4 mm in grade I oesophageal varices, while 13.9±1.5 mm in grade II and 15.3±3.0 mm in grade III varices (Fig.2) with a statistically significant difference (p=0.001).

There was a moderately positive correlation (r=0.520) between oesophageal varices

and portal vein diameter with a high level of significance (p=0.001). The mean splenic size was found 13.5±2.2 cm in oesophageal varices grade I, 14.0±2.0 cm in grade II and 14.6±2.4 cm in grade III with a statistically significant difference (p<0.05). There was a moderately positive correlation (r=0.348) between oesophageal varices and splenic size with a statistically insignificant difference (p=0.110).

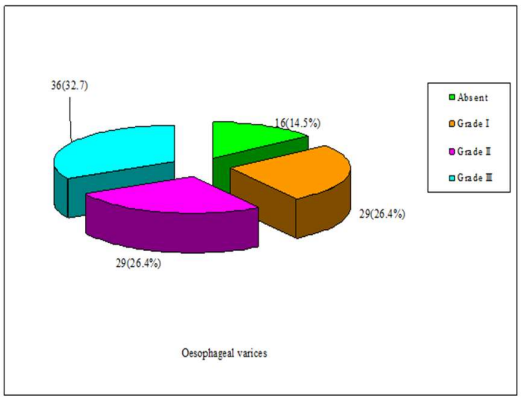


Figure 1: Pie chart showing oesophageal varices status of the patients

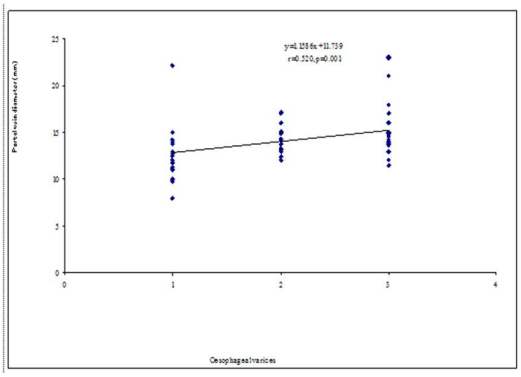


Figure 2: Scatter diagram showing correlation between oesophageal varices and portal vein diameter

Discussion

The mean age of the patients was found 47.0 years with a standard deviation of 14.3 years. Makarem et al.[8] and Mahassadi et al.[9] observed mean age of 48 years and 49 years respectively which are consistent with the current study findings. Majority 88(80.0%) patients were males, while 22(20.0%) were females which was similar to findings of study conducted by Masjedizadeh et al.[10], Sumon et al.[11] and Said et al.[12] It was observed that over two-thirds 75(68.2%) patients had HBV- related cirrhosis, 20(18.2%) had HCV, 12(10.9%) had NBNC, 1(0.9%) had NASH, 1(0.9%) had haemochromatosis and 1(0.9%) had Budd-Chiari syndrome. Study conducted by Tarzamni et al.[13] revealed that Hepatitis B virus (HBV) infection was the main cause of cirrhosis in most of study patients. This might be attributed to purposive sampling technique followed for the study and small sample size.

It was observed that mean portal vein diameter was found 13.8 mm and standard deviation of 2.8 mm with range from 8.0 to 23.1 mm. Makarem et al.[8] determined the mean Portal vein diameter of 13.04±1.9 mm, whereas Tarzamni et al.[13] revealed 13.5±2.4 mm which are in line with the current study findings. Hong et al.[15] revealed that mean portal vein diameter of 12.6±1.9 mm with a range from 9 to 26 mm. It was observed that mean splenic size was found 13.7±2.4 cm with a range from 7.7 to 20.0 cm. Tarzamni et al.[13] found the mean splenic size of 15.7±2.4 cm, while Umar et al.[6] observed a splenic size of more than 13 cm in 75.0 percent cases having mean splenic diameter of 14.5±2.39 cm. Out of 110 patients, 94(85.5%) had oesophageal varices. Of 94, 36(38.2%) had grade III, while 29(30.9%) each had grade I and II oesophageal varices. Study carried out by Baig et al.[16] revealed that patients with osesophageal varices, 36(34.0%) patients had grade I, while 54(50.9%) had grade II varices and 16 (15.1%) had grade III varices. Mahassadi et al.[9] observed no varices in 23.4%, grade I in 6.3%, grade II in 48.6% and grade III in 21.6% patients with liver cirrhosis which are almost similar to the current study findings. Said et al.[12] observed large oesophageal varices in 29.2% patients. Zaman et al.[17] observed that endoscopic findings included oesophageal varices in 68.0% of patients, whereas in 30.0% patients the varices were large in size. Masjedizadeh et al.[10] observed that out of 140 cirrhotic patients 85.0% had oesophageal varices. Of them, in 51.1% cases varices were small, whereas in 33.6% it as large. Tarzamni et al.[13] observed small varices (Grade I and II) in 58.8%, while large (Grade III and IV) in 22.3%, and it was absent in 18.8% patients that match with the current study findings.

The mean portal vein diameter was found 11.7±1.5 mm varied from 10 to 15 mm in with absence of oesophageal varices, whereas 13.0±2.4 mm varied

from 8 to 22.2 mm in oesophageal varices grade I, 13.9±1.5 mm varied from 12.0 to 17.2 mm in grade II and 15.3±3.0 mm varied from 11.5 to 23.1 mm in grade III. The difference was significant ($p<0.05$) that was increased with higher grade. Sarwar et al.[14] reported that patients with portal vein diameter more than 11 mm are more likely to have oesophageal varices. They observed mean portal vein diameter (PVD) of patients without gastro-oesophageal varices was 11.5±1.5 mm, whereas patients with varices was 14.0±1.1 mm where the difference was statistically significant ($p<0.05$). They concluded that gastro-oesophageal varices developed in cirrhotic patients with portal vein diameter more than 11.5 mm and with spleen size over 13.1 cm.

A moderately positive and statistical significant correlation ($r=0.520$; $p=0.001$) was observed between oesophageal varices with portal vein diameter. This is vindicated by findings of study carried out by Mandal et al.[18] ($r = 0.707$; $p<0.05$) and Rani et al.[19]

Furthermore, it was observed that the mean splenic size was found 11.8±2.0 cm with a range from 8.5 to 16.1 mm in patients without oesophageal varices, while 13.5±2.2 cm with a range from 7.7 to 17.6 mm in grade I oesophageal varices, 14.0±2.0 cm with a range from 9.0 to 20.0 mm in grade II and 14.6±2.4 cm with a range from 11.0 to 20.0 mm in grade III that was significant ($p<0.05$) and was increased with higher grade of oesophageal varices. This is supported by the study findings of Sudhindra et al.[20] who observed that portal vein diameter more than 13 mm, spleen size over 14 cm and splenic vein over 14 mm are indicators of oesophageal varices.

It was revealed that a moderately positive but statistical insignificant correlation ($r=0.348$; $p=0.110$) between oesophageal varices with splenic size. Rani et al.[19] also found was a positive correlation between grading of oesophageal varices and portal vein size that is very much similar to current study findings. Mandal et al.[18] observed a positive correlation between grading of oesophageal varices with splenic size ($r=0.467$; $p<0.05$)[19] which is inconsistent with the current study findings.

Conclusion

It can be concluded that the transabdominal ultrasonography is useful diagnostic modality in the evaluation of severity of oesophageal varices by measuring portal vein diameter and spleen size. It is noteworthy that transabdominal ultrasonography can be used as a noninvasive and reliable tool for screening in patients with suspected portal hypertension due to cirrhosis. So, it can help in appropriate management in majority of cases.

References

1. Heidelbaugh JJ, Bruderly M. Cirrhosis and chronic liver failure: part I. Diagnosis and evaluation, *Am Fam Physician,* 2006; 74(5): 756-762.

2. Amico GD, Morabito A. Noninvasive markers of oesophageal varices: Another round, not the last, *Hepatology*, 2004; 39(1): 30-34.

3.Tsokos M, Turk EE. Esophageal variceal hemorrhage presenting as sudden death in outpatients, *Arch pathol Lab Med*., 2002; 126: 1197-1200.

4. Nashat EH, Abd-Elaziz H, Sabry M, Ibrahim AA. Non endoscopic parameters of oesophageal varices and portal hypertensive gastropathy, *Nature and science,* 2010; 8(6): 43-50.

5. Kishimoto R, Chen M, Ogawa H, Wakabayashi MN, Kogutt MS. Oesophageal varices: evaluation with transabdominal US, *Radiology*, 1998; 206(3): 647-650.

6. Umar A, Qazi FA, Sattar RA, Umar B. Non-invasive parameters for the detection of variceal bleed in patients of liver cirrhosis, an experience of a tertiary care hospital in Pakistan', *Asian Journal of Medical Sciences*, 2015; 16(1): 61-66.

7. Gaduputi V, Patel H, Sakam S, Neshangi S, Ahmed R, Lombino M, Chilimuri S. Value of portal venous system radiological indices in predicting oesophageal varices, *Clinical and experimental gastroenterology,* 2015; 88: 89-93.

8. Makarem AE, Shatat ME, Shaker Y, Abdel Aleem AA, El Sherif, AM, Abdel Moaty M, *et al*. Platelet count/bipolar spleen diameter ratio for the prediction of oesophageal varices: The special Egyptian situation, *Hepat Mon,* 2011; 11(4): 278-284.

9. Mahassadi AK, Bathaix FY, Assi C, Bangoura AD, Kissi HY, Toure A, *et al*. Usefulness of Noninvasive Predictors of Oesophageal Varices in Black African Cirrhotic Patients in Cote d Ivoire (West Africa)', *Gastroenterology Research and Practice,* 2012; 1-11.

10. Masjedizadeh AR, Hajiani E, Nejad PA, Hashemi J, Shayesteh AA, Yasin Z. Efficacy Platelet/Spleen Diameter Ratio for Detection of Oesophageal Varices in Cirrhotic Patients', *Journal of Gastroenterology and Hepatology Research,* 2013; 2(5): 590-592.

11. Sumon SM, Sutradhar SR, Chowdhury M, Khan NA, Uddin MZ, Hasan, MI, *et al*. Relation of different grades of oesophageal varices with Relation of different grades of oesophageal varices with Child-Pugh classes in cirrhosis of liver. , 2013; 22(1): 37-41.

12. Said HEE, Elsayed EY, Ameen A, Elal, HA. Cytopenia As A Predictor Of Oesophageal Varices In Patients with Liver Cirrhosis', *Report and Opinion,* 2010; 2(7): 17-23.

13. Tarzamni MK, Som MH, Farhang S, Jalilvand M. Portal hemodynamics as predictors of high risk oesophageal varices in cirrhotic patients, *World J Gastroenterol,* 2008; 14(12): 1898-1902.

14. Sarwar S, Khan AA, Alam A. Non-endoscopic prediction of presence of oesophageal varices in cirrhosis, *J Coll Physicians Surg Pak,* 2005; 15(9): 528-531.

15. Hong WD, Zhu QH, Huan, ZM, Chen XR, Jiang ZC and Xu SH, *et al*. Predictors of oesophageal varices in patients with HBV-related cirrhosis: a retrospective study, *BMC Gastroenterol.*, 2009; 9(11): 116-121.

16. Baig WW, Nagaraja MV, Varma M, Prabhu R. Platelet count to spleen diameter ratio for the diagnosis of oesophageal varices: Is it feasible?', Can J Gastroenterol, 2008; 22(10): 825-828.

17. Zaman A, Hapke R, Flora K, Rosen, HR, Benner K. Factors predicting the presence of oesophageal or gastric varices with advanced liver disease, *Am J Gastroenterol,* 1999; 94(11): 3292-329.

18. Mandal, L, Mandal SK, Bandyopadhyay D, Datta S. Correlation of portal vein diameter and splenic size with gastro-oesophageal varices in cirrhosis of liver, *JIACM*, 2011; 12(4): 266-270.

19. Rani KVLS, Sudarsi B, Siddeswari R, Manohar S. Correlation of Portal Vein Size with Oesophageal Varices Severity in Patients with Cirrhosis of Liver with Portal Hypertension, *International Journal of Scientific and Research Publications,* 2015; 5(1): 1-5

20. Sudhindta D; Lakshman Kumar et al. JAPI, 2003;5.

ORIGINAL ARTICLE

Clinico-Epidemiological Study of Herpes Zoster: An Observational Study in Bangladesh

Chowdhury MMH[1], Mahmud MM[2], Haque S[3], Hazra SC[4], Chakrabarty H[5], Sultana S[6], Ahmed S[7]

Abstract

Herpes Zoster is a common infectious disease in our locality. It is caused by reactivation of latent varicella zoster virus from spinal ganglia and is usually self-limiting in healthy adults. A very few study had been conducted in Bangladesh and in south East Asia in this regard.

Aim of the study was to observe the clinical presentation, dermatomal distribution and the epidemiological factors of Herpes Zoster.

An observational study was conducted on 110 patients with Herpes Zoster attending 250 Bedded District Hospital Manikgonj and Kushtia Medical College Hospital, Kushtia. The cases were diagnosed clinically by two dermatologists and consent for participation was taken accordingly. One hundred and ten consecutive cases attending at hospitals were examined clinically and relevant history was taken and was recorded in a preformed data collection sheet. All data were preserved in a secured computer device. All valid data were analyzed and published in text and tables.

In this study there was 66% (73) male patients and 34% (37) was female and mean age of respondent was 37.08 (±17.13). Maximum patients (20%) came to hospital at day 4. The common presenting feature was pain (found in 65% cases) and the common skin lesion was vesicular (45%). History of chicken pox was positive in 70% cases. Maximum affected area of body was thoracic segments (57%) and the common dermatome was T5, T10 and L2. In follow up, 8 cases reported post herpetic neuralgia.

Males are more vulnerable than females. Pain is the main symptom of the disease, patients usually come to hospital at 4th day of appearance of skin lesion. Most common skin lesion is vesicular and commonly affected segment of body is thoracic part.

Key Words: Herpes Zoster, Dermatome, Chicken Pox.

KuMCJ, June 2017; Vol. 1 (1):08-12

Introduction

Herpes zoster is a localized, blistering and painful rash caused by reactivation of varicella zoster virus (VZV). It is characterized by dermatomal distribution, ie the blisters are confined to the cutaneous distribution of one or two adjacent sensory nerves. Herpes zoster is also called shingles. VZV is also called herpes virus 3, and is a member of the Herpesvirales order of double-stranded DNA viruses. Anyone that has previously had varicella (chickenpox) may subsequently develop zoster. This can occur in childhood but is much more common in adults, especially the elderly. People who have had zoster rarely get it again; the risk of getting a second episode is about 1%. Herpes zoster often affects people with poor immunity.[1]Varicella commonly occurs in childhood and is highly contagious affecting children aged under12 years. An increasing incidence of varicella in children has been found over the time with around 95% adults immune to the condition at the age of 20-29 years.[2]

After primary infection VZV remains dormant in dorsal root ganglia nerve cells in the spine for years before

Authors' Affiliations:

1. Dr. Md. Mostaque Hassan Chowdhury, Associate Professor, Dermatology, Kushtia Medical College, Kushtia.
2. Dr. Md. Mostaque Mahmud, Assistant Professor, Dermatology Bangabandhu Sheikh Mujib Medical University. Shahbagh, Dhaka.
3. Dr. Shawana Haque, Department of Biochemistry, BIRDEM General Hospital, Dhaka.
4. Dr. Samaresh Chandra Hazra, Assistant Professor, Dermatology, Dhaka Medical College Hospital.
5. Dr. Harashit Chakrabarty, Assistant Professor, Dermatology, Satkhira Medical College Hospital, Satkhira.
6. Dr. Sharmin Sultana, Medical Officer, Dermatology, BSMMU.
7. Dr. Sohel Ahmed, Junior Consultant, Dermatology, Upazilla Health Complex, Mirpur, Kushtia.

Address of correspondence: Dr. Md. Mostaque Hassan Chowdhury, Associate Professor, Dermatology, Kushtia Medical College, Kushtia. Mobile: 01944780451, E.mail: dr.mostaque69@gmail.com

it is reactivated and migrates down sensory nerves to the skin to cause herpes zoster. It is not clear why herpes zoster affects a particular nerve fibre.

The clinical presentation of herpes zoster depends on the age and health of the patient and which dermatome is affected. The first sign of herpes zoster is usually pain, which may be severe, relating to one or more sensory nerves. The pain may be just in one spot or it may spread out. The patient usually feels quite unwell with fever and headache. The lymph nodes draining the affected area are often enlarged and tender. Within one to three days of the onset of pain, a blistering rash appears in the painful area of skin. It starts as a crop of red papules. New lesions continue to appear for several days within the distribution of the affected nerve, each blistering or becoming pustular then crusting over.[3]The chest (thoracic), neck (cervical), forehead (ophthalmic) and lumbar/sacral sensory nerve supply regions are most commonly affected at all ages. Frequency of ophthalmic herpes zoster increases with age. Herpes zoster occasionally causes blisters inside the mouth or ears, and can also affect the genital area. Occasionally there is pain without rash. Pain and general symptoms subside gradually as the eruption disappears. In uncomplicated cases, recovery is complete within 2-3 weeks in children and young adults and within 3-4 weeks in older patients.

Post-herpetic neuralgia is defined as persistence or recurrence of pain in the same area, more than a month after the onset of herpes zoster. It becomes increasingly common with age, affecting about a third of patients over 40 years. It is particularly likely if there is facial infection. Post-herpetic neuralgia may be a continuous burning sensation with increased sensitivity in the affected areas or a spasmodic shooting pain. The overlying skin is often numb or exquisitely sensitive to touch. Sometimes, instead of pain, the neuralgia results in a persistent itch (neuropathic pruritus).

Antiviral treatment can reduce pain and the duration of symptoms if started within one to three days after the onset of herpes zoster. Acyclovir 800 mg 5 times daily for 7 days is most often prescribed. Valacyclovir and famcyclovir are also effective. The efficacy of prescribing systemic steroids is unproven. Note that herpes zoster is infectious to people who have not previously had chicken pox. Because the risk of severe severe and post-herpetic neuralgia is less likely to develop. Herpes zoster vaccination is contraindicated in complications from herpes zoster is more likely in older people, those aged over 60 years might consider zoster vaccine, which can reduce the incidence of herpes zoster by half. In people who do get herpes zoster despite being vaccinated, the symptoms are usually less immunocompromised patients due to the risk of disseminated herpes zoster infection.[4]

There are very few hospital-based studies on the epidemiology and clinical profiles of HZ in Bangladesh and even in South Asia. Although a relatively common cause of morbidity, especially among the elderly, contemporary estimates of HZ in different groups are lacking.

The objective of this study is to create awareness about herpes zoster and associated risk factors through highlighting its epidemiological and clinical profile so that it can be managed properly.

Materials and methods

This descriptive cross sectional study conducted among purposively selected 110 diagnosed cases of herpes zoster at the outpatient department of Sadar Hospital, Manikgonj and Kushtia Medical College Hospital, Kushtia in the year 2013 and 2014. Epidemiological and clinical data were collected by dermatologists of 2 different hospital of Bangladesh by using case record form. Treatment was given by dermatologist and each case was followed up for two weeks. Considering ethical issues carefully, informed consent was taken from each patient. After collection, data was checked for inadequacy, irrelevancy and inconsistency. Irrelevant data was discarded. All data were preserved in a secured computer device. Data were analyzed with appropriate statistical tools and SPSS program and published in text and tables.

Results

This study was conducted on 110 diagnosed cases of herpes zoster. Among them male (66%) participants was more than female. Male to female ratio was 1.97: 1. The mean age of participants was 37.08 (±17.13) years. The mean duration of disease was 5.88 (±3.40) days. (Table II)

Table I: Distribution of cases by age.

Age group in years	Frequency	Percentage
0-10	7	6.36
11-20	19	17.27
21-30	15	13.63
31-40	21	19.09
41-50	22	20
51-60	20	18.18
>60	5	4.54
Total	**110**	**100**

Table II: Distribution of cases by duration of infection.

Duration of infection (days)	Frequency	Percentage
2	5	4.5
3	18	16.4
4	22	20.0
5	20	18.2
6	12	10.9
7	17	15.5
8	1	0.9
9	1	0.9
10	3	2.7
12	3	2.7
13	3	2.7
15	4	3.6
22	1	0.9
Total	**110**	**100**

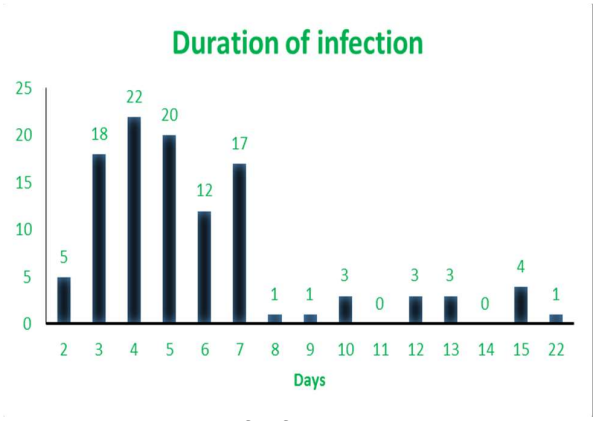


Figure 1: Duration of infection

Table III: Distribution of cases according to sex, symptoms, skin lesions, side of involvement and history of chicken pox.

Features	Frequency	Percentage
Sex		
Male	73	66
Female	37	34
Symptoms		
Pain	72	65.45
Burning	12	10.90
Pain and Burning	12	13.63
Pain and itching	5	4.54
Asymptomatic	6	5.45
Skin lesions		
Papular	15	13.63
Vesicular	50	45.45
Papulovesicular	27	24.54
Crusted	18	16.36
Lesion at side of body		
Right	52	47.27
Left	58	52.73
History of chicken pox		
Positive	77	70
Negative	33	30

Table IV: Dermatomal distribution with nerve root.

Root of Nerve	Dermatome	Frequency	Percentage
Trigeminal	Ophthalmic	7	6.36
	Mandibular	2	1.82
	Maxillary	1	.91
Cervical	C2	2	1.82
	C3	2	1.82
	C4	4	3.64
	C5	3	2.73
	C6	2	1.82
Thoracic	T1	1	.91
	T2	3	2.73
	T3	4	3.64
	T4	5	4.55
	T5	13	11.82
	T6	5	4.55
	T7	3	2.73
	T8	7	6.36
	T9	1	.91
	T10	11	10
	T11	5	4.55
	T12	5	4.55
Lumber	L1	7	6.36
	L2	9	8.18
	L3	5	4.55
	L4	1	.91
	L5	1	.91
Sacral	S1	1	.91
Total		110	100

Dermatomal distribution of herpes zoster infection was shown in table IV and in figure 2. The most affected area of body is thoracic area 57% next most affected area was lumber 21%. The most common nerve root was T5, T10 and L2 accordingly.

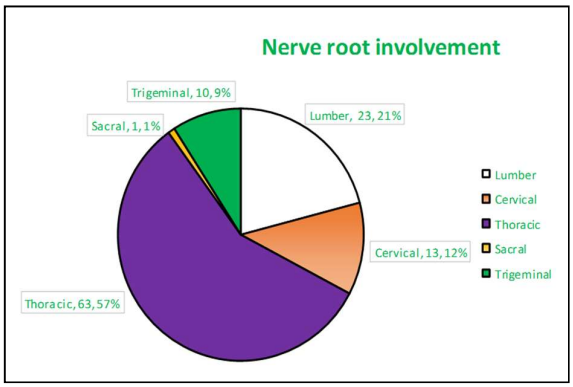


Figure 2: Affected segment of body.

Discussion

Herpes zoster is a common viral infection in our country but well-designed hospital based study of herpes zoster is lacking. This study was conducted on 110 cases of HZ to take a step towards finding the clinical and epidemiological parameter.

There were 73(66%) male and 37(34%) female participants. Male to female ratio was 1.97: 1.
A similar study conducted in Nepal on 174 patients where the male female ratio was 2.16:1.[7] In another study on 274 patients, Kayastha and Shilpakar has revealed the male to female ratio was 2.46:l.[8] Those two findings are very much similar to the findings of this study. So an inference can be drawn that the male are more prone to get HZ infection.

The mean age of patients was 37.08 (±17.13) years. The minimum age was 6 and the maximum age was 70 years. Age range of participants was 64 years. The highest number of cases occurring in 5th decade (20%) but 19% in 4th decade, 18% in 6th decade and 17% was in 2nd decade. Overall 77.28% of our HZ cases was in up to 5th decade.

In a study, Kayastha et.al[8] found that the mean age of patients was 34.5 (±17.85), the minimum age was10 years and maximum age was 81 years and 75% patients was aged below 50 years.
In a similar study of 274 cases of HZ, Kayastha and Shilpakar[8] has revealed that the mean age at presentation was 34.6 (±16.7) years with the highest number of cases occurring in 3rd decade (32.85%). They had reported that more than 82% of the patients were below 50 years of age. Those two study findings are nearer to our results.

The mean duration of disease was 5.88 (±3.40) days in their study. The highest number (22%) of patients came to hospital on 4th day of eruption of skin lesions. The most of the HZ patients (84%) attended at hospital within first week of disease.

Pain was the major complaints of patients (65.45%) but 5.45% patients found asymptomatic. The other symptoms were the burning and itching.

In the current study the highest number of patients came to us with vesicular type of skin lesion (45.45%), papular and crusted type was found in 13.63% and 16.36% cases respectively.

History of chicken pox was positive in 70% cases whereas scar mark of chicken pox was found in 22% cases. Skin lesions found at the left side of body in 52.73% cases and 100% patients was right handed person.

It was found that highest number of patients (57%) was affected in thoracic dermatomes. Lumber, cervical, trigeminal and sacral involvement was found in 21%, 12%, 9% and 1% accordingly. The most common infected dermatomes were T5 (11.8%), T10 (10%) and L2 (8.18%).

In a study Laxmisha et.al[9] described the dermatomes involved in decreasing frequency were thoracic (60%), followed by ophthalmic (15%) and sacral (12.5%). In a similar study Kayastha et al.[8] found that the maximum number of their cases had involved of thoracic dermatomes (56.32) which was followed by cervical (16.67%), cranial (12.07%) and lumbo-sacral (14.94%) dermatomes which is

similar to the study by De Biasi RL et al. According to De Biasi et al. ,14 to 20 % of patients had disease in the distribution of a cranial nerve.[10]

Conclusion

Males are more vulnerable than females. Pain is the main symptom of disease; patients usually come to hospital at 4th day of appearance of skin lesion. Most common skin lesion is vesicular and commonly affected segment of body is thoracic part.

References

1. Maia C, *et al.* Complicated infection by varicella-zoster virus, Acta Med Port 2015 Nov-Dec; 28(6): 741-748.
2. Bonhoeffer J, Baer G, Muehleisen B, Aebi C, Nadal D, Schaad U, et al. Prospective surveillance of hospitalizations associated with varicella-zoster virus infections in children and adolescents. Eur J. 2005; 164: 366– 70.
3. Papaloukas O, Giannouli G, Papaevangelou V. Successes and challenges in varicella vaccine. TherAdv Vaccines. 2014; 2: 39-55.
4. Merck Sharp & Dohme (New Zealand) Limited. 2016. Zostavax Data Sheet. 18 April 2016.URL: medsafe.govt.nz/profs/Datasheet/z/zostavaxinj. pdf.
5. Laxmisha C, Thappa DM, Jaisanker TJ. The spectrumof varicella zoster virus infection: a hospital based clinicin south India. Indian J Dermatol 2004; 49(1): 28 - 31.
6. Usha G, Srinivasulu P, Bharathi G. Clinico Epidemiological Study of Herpes Zoster in HIV Era in a Tertiary Care Hospital in South India. IOSR Journal of Dental and Medical Sciences 2015; 14(2) Ver. VII: 32-35.
7. Kayastha BMM, Shrestha P, Shrestha R, Lama L. Changing Profile of Herpes Zoster in Nepal: A Hospital-Based StudyNepal Journal of Dermatology, Venereology & Leprology 2009; 8(1): 1-4.
8. Kayastha BMM, Shilpakar R. Clinico-epidemiological profile of Herpes zoster in Nepal: a hospital- based study. Nepal J Dermatol Venereol Leprol 2005; 5 (5): 7-14.
9. Laxmisha C, Thappa DM, Jaisanker TJ. The spectrum of varicella zoster virus infection: a hospital based clinic in south India. Indian J Dermatol 2004; 49(1): 28 - 31.
10. De Biasi RL, Solbrig MY, Tyler KL. Infections of the Nervous System. B. Viral infections. In: Bradley WG, Daroff RB, Fenichel GM et al., eds. Neurology in Clinical Practice, 4th ed. Butterworth, Heinemann; 2004: 1515-154.

ORIGINAL ARTICLE

Bacterial Pathogens Isolated from Urine and Antimicrobial Susceptibility Pattern in Kushtia, Bangladesh

Rahman N[1], Ahmed AS[2], Munir MS[3], Akramuzzaman M[4], Hossain MD[5], Islam MM[6]

Abstract

Urinary tract infection is a very common infectious disease. Distribution of uropathogens and their antimicrobial susceptibility pattern vary with time and geographical area. Local epidemiological data is important for proper selection of empiric drug. This descriptive cross-sectional study was conducted to determine common uropathogens and their susceptibility pattern in Kushtia, Bangladesh. Urine samples were collected from patients with suspected UTI attending for urine culture during March 2016 to October 2016. All samples from male and female of age ranging 13 to 90 years were included in the study. A total of 470 urine samples were processed. Isolation and identification of bacteria followed by antimicrobial susceptibility was done. Of 470 urine samples, 112(23.8%) yielded significant growth. Out of 112 culture positive samples 61(54.5%) were from female and 51(45.5%) were from male patients. Most common organism was Escherichia coli 86(76.8%) followed by Klebsiella 15(13.4%). Pathogens showed good sensitivity to imipenem, amikacin, nitrofurantoin and gentamicin, and low sensitivity to second and third generation cephalosporins and ciprofloxacin.

Key Words: Bacterial Pathogens, Urine, Antimicrobial Susceptibility.

KuMCJ, June 2017; Vol. 1 (1):13-16

Introduction

Urinary tract infection (UTI) is defined as multiplication of organisms in the urinary tract. UTI is the most common bacterial infection managed in general medical practice and accounts for 1–3% of consultations. Up to 50% of women have a UTI at some time. UTI causes morbidity, and in a small minority of cases renal damage and chronic renal failure.[1] The diagnosis of UTI is usually made based on the presence of signs and symptoms and confirmed by urine culture with significant bacteriuria supported by high level pyuria.[2] UTIs including catheter- related bacteriuria constitute the most common nosocomial bacterial infection with an average rate of 13.1 cases per 1000 hospital discharges.[3] Proper and adequate antibiotic therapy is necessary to treat and prevent recurrence of UTI.[4] Reporting of antimicrobial susceptibility testing of the urinary tract infection is usually achieved 48 hours following sampling, and therefore, in the majority of UTIs, the treatment decision is empirical. The empirical therapy of UTI relies on the predictability of the causative agents and the knowledge of their antimicrobial susceptibility patterns.[5,6] This study was aimed at determining the common etiological agents of urinary tract infection and their antibiotic susceptibility pattern in Kushtia, Bangladesh.

Authors' Affiliations:

1. Dr. Nazmin Rahman, M.Phil (Microbiology), Assistant Professor, Microbiology, Kushtia Medical College, Kushtia.
2. Dr. Md. Ashraf-Ul-Haque, FCPS,MS (Surgery), Associate Professor, Surgery, Kushtia Medical College, Kushtia.
3. Dr. Abu Saleh Ahmed, FCPS (Medicine); MD (Nephrology), Assistant Professor, Nephrology, Kushtia Medical College, Kushtia.
4. Dr. Md. Sirajum Munir, MD (Cardiology), Assistant Professor, Cardiology, Kushtia Medical College, Kushtia.
5. Dr. Md. Akramuzzaman, MD (Medicine), Assistant Professor, Medicine, Kushtia Medical College, Kushtia.
6. Dr. Md. Deldar Hossain, M.Phil (Clinical Pathology), Assistant Professor, Pathology, Kushtia Medical College, Kushtia.
7. Dr. Md. Monir-Ul-Islam, MPH, Ph.D, Assistant Professor, Community Medicine, Kushtia Medical College, Kushtia.

Address of correspondence: Dr. Nazmin Rahman, M.Phil (Microbiology), Assistant Professor, Microbiology, Kushtia Medical College, Kushtia. Mobile: 01954-848843
E.mail: nazminfairy@gmail.com

Materials and Methods

This was a descriptive, cross sectional study, carried out at Amin Diagnostic Center, a renowned diagnostic center in Kushtia, Bangladesh which caters for all the laboratory tests requested from different hospitals and

clinics in and around the city. Urine samples were collected from patients with suspected UTI attending for urine culture during the period of March 2016 to October 2016. All samples from male and female of age ranging 13 to 90 years were included in the study. Clean catch mid-stream urine samples were collected in disposable sterile containers. In case of catheterized patient urine was collected with sterile syringe from rubber tubing of the catheter. Information regarding patient's name, age, sex, occupation, consulting doctor or clinic or hospital, date and time of sample collection were recorded. A total of 470 urine samples were processed. Semiquantitative culture was done. A standard bacteriological loop was used to inoculate blood agar and MacConkey's agar plate. The plates were incubated aerobically at 37°C overnight. Growth of single bacterial species from the urine sample with a count of $\geq 10^5$ cfu/ml was considered significant to indicate UTI. Identification of the isolated organism was done on colony morphology, gram staining and relevant biochemical tests. Antimicrobial susceptibility testing of the isolates was performed by disc diffusion method using Mueller-Hinton Agar according to the recommendations of the National Committee for Clinical Laboratory Standards.[7] The antibiotic discs used in antibiogram were amikacin (10 µg), amoxicillin-clavulanic acid (30 µg), cefixime (5 µg), ceftazidime (30 µg), ceftriaxone (30 µg), cefuroxime (30 µg), ciprofloxacin (5 µg), cotrimoxazole (25 µg), doxycycline (30 µg), gentamicin (10 µg), levofloxacin (5 µg), Imipenem (10 µg) and nitrofurantoin (300 µg). Data were recorded in a predesigned data sheet. Descriptive results were expressed as frequency and percentage.

Results

A total of 470 urine samples were processed, of which 196 (41.7%) were from males and 274 (58.3%) were from females. Out of 470 samples 112 (23.8%) gave significant growth of pathogenic bacteria.

Among 112 culture positive specimens 51(45.5%) were from males and 61(54.5%) were from females. Among the female samples yielding positive culture 13-20 years age group showed 6(9.8%), 21-30 years age group 19(31.2%), 31-40 years age group 15(24.6%), 41-50 years age group 8(13.1%), 51-60 years age group 7(11.5%), 61-70 years age group 5(8.2%), and 71-80 years age group showed 1(1.6%) of isolation of uropathogens. Among the culture positive male samples 13-20 years age group showed 1(2.0%), 21-30 years age group 3(5.9%), 31-40 years age group 9(17.6%), 41-50 years age group 5(9.8%), 51-60 years age group 15(29.4%), 61-70 years age group 7(13.7%), and71-80 years age 9(17.6%) and 81-90 years age group showed 2(4.0)% of isolation of uropathogens. (Table-I).

Table I: Distribution of culture positive samples according to age groups and gender (n=112)

Age group in years	Female	Male
11 – 20	6 (9.8)	1 (2.0)
21 – 30	19 (31.2)	3 (5.9)
31 – 40	15 (24.6)	9 (17.6)
41 – 50	8 (13.1)	5 (9.8)
51 – 60	7 (11.5)	15 (29.4)
61 – 70	5 (8.2)	7 (13.7)
71 – 80	1 (1.6)	9 (17.6)
81 – 90	0 (0.0)	2 (4.0)
Total	61 (54.5)	51 (45.5)

Figures within parentheses indicate percentage.

The highest growing urinary pathogen was Escherichia coli 86(76.8%), then Klebsiella 15(13.4%), Enterobacter 4(3.5%), Proteus 2(1.8%), Acinetobacter 2(1.8%), Pseudomonas 1(0.9%), Citrobacter 1(0.9%), and Staphylococcus saprophyticus 1(0.9%) as shown in Table II.

Table II: Organisms isolated (n=112)

Isolated bacteria	Frequency
Escherichia coli	86 (76.8)
Klebsiella	15 (13.4)
Enterobacter	4 (3.5)
Proteus	2 (1.8)
Acinetobacter	2 (1.8)
Pseudomonas	1 (0.9)
Citrobacter	1 (0.9)
S. saprophyticus	1 (0.9)
Total	112 (100.0)

Figures within parentheses indicate percentage

Table III shows the list of antibiotics used and their general sensitivity patterns of all the isolates. The pathogens showed more sensitivity to imipenem (99.1%) followed by amikacin (83.0%), nitrofurantoin (72.3%) and gentamicin (71.4%), while the sensitivity was much lower in cefuroxime (13.4%), amoxicillin-clavulanic acid (21.4%) and cefixime (22.3%).

Percentage of sensitivity to other antimicrobial agents were ceftazidime 43.8%, ceftriaxone 33.9%, ciprofloxacin 30.4%, cotrimoxazole 44.6%, doxycycline 41.0% and levofloxacin 44.7%.

Table III: General performance of the antibiotics n=112

Antibiotic	Sensitive	Intermediate	Resistant
Amikacin	93 (83.0)	12 (10.7)	7 (6.3)
Amoxicillin-clavulanic acid	24 (21.4)	7 (6.3)	81 (72.3)
Cefixime	25 (22.3)	7 (6.3)	80 (71.4)
Ceftazidime	49 (43.8)	4 (3.6)	59 (52.6)
Ceftriaxone	38 (33.9)	3 (2.7)	71 (63.4)
Cefuroxime	15 (13.4)	8 (7.1)	89 (79.5)
Ciprofloxacin	34 (30.4)	9 (8.0)	69 (61.6)
Cotrimoxazole	50 (44.6)	2 (1.9)	61 (54.5)
Doxycycline	46 (41.0)	9 (8.0)	57 (51.0)
Gentamicin	80 (71.4)	12 (10.7)	20 (17.9)
Levofloxacin	50 (44.7)	7 (6.2)	55 (49.1)
Imipenem	111 (99.1)	00.0	1 (0.9)
Nitrofurantoin	81 (72.3)	14 (12.5)	17 (15.2)

Figures within parentheses indicate percentage

Discussion

In this study, a total of 470 urine samples were processed, out of which 112 (23.8%) showed significant growth of pathogenic bacteria. The isolation rate varied from study to study in different parts of the world. In studies in Manisa, Turkey and Aligarh, India, the isolation rate was 16.4% and 10.86% respectively.[8,9] In Windhoek-Namibia 59.7% and in Western India 76.29% specimens yielded growth.[10,11] In Bangladesh, Jhora *et al.* got 55.27% significant growth of bacteria and Islam *et al*. isolated bacteria from 75.9% of hospital and 42.3% of community urine specimens.[4,12] The different isolation rates might be due to difference in population settings, and variation of doctor's selection of patient for urine culture as UTI symptoms are not a reliable indicator of infection.[6]

Among the specimens yielding significant growth in culture, females showed higher percentage of isolation (54.5%) than males (45.5%) in the present study. This was in agreement with report from India where rate of positive culture was 77.40% for female and 22.60% for male. In Namibia 65.5% female had positive culture. [10,11] One study in Nigeria was in contrary to this, where males were more affected by UTIs than females – male 52.4% and female 47.6%.[13] In the present study, in case of female samples the most prevalent age group was 21-30 years in isolation of uropathogens. This finding was similar to other studies and this could be because the age group is the most sexually active; which is one of the common risk factors attributed to UTIs.[10,14,15] In case of culture positive male samples maximum growth yielding group was 51- 60 years age group.

In this study, Escherichia coli was the most abundant (76.8%) urinary pathogen, followed by Klebsiella (13.4%), Enterobacter (3.6%), Proteus (1.8%), Pseudomonas (0.9%), Citrobacter (0.9%), Acinetobacter (1.8%) and S. saprophyticus (0.9%) (Table II). A similar trend was reported in many retrospective and prospective studies in different countries, especially the predominance of Escherichia coli. In Bangladesh, Jhora *et al*. got 82.61% and Islam *et al*. got 76.1% and 81.8% Escherichia coli in hospital acquired UTI and community acquired UTI respectively; in Turkey 73.2%, and in Iraq 60% isolates were Escherichia coli . [4,8,12,16] In contrast to the present study, isolation rate of Escherichia coli was18.9% in Namibia and 26.8% in Nigeria which is much lower, that might be due to different population sizes and the number of microbes isolated.[10,13] The reason of highest rate of isolation of Escherichia coli is that they are the normal fecal flora and uropathogenic strains of Escherichia coli have an adherence factor called P fimbriae, which mediate the attachment of Escherichia coli to uroepithelial cells.[4,17]

The isolates were tested for susceptibility against 14 antimicrobial agents (Table III). The pathogens showed high sensitivity to imipenem (99.0%) followed by amikacin (83.0%), nitrofurantoin (72.4%) and gentamicin (71.4%), while the lowest sensitivity was shown in cefuroxime (13.0%), amoxicillin-clavulanic acid (21.6%) and cefixime (22.0%). Ceftazidime (43.1%), ceftriaxone (33.9%), ciprofloxacin (30.2%), levofloxacin (44.7%), cotrimoxazole (44.8%), doxycycline (41.2%) also showed low sensitivity. In a study done by Jatileni *et al*. in Namibia, sensitivity rate was imipenem 92%, amikacin 92%, nitrofurantoin 76%, gentamicin 78%, cefuroxime 76%, amoxicillin-clavulanic acid 67%, ceftazidime 36%, ciprofloxacin 63%, cotrimoxazole 27%.[10] A study in Iraq reported sensitivity to imipenem 100%, amikacin 97%, nitrofurantoin 80%, gentamicin 47%, amoxicillin-clavulanic acid 12%, ceftriaxone 23%, ciprofloxacin 40%, levofloxacin 29% cotrimoxazole 12%.[16] In Bangladesh, Jhora *et al*. showed 95% sensitivity to imipenem, 45% to nitrofurantoin, 66% to gentamicin, 68% to cefuroxime, 56% to ceftazidime, 41% to ciprofloxacin and 18% to cotrimoxazole.[4] The association between antimicrobial drug consumption and selection of resistant bacterial strains is widely acknowledged.[18]

The present study demonstrates very high resistance to second and third generation cephalosporins (cefuroxime, cefixime and ceftriaxone) and ciprofloxacin which indicates overprescription of these groups of drugs in this area of Bangladesh.

Conclusion

It was observed that, Escherichia coli was the most prevalent pathogen of UTI, followed by Klebsiella. Uropathogens showed high sensitivity to imipenem, amikacin, nitrofurantoin and gentamicin and lowest sensitivity to second and third generation cephalosporins and ciprofloxacin.

References

1. Goddard J, Turner AN, Cumming AD, Stewart LH. Kidney and Urinary Tract Disease. In: Boon NA, Colledge NR, Walker BR, Hunter JAA, editors: Davidson's Principles and Practice of Medicine. 20th ed. London: Elsevier Churchil Livingstone; 2006. P. 455-518.
2. Khoshbakt R, Salimi A, Shirzad AH, Keshavarz H. Antibiotic susceptibility of bacterial strains isolated from urinary tract infections in Karaj, Iran. Jundishapur. J Microbiol 2013; 6(1): 86-90.
3. Hooton TM, Bradley SF, Cardenas DD, et al. Diagnosis, prevention, and treatment of catheter associated urinary tract infection in adults: 2009 International Clinical Practice Guidelines from the Infectious Diseases Society of America. Clin Infect Dis. 2010; 50(5):625-63.
4. Jhora ST, Paul S, Begum BA, Chowdhury AQ. Antibiogram of Urinary Escherichia coli isolated in Sir Salimullah Medical College & Mitford Hospital, Dhaka. Bangladesh. J Med Microbiol 2011; 05(02): 23-26.
5. Blondeau JM and Tillotson GS. Formula to help select rational antimicrobial therapy (FRAT): its application to community and hospital-acquired urinary tract infections. Int J Antimicrob Agents 1999; 12: 145-150.
6. Safar F, Mohammed YA, Reza G, Behrooz N, Ailar N. Causative agents and antimicrobial susceptibilities of urinary tract infections in the North-West of Iran. Int J Infect Dis 2008; 13(2): 140-144.
7. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. Fourteenth international supplement, approved standard M 100 SI4, NCCLS, Wayne, PA, 2004.
8. Kurutepe S, Surucuoglu, Sezgin C, Gazi H, Gulay M and Ozbakkaloglu. Increasing antimicrobial resistance in Esch. coli isolates from community-acquired urinary tract infections during 1998-2003 in Manisa, Turkey. JPN J Dis 2005; 58: 159-161.
9. Akram M, Shahid M and Khan AU. Aetiology and antibiotic resistance patterns in community-acquired urinary tract infections in J N M C Hospital Aligarh, India. Ann Clin Microbiol Antimicrob 2007; 6: 4. Published online 2007 Mar 23. doi: 10.1186/1476-0711-6-4.
10. Jatileni NJV, Maposa I, Mavenyengwa RT. A Retrospective Study of the Variability in Etiological Agents of Urinary Tract Infections among Patients in Windhoek-Namibia: Open Journal of Medical Microbiology 2015; 5: 184-192.
11. Shah LJ, Vaghela GM, Mahida H. Urinary tract infection: bacteriological profile and its antibiotic susceptibility in Western India. National Journal of Medical Research 2015; 5(1): 71-73.
12. Islam MB, Yusuf MA, Chowdhury MS, Sattar AFMA, Afrin S. Frequency and Distribution of Gram Negative Bacteria among Hospital and Community Acquired UTI Patients. Bangladesh Journal of Infectious Diseases 2014; 1(2): 24-26.
13. Nuhu T. Retrospective studies on the prevalence of uropathogens in Sokoto metropolis. Afr J Microbiol Res 2015; 9(20): 1366-1370.
14. Manikandan C and Amsath A. Prevalence and Distribution of Bacteria and Fungi Isolated from Patients with Urinary Tract infections in Pattukkottai, Tamilnadu, India. Int J Pure Appl Zool 2013; 1(3): 213-222.
15. Olandeinde, B.H., Omoregie, R., Olley, M, Anunibe, J.A. (2011) Urinary Tract Infection in a Rural Community of Nigeria. North American Journal of Medical Sciences, 3, 75-77.
16. Hadi AM, Sheri FH, Jaccob AA. Urinary Tract Infection Prevalence and Antibiotic Resistance A Retrospective Study in Basra Governorate, Iraq. AJPS 2014; 14(2): 129-135.
17. Johnson JR. Virulence factors in Escherichia coli urinary tract infection. Clin Microbiol Rev 1991; 4(1): 80-128.
18. Saleh AA, Ahmed SS, Ahmed M, Sattar ANI, Miah MRA. Changing trends in Uropathogens and their Antimicrobial sensitivity pattern. Bangladesh J Med Microbiol 2009; 03 (01): 9-12.

Lung Function Test by Spirometry of Patients with Diabetes Mellitus

Akramuzzaman M [1], Miah SM [2], Uddin R[3], Moniruzzaman M [4], Rahman N [5], Islam MS [6], Rahman MA[7]

Abstract

With an increasing incidence world wide, Diabetes Mellitus will be a leading cause of morbidity and mortality for the foreseeable future. So it is utmost important to define every effects of diabetes on every body system where it can affects adversely. But till now the effects of Diabetes Mellitus on pulmonary system are not well documented. So this study was intended to find out the effects of diabetes mellitus on pulmonary function in our study population. Our study was an analytical cross sectional comparative study. Total 88 diabetic patients (Type -1 14 and Type -2 74) and equal number of non -diabetic healthy people were included. For assessment of lung function, FVC, FEV_1 and FEV_1 /FVC% of all the subjects were measured by electronic desktop spirometer. Results were compared by independent student 't' test. The mean FVC and FEV_1 values were significantly low and FEV_1/FVC% was within normal limit (>80%) in both type of diabetics patients compared to controls. Out of total 74 type -2 diabetic subjects 58 patients (78%) had FVC% predicted <80% i.e. restrictive lung function and 16 patients (22%) had normal finding (FVC% predicted >80%). In type 1 diabetic patients all had restrictive type spirometry. Considering the spirometric results it is obvious that diabetes mellitus adversely affects the pulmonary functions and that is predominantly restrictive pattern.

Key Words: Diabetes Mellitus, Lung Function Test, Spirometry.

KuMCJ, June 2017; Vol. 1 (1): 17-21

Introduction

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia caused by absolute or relative deficiency of insulin[1]. It is estimated that, the total number of people worldwide with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030[2].

Authors' Affiliations:

1. Dr. Md. Akramuzzaman. MD (Internal Medicine), Assistant Professor, Medicine, Kushtia Medical College, Kushtia.
2. Dr. Saleque Masud Miah. MD (Int. Medicine), DTCD, Associate Professor (cc), Medicine, Kushtia Medical College, Kushtia.
3. Dr. Refaz Uddin. MD (Cardiology), Junior Consultant, Cardiology, 250 Beded General Hospital, Kushtia.
4. Dr. Md. Moniruzzaman. MD (Radiology & Imaging), Junior Consultant, Radiology & Imaging, 250 Beded General Hospital, Kushtia.
5. Dr. Nazmin Rahman. M.Phil (Microbiology), Assistant Professor, Microbiology, Kushtia Medical College, Kushtia.
6. Dr. Md. Shohidul Islam. MD (Neuro-Medicine), Assistant Professor, Neuro-Medicine, Kushtia Medical College, Kushtia.
7. Dr. Md. Ashadur Rahman. MD (Neuro-Medicine), Lecturer, Community Medicine, Kushtia Medical College, Kushtia.

Address of Correspondence: Dr. Md. Akramuzzaman. Assistant Professor, Medicine, Kushtia Medical College, Kushtia. Mobile No: 01716078725, E.mail: dr.akramuzzaman@gmail.com.

Greater longevity, obesity, unsatisfactory diet, sedentary life style and increasing urbanization are main factors for this[3]. Diabetes is a multisystem disorder accompanied by widespread biochemical, morphological and functional abnormalities which mayprecipitate certain complications that affect the renal, cardiovascular, neural systems and also organs and tissues like skin, liver, collagen and elastic fibers[4]. In spite of having almost same structural components of the lungs with other organs where diabetes affects adversely, the long term effects of diabetes mellitus on this system are not well documented. Dyspnea on exertion in a diabetic patient traditionally arouses suspicion of cardiac failure secondary to ischaemic heart diseases or hypertension. In the recent past a significant number of studies were conducted worldwide, though not so abundant in our country to see whether diabetes itself have any detrimental effect on pulmonary function rendering the patient dyspnoeic. The improvement in lung function following intensive insulin therapy supports the concept that the lung may be a target organ for damage in diabetes[5]. The suggested basic pathophysiology behind this notion are - firstly, chronic hyperglycemia

induced activation of reactive oxygen species and their damaging effects on pulmonary tissue [1,3,4,6]. Secondly, non enzymatic glycosylation of chest wall and bronchial tree matrix protein (i.e. collagen and elastin) leading to accumulation of advanced glycation end product (AGE) progressively reduces lung compliance [1,4,7] and finally, binding of plasma protein mainly albumin to the glycosylated basement membrane accounting in part for the thickening of the pulmonary basal lamina leading to impairment of pulmonary diffusion capacity[8,9,10]. The normal lung mechanics and gas exchange are influenced by the integrity of the pulmonary connective tissue and microvasculature. So, diabetes mellitus causes abnormalities in both of these structural components leading to development of abnormalities in the pulmonary function[11]. As the pulmonary reserves are larger, the symptoms and disability from diabetes develops later in lungs than other organs. Except a few [12,13], most of the study results come to a conclusion that both [14,15] type 1[9,16] and type 2[3,17-22] diabetes mellitus patients have impaired lung function evidenced by statistically significant reduction of the forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1) and normal FEV_1/FVC % suggesting restrictive pattern of lung function.
This study was intended to find out the effects of diabetes mellitus on pulmonary function in our study population.

Materials and methods

An analytical cross sectional comparative study was carried out among diabetic patients admitted in department of medicine of Rajshahi Medical College Hospital and Rajshahi Diabetics Association General Hospital, Rajshahi during the period of January 2012 to December 2013.

By purposive sampling method total 88 number of previously diagnosed diabetes mellitus patients for at least five (5) years having no evidence of any cardio respiratory disease, non smoker and non obese person having BMI < 25kg/ m^2 and equal number of non diabetic healthy persons were included in this study. Data collection from the respondents were conducted according to structured questionnaire and pulmonary function (FVC, FEV_1, FEV_1/ FVC %) were assessed by electronic desktop spirometer by the investigators themselves. Data were analyzed using the SPSS statistical software version 16.0. Association between variables conducted by applying student t test.

Results

This study included 88 diabetic patients, 74 type 2 and 14 type 1 and 88 comparison subject. Type 2 diabetes mellitus included 33 males and 41 females and type 1 diabetic patients included 7 males and 7 females. Equal numbers of male and female subjects were included in comparison group.

Age and anthropometric parameters of subject of type 1 diabetes mellitus and comparison groups in case of male and female are shown in table I. Subjects were closely comparable ($p > 0.05$) in their age, body weight, height and BMI distribution within groups i.e. male diabetics against male comparison and female diabetics against female comparison.

Table II shows age and anthropometric parameters of type 2 diabetes mellitus and comparison group in male and female, where subjects were also closely comparable ($P>0.05$) in their age, body weight, height and BMI distribution within groups except body weight of male subjects ($P<0.05$).

Table-I: Age and anthropometric parameters of type-1 diabetics and comparison groups.

Age and anthropometric parameters	Sex	Diabetic (n=7) Mean (SD)	Non-Diabetic (n=7) Mean (SD)
Age (years)	Male	19.71 (3.45)	17.85 (4.41)
	Female	21.71 (4.27)	20.28 (5.34)
Body Wt. (kg)	Male	54.42 (10.40)	46.00 (7.30)
	Female	41.85 (8.59)	40.14 (8.07)
Height (cm)	Male	163.86 (8.25)	156.14 (5.30)
	Female	152.29 (6.18)	151.71(5.56)
BMI (kg/m^2)	Male	20.07 (1.99)	18.75 (1.89)
	Female	17.87 (2.30)	17.31 (2.25)

Table-II: Age and anthropometric parameters of type-2 diabetics and comparison group.

Age and anthropometric parameters	Sex	Diabetic (n=33) Mean (SD)	Non-Diabetic (n=33) Mean (SD)
Age (years)	Male	58.10 (10.57)	54.01 (9.21)
	Female	52.02 (8.63)	49.78 (7.05)
Body Wt. (kg)	Male	60.89 (10.03)	65.12 (6.24)
	Female	55.21 (9.09)	52.75 (7.82)
Height (cm)	Male	165.91 (6.01)	165.70 (4.96)
	Female	154.15 (4.52)	152.29 (5.66)
BMI (kg/m^2)	Male	22.29 (3.44)	23.94 (2.05)
	Female	23.12 (3.66)	22.67 (3.10)

Table-III shows spirometric results of type 2 diabetics and comparison groups in male and female sex which revealed there was significant decrease in FVC measured, FVC% predicted, FEV_1 measured and FEV_1% predicted values ($p<0.005$) compared to non-diabetic subjects. Significant changes also occurred in FEV_1/FVC% value ($p<0.005$).

Statistically significant reduction was also observed in all parameters of mean spirometric values in type 1 diabetes mellitus in both sexes compared to comparison group ($p<0.05$) except FEV_1 measured value in male subjects ($P>0.05$). [Table IV]

Out of total 74 type-2 diabetic subjects 58 patients (78%) had FVC% predicted <80% i.e. restrictive

Table-III: Spirometric results of type-2 diabetes mellitus and comparison groups in male & female.

Spirometric results	Sex	Diabetic (n=33) Mean (SD)	Non-Diabetic (n=33) Mean (SD)	P value
FVC measured value (Litres)	Male	2.53 (0.57)	3.37 (0.55)	0.002
	Female	1.77 (0.35)	2.20 (0.37)	0.003
FVC% predicted	Male	69.30 (11.27)	89.00 (6.77)	0.001
	Female	68.78 (11.41)	84.87 (3.18)	0.001
FEV_1 measured value (Litres)	Male	2.34 (0.50)	2.82 (0.51)	0.003
	Female	1.65 (0.33)	1.91 (0.30)	0.004
FEV_1% predicted	Male	81.03 (11.54)	91.70 (9.50)	0.001
	Female	75.90 (13.96)	86.70 (5.67)	0.001
FEV_1/FVC %	Male	93.65 (6.42)	83.57 (5.21)	0.001
	Female	93.09 (7.15)	86.88 (4.67)	0.002

Table-IV: Spirometric results of type-1 diabetes mellitus and comparison groups in case of male and female.

Spirometric results	Sex	Diabetic (n=7) Mean (SD)	Non-Diabetic (n=7) Mean (SD)	P value
FVC measured (Litres)	Male	2.48 (0.51)	3.27 (0.50)	0.013
	Female	2.00 (0.56)	2.87 (0.29)	0.004
FVC% predicted	Male	58.14 (10.38)	89.57 (3.50)	0.001
	Female	62.71 (13.18)	93.71 (3.45)	0.000
FEV_1 measured value (Litres)	Male	2.25 (0.47)	2.68 (0.51)	0.127
	Female	1.80 (0.49)	2.41 (0.24)	0.013
FEV_1% predicted	Male	61.00 (10.89)	84.85 (5.42)	0.001
	Female	64.57 (12.17)	89.57 (3.59)	0.001
FEV_1/FVC %	Male	90.95 (8.36)	81.60 (5.13)	0.027
	Female	90.80 (7.55)	84.77 (4.17)	0.049

lung function and 16 patients (22%) had normal finding (FVC% predicted >80%). All type-1 diabetic subjects had restrictive pattern of lung function (Table-V).

Table-V: Number of subjects according to FVC% predicted value of type-1 and type-2 DM.

FVC % predicted	Number of patients	
	Type-1 DM N (%)	Type-2 DM N (%)
FVC % predicted >80%	00 (00%)	16 (22.0%)
FVC % predicted <80%	14 (100%)	58 (78.0%)
Total	14 (100%)	74(100%)

Discussion

In this study out of total 88 diabetic subjects, 74 type 2 and 14 type 1, 40 patients(45.54%) were male and 48(54.54%) were female. Equal numbers of male and female subjects were taken in comparison group. The possible cause for this female preponderance was the fact that many males were excluded on account of their smoking history.

The groups were almost homogenous in respects of age, sex, height, weight, BMI, absence of known cardio-respiratory diseases and all being non-smokers except body weight of type 2 male diabetics and comparison group ($p<0.05$). To overcome this variation spirometric values were also assessed as percentage of predicted. So it is clear that in comparing the spirometric values presence or absence of diabetes mellitus is the main factor.

Patients with duration of diabetes <5 years were excluded as most of the long term complication usually occurs after 5 years of illness[18].

This study revealed that mean FVC measured value was significantly reduced ($p<0.05$) than comparison group in both type 1 and type 2 diabetic patients and it was much less than 80% of its predicted value, which indicate restrictive pattern of lung function. Similar finding were also stated by Dhaher J.S Al Habbo et al[14], Sanjeev Verma *et al*[15], Khan Mohammad Arif *et al*[16] , Sultan A. Meo et al[20], Shravya Keerthi G *et al*[21], Ali M.O *et al*[24]. FVC is the maximal volume of air that can be exhaled from full inhalation by exhaling as forcefully and rapidly as possible. In diabetes mellitus there is increase cross-linkage formation between polypeptide of collagen in pulmonary connective tissue which decreases FVC in all diabetic patients and hence responsible for restrictive respiratory defects[3].

In the current study the mean FEV_1 value was reduced in both type of diabetes in both sexes in compare to comparison group and FEV_1/FVC% was much higher which signified no obstructive features were found. These findings were consistent with findings of Sanjeev Verma *et al*[15] and Nakajima *et al*[25]. Against this observation Kanya Kumari DH *et al*[17] found 14% patients and Mahadeva Murthy[22] got 28% of men and 8.2% of women had obstructive pulmonary function. When considering FEV_1/FVC% it was with in normal limit in both type of diabetes and comparison groups i.e. ≥80% and it was more in diabetic subjects than comparison groups. Similar finding was also mentioned by Dennis *et al*[18], Sultan A. Meo *et al*[20], and Mahadeva Murthy[22].

Though the mean FVC% predicted of all diabetic subjects was <80% (i.e. restrictive pattern), but when this parameter considered in individual basis, 78% of type 2 diabetic patients had restrictive lung function and 22% had normal study. A same finding was also observed by Kanya Kumari DH *et al*[17] and Mahadeva Murthy[22] in their study. In case of type 1 diabetes, all patients had restrictive lung defects. Very small number of case may be the reason behind this finding. In contrary to our observations Pinar Celik *et al* in the study " Pulmonary function parameters in patients with diabetes mellitus "[12] did not found any possible association between diabetic micro vascular pathology and pulmonary functional changes and this was thought to be due to the insufficient number of patients. Benbassat *et al*[13] showed that FVC and FEV_1 were within the predicted values in both type 1 and type 2 populations. The most probable reason for the contradiction was that they studied pulmonary function among a group of diabetic patients by considering their predicted values but they did not compare their results with the matched comparison group.

Conclusion

Considering the spirometric results in this study, it was obvious that diabetes mellitus adversely affects the pulmonary functions and that was predominantly restrictive pattern.

References

1. B.M. Frier , M.Fisher. Diabetes mellitus. In: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston, editors. Davidson's Principal & Practice of Medicine. 21st Edition. Churchill Livingstone: 2010: p.798-826.

2. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes : estimates for the year 2000 and projections for 2030. Diabetes care, 2004 May; 27(5): 1047-1053.
3. Shravya keerthi G, Hari Krishna B, Suresh M, Preetham J K, Mallikarjuna Reddy N, Sharan B Singh M. Role of Duration of Diabetes on Ventilatory Capacities and Expiratory Flow Rate in Type 2 Diabetes Mellitus, J Biol Agri Health, 2012: 2(6).
4. Michael Brownlee L P, Aiello M E, Cooper A I, Vinik R W, Nesto A. J. M. Boulton. Complications Of Diabetes Mellitus. In: Henry M. Korenberg, Shlomo Melmed, Kenneth S. Polonsky, P. Reed Larsen, editors. Williams textbook of endocrinology. 11th Edition. Elsevier India Publisher, 2008.
5. Niranjan V, McBrayer DG, Ramirez LC, Raskin P, Hsia CC. Glycemic control and cardiopulmonary function in patients with insulin-dependent diabetes, Am J Med, 1997; 103:504–513.
6. Niedowicz DM, Daleka DL. The role of oxidative stress in diabetic complication. Cell Biochem Biophys. 2005 ; 43(2) : 289-330.
7. Kumar Sampath M.N. Changes in lung function tests in type-2 Diabetes Mellitus[Internet]: [place unknown]: [publisher unknown]: 2011 Oct 16 [cited 2012 Aug 9]. Available from: http://www.scribd.com/doc/.../Thesis-Topic-List
8. Vracko R, Thorning D, Huang TW. Basal lamina of alveolar epithelial and capillaries, Quantitative changes with aging and diabetes mellitus, *Am Rev Respir Dis.* 1979; *120* : 973-83.
9. Sandler M, Bunn A.E and Stewart R.I. Pulmonary function in young insulin-dependent diabetic subjects, *Chest*. 1986; 90: 670 – 675.
10. Weynand B, Jonckheere A, Frans A, Rahier J. Diabetes mellitus induces a thickening of the pulmonary basal lamina. Respiration. 1999; 66(1): 14-9.
11. Sandler M, Stewart R I, Gemperli B M, Hanekom C, Kuhn S H. Is the lung a 'target organ' in diabetes mellitus? *Arch int Med*. 1990 July: 150: 1385 – 1388.
12. Celik P, Ozmen B, Yorgancioglu A, Ozmen D, Cok G. Pulmonary function parameters in patients with Diabetes Mellitus. Turkish journal of endocrinology and metabolism. 1999: 1: 5-10.
13. Benbassat CA, Stern E, Kramer M. Pulmonary function in patients with diabetes mellitus. Am J Med Sci 2001: 322(3): 127-132.
14. Dhaher J.S, Al-Habbo, Afraa M, Al-Ameen. Diabetes mellitus and lung function tests. Ann. Coll. Med. Mosul 2012: 38(1): 27-32.
15. Sanjeev V, Mumtaz G, Rattan P K. Assessment of Pulmonary Functions in Patients with Diabetes Mellitus. JK SCIENCE . 2009 April-June: 11(2).
16. Khan Mohammed Arif, Jahan N, Sultana N, Akter R. FVC, FEV_1 and FEV_1/FVC % in Type-1 Diabetic Male and their relationship with HBA_1c. J Bangladesh Soc Physiol. 2012 June: 7(1): 23-28.
17. Kanyakumari D H, Natraj S M, Devaraj H S. Correlation of duration of diabetes and pulmonary function tests in type 2 diabetes mellitus patients. Int J Biol Med Res. 2011; 2(4): 1168-1170.
18. Rodolfo J Dennis, Dario Maldonado, Maria X Rojas, et al. Inadequate glucose control in type 2 diabetes is associated with impaired lung function and systemic inflammation: a cross-sectional study. BMC Pulmonary Medicine. 2010: 10:38.
19. Davis WA, Knuiman M, Kendall P, Grange V, Davis TM. Glycemic exposure is associated with reduced pulmonary function in type 2 diabetes; The Fremantle diabetes study. *Diabetes Care.* 2004 March: 27(3): 752 - 757.
20. Sultan A. Meo, Abdul Majeed AL-Drees, Muhammad Arif, Khalid Al-Rubean. Lung function in type 2 saudi diabetic patients, Saudi Med J, 2006; 27(3): 338-343.
21. Shravya keerthi G, Hari Krishna B, Suresh M, Preetham J K, Mallikarjuna Reddy N, Sharan B Singh M. Deterioration of Pulmonary Functions in Type 2 Diabetes Mellitus. IOSRJPBS. 2012 May-June; 1(1): 39-43.
22. Mahadeva Murthy. Changes in lung function tests in type-2 Diabetes Mellitus. Int. J. Basi. Med. Sci. 2012 June: 3(2).
23. Anonymous; The diabetes control and complication trial research group, The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Eng J Med, 1993; 329: 977-986.
24. Ali MO, Begum S, Ali T and Ferdousi S. FVC, FEV_1 and FEV_1/FVC% in Type 2 Diabetes and Their Relationships with Duration of the Disease. J. Bangladesh Soc Physiol. 2009 Dec; 4(2): 81-87.
25. Nakagima K, Kubouchi Y, Muneyuki T, et al. A Possible association between suspected restrictive pattern as assessed by ordinary pulmonary function test and the metabolic syndrome, Chest, 2008; 134(4): 712-18.

ORIGINAL ARTICLE

A Study on Diarrhoea in Under Five Children at a District Level Hospital in Bangladesh

Ali MA[1], Uddin SMN[2], Rahman N[3], Munir MS[4]

Abstract

Diarrhoeal diseases remain an important cases of mortality and morbidity among children particularly in low and middle income countries. Passage of three or more watery stools in 24 hours is defined as diarrhea Acute watery diarrhea lasts less than 14 days. Failure to manage properly diarrhea leads to severe dehydration, electrolyte imbalance and septicemia. Patient may die also. Aim of this study was to find out the most common ages in under five children with diarrhoeal diseases.

This descriptive cross sectional study was conducted at diarrhea ward of district hospital, Kushtia, during the period of June to December 2016. A total 864 cases were studied irrespective of age, sex along with clinical diagnosis. Among the study cases, common age group was 06 to 11 months (44.21%), next common age group was 12-24 months (28.47 %). It occurs more in rural children than urban children.

Diarrhoea occurs more in children aged 6 to 24 months of under 05 children. Children live in rural areas suffer more than those of urban areas.

Key words: Age, Diarrhoea, Rural,

KuMCJ, June 2017; Vol. 1 (1): 22-25

Introduction

WHO defined diarrhoea as the frequent passage of loose stool[1]. Passage of three or more loose or watery stool daily is defined as diarrhoea[2].Diarrhoea is commonly caused by viruses, bacteria, parasite and others[3].It is not unusual to find mucus and blood in stool[4]. Vomiting and low grade fever may be present. Other signs include abdominal cramps, dehydration, weight loss, perianal erythema and malnutrition. Transmission of diarrhoeal agents is usually through feco-oral route resulting from intake of contaminated food or water. Some predisposing factors are unhygienic environment, poor nutritional status and inadequate breast feeding.

Acute diarrhoea is one of the principal causes of morbidity and mortality among children in low income countries[5]. Diarrhoea is the second common cause of death among under-five children[6-8]. Under-five mortality is 46 per thousands live births. Ten percent of under-five children are known to have diarrhoea at any point of time. Due to lack of proper management of diarrhoea leads to severe dehydration, electrolyte imbalance and septicemia[9]. As a result patient may die also. Use of ORS, exclusive breast feeding, intravenously used of cholera saline, zinc therapy saves life of under five childrens[10-12].The aim of the study was to assess the incidence of diarrhoea and most common ages among the under-five children.

Authors' Affiliations:

1. Dr. Md Ayub Ali. DCH, Associate Professor, Pediatrics, Kushtia Medical College, Kushtia,
2. Dr. SM Nazim Uddin, DCH, Junior Consultant, Pediatrics, 250 bedded General Hospital, Kushtia.
3. Dr. Nazmin Rahman, M.Phil (Microbiology), Assistant Professor, Microbiology, Kushtia Medical College, Kushtia.
4. Dr. Md. Sirajum Munir, MD (Cardiology), Assistant Professor, Cardiology, Kushtia Medical College, Kushtia.

Address of Correspondence: Dr. Md Ayub Ali. Associate Professor, Pediatrics, Kushtia Medical College, Kushtia, Mobile: 01715081283, Email: mdayuba35@gmail.com

Materials and Methods

This descriptive type of cross sectional study was carried out during the period of June to December 2016 at diarrhoea ward of district hospital, Kushtia, Bangladesh. During the study period, 897 sick children were admitted into the diarrhoea ward of District Hospital, Kushtia, Bangladesh. Out of 897, 864 under-five diarrhoea cases were enrolled in this study. At enrolment a detailed case history was taken and through physical examination was performed and recorded on standard case record forms. Thirty three children were excluded from this study because of more than five years of age,

co-morbidities and complications. Data were analyzed manually and by using computer. Informed written consent was obtained from parents or primary caregivers of the children before enrolment.

Results

This descriptive type of cross sectional study was carried out during the period of Jun 2016 to Dec 2016. In this study, 864 diarrhoea children were enrolled during the study period. Out of them, 458 (53%) were male children and 406 (47%) were female children.(Table I).

Out of 864 diarrhoea children, 708 (81.94%) were rural children and 156 (18.06%) were urban children (Table II). Monthly admission of the children into the diarrhoea ward shown in the bar diagram (Figure-1). It showed that highest admission in the month of December (24.07%) and next highest in the month of November (22.91%).

Table I
Sex distribution of children.

Sex	Frequency	Percentage
Male	458	53.0
Female	406	47.0
Total	864	100.0

Table II
Residence of the children.

Residence	Frequency	Percentage
Rural	708	81.94
Urban	156	18.06
Total	864	100.0

Out of 864 sick children, 12 (1.38%) were below 1 month of age, 88 (10.18d%) were 2-5 months of age, 382 (44.21%) were 6-11 months of ages, 246 (28.47%) were 12-24 months of ages, 88 (10.18%) were 24-35 months of ages, 34 (3.93%) were 36-47 months of age and 14 (1.65%) were 48-59 months of ages. (Table IV)

Table III
Feeding practices of children.

Feeding	Frequency	Percentage
Breast Feeding (Exclusive)	64	7.40
Mixed Feedings	800	92.60
Total	864	100.0

Table-IV: Age of children

Age of Child (Months)	Frequency	Percentage
<1	12	1.38
2-5	88	10.18
6-11	382	44.21
12-23	246	28.47
24-35	88	10.18
36-47	34	3.93
48-59	14	1.65
Total	864	100.0

Monthly admission of the children into the diarrhoea ward shown in the bar diagram (Figure 1).It showed that highest admission in the month of December (24.07%) and next highest in the month of November (22.91%).

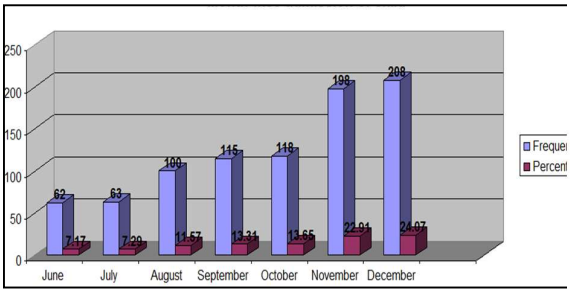


Figure-1: Simple bar diagram showing monthly wise admission of children.

Discussion

In this descriptive type of cross sectional study, 864 children with diarrhoea were studied during the months of June to December 2016. Among 864 study cases, 53.0% were male children and 47.0% were female children. Male children suffer more than female children. This finding is consistent with report of Ugum lesi el at,[13] Obu et at,[14], Ohaeri & Odre kaesieme,[15,] Ansari[16] and in children under 5 years of age in kathmundu[17] .

A biological explanation may be related to the fact that boys during infancy have to build a larger muscles mass than girls, consequently,

risk of diarrhoea and place the boys as boys might have increased demand for micronutrients and are therefore more at risk of negative balance, including vitamin A and Zinca[18]. This vulnerability might increase the the weaker sex regarding infections. Among the older children, because boys are more active than girls, boys tends to move more around and touch objects in the surrounding ground, where as girls might tend to stay close to their mothers and play with more hygienic toys. This study also shows that 81.94% diarrhoea children came from rural area and 18.06% from urban. Rural children suffer more than urban children in diarrhoea. Yassin stated that in rural areas, due to low income, inadequate water source and unhygienic environment, people suffer more to infections diseases.[19]

This study showed that 7.40% children got exclusive breast feeding and 91.60% got mixed feeding. Diarrhoea is common in mixed feeding children. Complete or exclusive breast feeding of a baby gives adequate protection against various gastrointestinal diseases[20].

This study also showed that 44.21% of sick children were aged 6-11 months and 28.47% of children were aged 12 to 24 months. According to Thielman and Guerrant[21] the rates of diarrhoea were highest among children of 6-11 months of age, remained at high level among 1-year-old children and decreased when children got older. This study is consistent with the study of Thielman and Guerrant[21]. A decrease in number of cases among older children might be due to fact that immune system of older children get stranger in resistance against agents of diarrhoea as can be seen in work of Gascan et al,[22] This findings are similar to the result from a study of Niriema LW et al in Barkina Faso[23], children under the age of 12 months had the highest rate diarrhoea (44%).

This study also showed that incidence of diarrhoea is more in November (22.91%) and December (24.07%). Diarrhoea occurs more in winter season.This is similar with the study of Azad A et al near Tehran, Iran[24]. Therefore under 5 diarrhoea has got seasonal variation.

Conclusion

Diarrhoea occurs more in the age of 6 months to 24 months of under five children. Children live in rural area suffer more than those of urban area.

Reference

1. Diarrhoeal disease Fact sheet N^{0}330" World Health Organization April 2013. Archived from the original on 17 July 2014. Retrieved 9 July 2014.
2. Catherine AC, Zahra A. Gastroenteritis in children: Part-II. Prevention and Management. Afr Med Fam Physician. 2012; 85:1066-70.
3. Navaneethan U, Giannella RA. "Mechanism of infectious diarrhoea" Nature Clinical practice Gastro enterology and hepatology, November 2008; 5(11): 637-47. Doi:10.1038/NCPG asthep 1264 PMID 18813221.
4. Nancy Roper. Churchill Living stone pocket Medical Dictionary. 14th edition, London, Madrid, Melbourne and New York; 995. P-23.
5. UNICEF. Commiting to child Survival, A promise Renewed Progress Reports 2015. New York, NY: UNICEF 2015.
6. Murray C, Lopez A. Alternative projection of mortality and disability by cause 1990-2020. Global burden of disease study. Lancet 1997; 349: 1498-504 Google Scholar.
7. Jones G, Sketetee RW, Black RE, Bhutta ZA, Morris SS and the Bellagio child survival study group. How many child deaths can we prevent this year? Lancet 2003; 362: 65-71 Google scholar.
8. World Health Organization. Integrated management of childhood illness (IMCI). World Health Organization 2012. P 17-8.
9. Korie FC, Ikefuna AN, Ibe BC. Bacterial agents associated with acute diarrhoea in under 5 children in Enugu Nigeria. J Dent Med Sci 2012; 2: 40-5.
10. United Nations Children's Fund (UNICEF). State of the world's children 2005: children under Treat New York: UNICEF House, 2005. Google scholar.
11. WHO/UNICEF Joint statement. Clinical Management of Acute diarrhoea, Geneva: World Health Organization, 2004, P-8. Google Scholar.
12. Singh J, Gowriswari D, Chavan BR, Patiat RA, Debnath AC, Jain DC, et al. Effect on maternal literacy on nutritional status of children under the age of 5 years in the Babban Dodo community Zaria city, North West Nigeria. Ann Niger Med 2012; 6: 61-4.
13. Ogunlesi, T, Okeniyi, J. Oseni, S. Oyelami O, Nijokanma, F & Dedeke, O. Parasitic etology of child hood diarrhoea, Indian Journal of Pediatric, 2006; 73, 1081-1048.

14. Obu, O. Aguomadu, N. Uneke, C.J & Amadi, E.S. Aetiology of acute infantile diarrhoea in south Estern Nigeria. An assessment of microbiological and antibiotics sensitivity profile the intermit journal of Third world medicine, 2008; 7(1): 2.
15. Ohaeri, C.C. & Odrekasieme, O.P. Survey of geohelminthiasis among primary schoold children in Umuahia metropolis of Abia state, Nigeria. International Journal of Environment at health and Human development 2011; 11(11): 41-45.
16. Anasari, S. Bahadur S, Parajuli, K and Paudyal, R.P.A. Pattern of acute parasitic diarrhoea in children under 5 years of age in Kathmandu, Nepal. Open journal of Medical Microbiology, 2012; 2: 95-100.
17. Ishiyama, S. Ono, K. and Rai, S.K. Study of enteropathogen and its predisposing features in suburban public schoold children in Kathmandu, Nepal, Nepal Medical College Journal, 2001; 3: 5-9.
18. Jinadu, M.K. Olusi, S.O. Agun J.I & Fabiyi, A.K. Child hood diarrhoea in rural Nigeria studies on prevalence, mortality and socio-environmental features. Journal of Diarrhoeal disease Research, 1991; 9 (4), 323-327.
19. Yassin, K. Morbidity and risk factors of diarrhoeal diseases among under-fine children in rural upper Egypt journal of Tropical Pediatrics, 2000; 46 (5) 282-287.
20. Khan MR, Rahman ME. Essence of Pediatrics, 4th ed. New Delhi: Elsevier; 2011. Chapter 3, Infant and Young child feeding; P. 49-55.
21. Thielman, N. M. & Gurrant, R, L. Acute infectious diarrhoea. The England Journal of medicine, 2004; 350 (1) 38-47.
22. Gascon, J Vargas, M. Schllenberg, D. Urassa, H. Casals, C. kahigva, E Aponte J, T. Mshinda H, & Villa, T. Diarrhoea in children under 5 years of age from Ifakara, Tanzania, Journal of Clinical Microbiology, 2000; 38 (12), 4459-4462..
23. Niriema LW, Nordegrens J, Ouermi D, Dianou D, Traor AS, Svenson L et al. Burden of rotavirus and other enteropathogens among children with diarrhoea in Barkino faso Int. Infect disease 2011; 15(9) ; e 646-52.
24. Azad A, Nourijah N, Shahbazi F. Relationship of intestinal parasite among school children near Tehran,Iran, Southest Asian J Trop Med Public health 2004; 35: 116-9.

Pterygium Excision: Outcome of Conjunctival Autograft Versus Adjunctive Mitomycin-C

Islam ME[1], Rahman KA[2], Ahamed AU[3]

Abstract

Pterygium, an external eye disease, is common worldwide but is particularly prevalent in tropical and subtropical areas. Despite the chance of recurrence– surgical removal remains the treatment of choice. Two methods of treatment, one is pterygium excision and another is excision of pterygium with Mitomycin-C as an adjuvant therapy in bare sclera technique were used & evaluated in this study. The aim of this study is to assess the recurrence of pterygium & other complications of pterygium excisions between two methods. In this prospective, interventional study hundred patients with primary pterygium were treated randomly in two groups - in group A (n=50) with excision of pterygium followed by conjunctival auto graft and in group B (n=50) with Mitomycin-c (0.02%) as an adjuvant therapy after bare sclera technique. These patients were followed up on 1st, 2nd,6th ,12th and 24th weeks after operation and evaluated for recurrence of their pterygium, and other post-operative complications. After 6 months follow up in Group A- with conjunctival auto-graft, recurrence was 2 (4%), in group B- with Mitomycin-C recurrence was 4 (8%) where P value is 0.10 which is statistically not significant. In Group A - other postoperative complications were Superficial Punctate Keratitis 0, ulceration on the bed of pterygium 0(0%) patients, delayed wound healing 3(6%), granuloma 6(12%), giant papilla 1(2%). In Group B - Superficial Punctate Keratitis. 3(6%), ulceration on the bed of pterygium 1(2%), delayed wound healing 7(14%), granuloma 2(4%), giant papilla 0 were noticed. It is concluded that both techniques are effective methods to reduce the recurrence rate after pterygium surgery. But recurrence rate and complications were higher in Mitomycin-C as an adjuvant therapy after bare sclera technique.

Key words: Pterygium, Mitomycin-c, Superficial Punctate Keratitis.

KuMCJ, June 2017; Vol. 1 (1): 26-30

Introduction

Pterygium is a common ocular surface disorder of sub-conjunctival tissue characterized by fibro- vascular growth encroaching upon cornea. Pterygia usually develop in patients residing in hot climates. There is also higher incidence of pterygia in people chronically exposed to sunlight and wind such as farmers and fishermen. Pterygia occur in palpebral fissure generally nasally, may occasionally temporally or bilaterally[1,2].

Authors' Affiliations:

1. Dr. Md. Emranul Islam, F.C.P.S. (Ophthalmology), Junior consultant, Ophthalmology, 250 Bedded General Hospital, Kushtia.
2. Dr. Kazi Anisur Rahman, MS (Ophthalmology), Junior consultant, Ophthalmology, 250 Bedded General Hospital, Kushtia.
3. Dr. Arman Uddin Ahmed, FCPS , MS , Assistant professor, Ophthalmology, Kushtia Medical College, Kushtia

Address of Correspondence: Dr. Md. Emranul Islam, Junior consultant, Ophthalmology, 250 Bedded General Hospital, Kushtia, Mobile: 01712-297450, Email: emran2590@gmail.com

A pterygium may be stationary or progressive which can obscure visual axis and can cause severe visual problem. Indication for surgical excision ofPterygium includes chronic inflammation not responding to medical treatment or causing visual disturbance due to induced astigmatism by invading cornea ocular motility restriction and cosmetic reason[3]. Despite the chance of recurrence – surgical removal remains the treatment of choice[4]. The principal goal of pterygium management is prevention of recurrence or to lower recurrence rate, absence of complication and satisfactory cosmesis[5]. Numerous surgical procedures have been recommended for pterygium excision including bare sclera technique, bare sclera excision with Mitomycin-c, β radiation, conjunctival graft, amniotic membrane graft, rotational auto graft etc[5]. Most pterygium recurrences occur in the early post operative period, more than 90% occur within the first year surgery[5]. Pterygium morphology has been shown to be a significant risk factor for recurrences; young individuals have a higher

rate of recurrences5. Simple excision carries a high recurrence rate ranging from 24%-89%2. The addition of Mitomycin-C (0.02%) of various concentrations has been reported to be effective in preventing recurrence6-8.

However Mitomycin-C may result in devastating complications such as scleral necrosis and microbial infections in high concentration (0.4mg/ml-1mg/ml)9-11. Another alternate adjunct is conjunctival auto graft. The limbal epithelium acts as a junctional barrier to conjunctival overgrowth and pterygium is considered to represent a local limbal deficiency12-14. In this study a comparison of efficacy in preventing recurrence of pterygium is shown between excision of pterygium followed by a transplantation of a conjunctival autograft and using Mitomycin-C as an adjuvant therapy after bare sclera excision.

Material and methods

Hundred patients with primary pterygium were treated randomly in two groups - in group A (n=50) with excision of pterygium followed by conjunctival autograft and in group B (n=50) with Mitomycin-C (0.02%) as an adjuvant therapy after bare sclera technique. After proper consent of the patients all information recorded in a data base chart. Parameter of the study were visual acuity both pre & post operatively, examination of conjunctiva (bulbar & palpebral), cornea, postoperative recurrence of pterygium. The symptoms like redness, foreign body sensation, photophobia, lacrimation, dimness of vision, itching followed-up and recorded for period of 6 months.

Surgical Procedure

Group A:
The procedure is performed on an outpatient basis, using topical plus peribulbar anaesthesia. Beginning at the head of the pterygium, a disposable crescent knife was used to excise superficially the corneal portion of the pterygium to the limbus, followed by complete excision of the conjunctiva. The size of the conjunctival graft is determined by measuring the area of exposed bare sclera with caliper. Graft size was 10% more than the bare area. The conjunctival graft is thinly dissected, avoiding Tenon's capsule and episclera. After the graft is freed, it is transferred to the recipient bed and secured to adjacent conjunctiva and episclera with absorbable suture material. Topical antibiotics and corticosteroids were instilled and followed be eye pressure patching.

Group B:
Mitomycin-C with bare sclera technique was performed by removing the pterygium while keeping the overlying conjunctiva intact followed by application of a sponge soaked in Mitomycin-C 0.02% for 1 minute and irrigation by Hartmann solutions.

Postoperative Treatment was combination steroid and antibiotics drop. Patients were examined after 1st, 2nd, 6th, 12th and 24th weeks after operation. Recurrence was diagnosed when vessels invaded through the limbal area into the clear cornea. All statistical analyses were carried out using commercial software (SPSS). Observation and results of the study and statistical analysis are presented by suitable charts, figures, tables & diagrams.

Results

One hundred patients divided in two groups were included in this study. Patients ages were between 20 and 50 years (Mean age 32.39 $\pm$ 5.27). There were 31 female and 69 male patients. The demographic data and the follow up periods in the studied groups are presented in Table I, II & III. Patients with conjunctival auto graft, the postoperative complications were delayed wound healing 3(6.0), granuloma 6(12.0), giant papilla 1(2.0), recurrence 2(4.0) patients. Patients with intra-operative MITOMYCIN-C, S.P.K. 3(6.0), ulceration on the bed of pterygium 1(2.0), delayed wound healing 7(14.0), granuloma 2(4.0), and recurrence 4(8.0) were noticed (Table IV& V). In the group A, temporary graft edema was seen in all cases. Superficial keratitis lasting an average of 10 postoperative days was the most common finding observed in the Mitomycin-C group. Recurrence rate in group A was 2 (4.0) and in group B was 4 (8.0). The rate of recurrence was significantly higher in the MITOMYCIN-C group than the conjunctival auto graft group. In both groups no serious complication was noticed during the postoperative follow up period. The preoperative VA in Mitomycin-C group, between6/18 and 6/6, and patient with conjunctival auto graft were 6/12-6/6.

Postoperative VA in Mitomycin-C group patients was 6/12-6/6 and 6/6 in conjunctival auto graft patients. The improvement of VA after operation was due to correction of induced astigmatism by pterygium. All patients were followed up for 6 months.

Table I
Showing the age distribution of patient in Group-A & Group-B

Age (yrs)	Group A (n = 50) No. of patient	Group B (n = 50) No. of patient
20 - 30 years	20 (42.0)	15 (30.0)
31 - 40 years	17 (54.0)	32 (64.0)
41 - 50 years	3 (6.0)	3 (6.0)

Figure within parentheses indicate percentage

Table II
Showing the sex distribution of patient in Group-A and Group-B

Sex	Group A (n = 50)	Group B (n = 50)	Total
Male	33 (66.0)	36 (72.0)	69
Female	17 (34.0)	14 (28.0)	31
Total	50	50	100

Figure within parentheses indicate percentage

Table III
Showing the symptoms and signs among patients in Group-A and Group-B

Complaints	Group-A Frequency (%)	Group-B Frequency (%)
Burning	10 (20.0)	8 (16.0)
Irritability	12 (24.0)	13 (26.0)
FB sensation	13 (26.0)	8 (16.0)
Cosmesis	10 (20.0)	12 (24.0)
Visual disturbance	5 (10.0)	9 (18.0)

Figure within parentheses indicate percentage

Table IV
Showing the comparison of complications between Group-A & Group B

Events	Group A	Group B
SPK	0	3
Ulceration on the bed of pterygium	0	1
Delayed wound healing	3	1
Granuloma	6	2
Giant papilla	1	0
Recurrence	2	4

Table V
Showing the comparison of recurrence between Group-A & Group B

Groups	Frequency	Percentage
Group A	2	4%
Group B	4	8%

Discussion

Excision and adjunctive treatment with mitomycin C or conjunctival autograft is currently the most accepted and popular way of treating both primary and recurrent pterygium[15].

In this study the mean age of the patients was 32.39 ± 5.27 which is similar with Young et al[20] study the mean age was 37.18 years. The sex distribution of the study patients were male 66.67% and female 33.33%, this result is not similar with Young et al study[20] where male 39.13% and female 60.87%.

In this study, in the MMC, recurrence rate was 8% in comparison with 38% reported by Chen et al[16] and 10.5% by Manning et al[9] with the application of 0.4mg/ml for 3 minutes[14] and both of these studies compare and conjunctival graft. Young et al showed[22] the recurrence rate was 15.9% after intra-operative use of MMC 0.2mg/ml for 5 minutes and recurrence rate was 1.9% in the conjunctival auto graft group. In 1985, Kenyon and colleagues[17] reported the first study using conjunctival autograft transplantation and found a 7.3% secondary recurrence in patients with recurrent pterygium, whereas there was no recurrence with primary pterygium. Recurrence rate is also similar with the study done by Sarnicola V, Vannozzi L, Motolese PA[18].

A 5.35 recurrence rate was reported after procedures with a mean follow up of 24 months[17] and many ophthalmologists recommended this treatment modality for advanced primary and recurrent pterygium. Another retrospective review of pterygia treated with conjunctival autografting by Allen *et al*[19] in Australia reported a 6.5% recurrence rate with a minimum of 6 months follow up. A retrospective survey of 71 patients with primary pterygium by Figueiredo *et al*[21]showed a 1 year recurrence rate of 16% those treated with conjunctival autograft and 40% when treated with simple excision. Overall recurrence rate after conjunctival autografting are low.

In the current study no serious postoperative complication was noticed after intra-operative use of 0.2mg/ml MMC. In Young et al[20] study no scleral thinning, necrosis or any of the visually significant complications were encountered after application of intra-operative of 0.2mg/ml MMC[20].

Although postoperative topical mitomycin C therapy has proved to be a simple and effective method to prevent recurrence of prerygium, [20,22] a variety of complications, including scleral ulceration and necrosis, secondary glaucoma, corneal perforation, cataract formation, iritis, and irreversible damage to basal epithelial and limbal stem cells, have been reported.[13]

In this study, graft edema due to desiccation, handling with forceps, were disappeared in 10 days. In the Conjunctival autograft group, temporary graft edema was seen in all cases, Tenon's granuloma in five cases, graft failure in one case, and hematoma under the graft in one case[10]. Superficial keratitis lasting an average of 10 Postoperative days was the most common finding observed in the MMC group[10]

In this study patients with limbal conjunctival auto graft, the postoperative complications were delayed wound healing 3(6%), granuloma 6(12%), giant papilla 1(2%) patients. Patients with intra-operative MMC, S.P.K. 3(6%), ulceration on the bed of pterygium 1(2%), delayed wound healing 7(14%), granuloma 2(4%) were noticed. Another study by Dunn JP & Dougherty U, patient with intra-operative MMC, the postoperative complication were severe pain 4 (13.33%), foreign body sensation 2 (6.67%), corneal infiltrate 2 (6.67) patients, ulceration on the pterygial bed 1(3.33%) patients. Patients with conjunctival auto graft, severe pain 3 (10%), foreign body sensation 6 (20%), corneal infiltrate 1 (3.33%) were noticed[11,12].

During follow-up, none of the patients in MMC group showed serious side effects or reactions after 10 days. Superficial punctate keratitis was the most common finding and this can be related not only to mitomycin C application but also to general surgical trauma. Postoperative side effect of mitomycin C by patients and coexistence of ocular surface pathologies, such as severe dry eye, chronic blepharitis, or acne rosacea. [12,13]

The patients in group B took less time for operation and relatively easy, simple procedure to perform but care should be taken for proper washing of MMC from conjunctival sac. The patients in group A took more time for operation and relatively difficult procedure to perform and surgeon should be experienced. So no procedure is simple but in MMC group there are more complications.

The main limitation of sample size (n = 100) and follow up period was six months. The patients who were not able to attend the follow up visit on schedule day were excluded from the study.

Conclusion

Pterygium is a common problem in our country and surgery is the treatment of choice. The result of this study shows that both treatment modalities with excision of pterygium followed by conjunctival autograft and MMC as an adjuvant therapy after bare sclera technique achieved similar success but in MMC group chance of complications are more than that of conjunctival autograft. So conjunctival autograft is a better simple technique in the treatment of pterygium.

References

1. Kanski J. J.Clinical ophthalmology - a systemic approach 2007; 6: 242-244.
2. Saw SM, Tan D. Pterygium: prevalence, demography and risk factors. Ophthalmic Epidemiology, 1999; 6(3): 219-28.
3. Coroneo MT, Di Girolamo N, Wakefield D. The pathogenesis of pterygia. Current Opinion in Ophthalmology, 1999; 10(4): 282-8.
4. Kammoun B, Kharrat W, Zouari K, et al. Pterygium: Surgical treatment [in French}.] *Fr Ophtalmol.* 2001; 24: 823-828.
5. Sandeep Saxena,Clinical practice in opthalmolgy 2003; 1: 15-18.
6. D Ombrain A. The surgical treatment of pterygium. B.J.O 1998; 32:65-70.
7. Jaros PA, Deline VP, Pingueculae and pterygium. *Surg Ophthalmol* 1988; 33: 41-9.

8. Lam DS, Wong AK, Fan DS et al. Intra-operative Mitomycin C to prevent recurrence of pterygium after excision: 30 month follow up study. *Ophthalmology* 1998; 105: 901-4.
9. Manning CA, Class PM, Diaz MD et al. Intra-operative Mitomycin in primary pterygium excision. A prospective randomized trial. *Ophthalmology* 1997; 104: 844-8.
10. Mutlu FM, Sobaci G, Tatar T et al. A comparative study of recurrent pterygium surgery limbal conjunctival auto graft transplantation versus Mitomycin C with conjunctival flap. *Ophthalmology* 1999; 106: 817-21.
11. Dunn JP, Seorite CD, Ostler HB, et al. Development of scleral ulceration and calcification over pterygium excision and Mitomycin therapy. *Am J. Ophthalmol* 1991; 112: 301-4.
12. Dougherty U, Cardten DR, Lindstram RL, Corneo scleral melt after pterygium surgery using a single intra-operative application of Mitomycin C. *Cornea* 1996; 15: 538-40.
13. Rubinfeld RS, Pfister RR, Stein RM, et al. Serious complications at topical Mitomycin C after pterygia surgery. *Ophthalmology* 1992; 99: 1647-54.
14. Tseng SCG: Concept and application of limbal stem cells. Eye1989; 3: 141-157.
15. Mohammed I. Treatment of pterygium. Ann Afr Med. 2011; 10(3): 197-203.
16. Chen PP, Abyasu RG, Kaza V , et al. A randomized trial comparing Mitomycin C and conjunctival auto graft after excision of primary pterygium. *Am J Ophthalmol* 1995; 120: 151-60.
17. Kenyon KR, Wagoner MD, Hettinger ME. Conjunctival auto graft transplantation for advanced and recurrent pterygium. *Ophthalmology* 1985; 92: 1461-70.
18. Sarnicola V, Vannozzi L, Motolese PA. Recurrence rate using fibrin glue-assisted ipsilateral conjunctival autograft in pterygium surgery: 2-year follow-up. Cornea. 2010; 29(11): 1211-4.
19. Allan BDS et al: Pterygium excision with conjunctival auto grafting: an effective and safe technique, *Br J Ophthalmology,1993;* 77: 698.
20. Young A.L. Leung GY, Wong WKK, Cheng L L, Lam D S C, Randomized trial comparing 0.02% Mitomycin C and Limbal conjunctival auto graft after excision of primary pterygium. *Br J Ophthalmology, 1996;* 3: 674-677.
21. Figueiredo RS, Cohen EJ, Gomens JAP et al: Conjunctiva auto graft for pterygium surgery: how well does it prevent recurrence? *Ophthalmic surg Lasers* 1997; 28: 99-104.
22. Hayasaka S, Noda S, Yamamoto Y, Setogawa T. Postoperative instillation of low-dose mitomycin C in the treatment of primary pterygium. Am J Ophthalmol 1988; 106: 715-8.

A case of Primary Hyperparathyroidism Presented with Recurrent Abdominal Pain

Ahmed AS[1], Uddin M[2], Islam K[3].

Abstract

The association between recurrent abdominal pain and hyperparathyroidism (PHPT) is controversial. We report a 45-year-old man who presented with recurrent episodes of abdominal pain. Primary hyperparathyroidism was diagnosed after several episodes of abdominal pain. Two years after successful parathyroid surgery, there has been no recurrence of abdominal pain and his serum calcium is within the normal range.

KuMCJ, June 2017; Vol. 1 (1): 31-32

Introduction

Primary Hyperparathyroidism (PHPT) syndrome is an endocrine disorder, characterized by excessive secretion of parathyroid hormone from one or more parathyroid glands[1]. The elevation of PTH leads to hypercalcemia and hypophosphatemia; patients may present with classic skeletal disease, recurrent nephrolithiasis, or be asymptomatic, detection routine biochemical screening. A dramatic increase in the incidence of PHPT occurred in the late 1960s, due primarily to the introduction of the multichannel autoanalyzer. The clinical profile of PHPT in the western countries had shifted from a symptomatic disorder, toward a more asymptomatic state[2]. However PTPH has a variable clinical expression and symptomatic PHPT is still the predominant form of disease in many developing countries, skeletal manifestation (osteitis fibrosa cystica) being very common[3].

Case Report

Mr. Shamir 45 years garments salesman was admitted to district hospital with recurrent episode of abdominal pain. One year after the first episode ultrasonogram of abdomen revealed cholelithiasis and bilateral nephro-calcinosis but did not reveal pancreatitis, with no calcification or dilated bile duct, repeated assay of serum amylase and lipase were normal during the episode of abdominal pain. Over the next 3 years he suffered several episodes of acute abdominal pain, vomiting and losing his body weight.

He was performed laparoscopic cholecystectomy but his agony did not resolve. Then he was admitted in Kushtia Medical College Hospital, Kushtia. He was occasionally alcoholic. Investigation revealed elevated serum calcium and parathyroid hormone level but 24-hour urinary calcium level normal, serum albumin 4.9 gm/dl, Endoscopy of upper GIT was normal, X-ray of KUB shows bilateral nephrocalcinosis. Nephrocalcinosis and elevated serum calcium gave us clue that he may be having underlying hypercalcemic disorder or related disorders pointing to nephrocalcinosis.

After diagnosis of hypercalcemic disorder, serum biochemical parameters were repeated. Repeat serum calcium was 3.54 mmol/l, phosphate 2.0 mg/dl, albumin 4.9 gm/dl, creatinine 1.5 mg/dl, serum alkaline phosphatase 284 U/L and serum iPTH was 69.36 pmol/l. On the basis of biochemical parameters, a diagnosis of PHPT was made. Right sided parathyroid adenoma was localized on a radionuclide parathyroid scan (99mTC MIBI) [Figure 1], ultrasonography of neck revealed a hypoechoic lesion 3.0 x 1.5 x 1.1 cm at the posterior inferior pole of right lobe of thyroid and skeletal survey was done which was normal.

1. Dr. Abu Saleh Ahamed, FCPS (Medicine); MD (Nephrology), Assistant Professor, Department of nephrology, Kushtia medical college.
2. Dr. Moin Udding. MBBS; MCPS (Medicine), Junior Consultant (Medicine), Department of medicine, Kushtia Medical College Hospital.
3. Dr. Khariul Islam. MBBS; Assistant- Registrar. Department of medicine. Kushtia medical college Hospital.

Address of correspondence: Dr. Abu Saleh Ahamed, Assistant Professor, Department of nephrology, Kushtia medical college.

He denied any history of bone pain, bone fractures, neuropsychiatric symptoms, or muscle weakness.

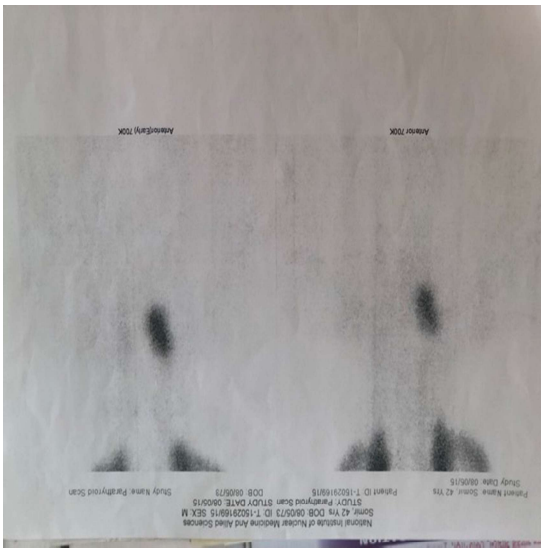


Figure 1: Radionuclide parathyroid scan.

Parathyroid adenoma was removed. The postoperative period was uneventful. Two years after successful parathyroid surgery, there has been no recurrence of abdominal pain and his serum calcium is within the normal range. His current biochemical parameters are serum calcium 9.4 mg/dl, PTH 34.50 pg/ml, and TSH 3.01 uIU/ml.

Discussion

The most common cause of primary hyperparathyroidism is a solitary adenoma which accounts for 80%, hyperplasia for 15% and parathyroid carcinoma for 1%[4]. In our case the cause of PHPT was solitary adenoma that coincides with the reference. The classical presentation of primary hyperparathyroidism presents with a pentad of symptoms, i.e., renal stones, painful bones, abdominal groans, psychic moans, and overtones has now become exceedingly rare. Asymptomatic primary hyperparathyroidism can lead to complications like nephrolithiasis, pancreatitis, gastrointestinal ulcer, and endangering hypercalcemic crisis[4]. Mr. Shomir presented to us with only nephrolithiasis may not be present simultaneously. The typical biochemical abnormalities seen in these patients are hypercalcemia, hypophosphatemia, elevated serum parathormone, and alkaline phosphatase levels[5]. In our case all of these biochemical parameters were along with reference except normal ALP which can be explained by absence of skeletal lesions. Sestamibi scan is the gold standard for the diagnosis of parathyroid adenoma.

All patients with a past history of abdominal pain had suffered several attacks. In our patient there was delay of 3 years before the diagnosis of PHPT was established. Serum calcium estimation after two or three episodes of abdominal pain would have eliminated this delay. It is important to estimate serum calcium after recurrent episode of unexplained abdominal pain. This will minimize the delay before the diagnosis of PHPT is made.

Despite its rarity, a cause and effect relationship is still in dilemma which actually alleviates abdominal pain after parathyroidectomy. Nearly 100% improvement in abdominal pain symptoms after the cure of PHPT has been reported.

There are several mechanisms of abdominal pain. But patient did not have any pancreatitis or peptic ulcer disease. So, another explanation is hyper-viscosity. The calcium level is probably of major importance in the development of abdominal pain.

References

1. Bilezikian JP, Brandi ML, Rubin M, Silverberg SJ, Primary hyperparathyroidism: New concepts in clinical, densitometric and biochemical features. J Intern Med. 2005; 257:6-17. [PubMed]
2. Mundy GR, Cove DH, Fisken R. Primary hyperparathyroidism changes in the pattern of clinical presentation. Lancet. 1980; 1:1317-20. [PubMed]
3. Silverberg SJ, Sahne E, Demster DW, Bilezikian JP, The effects of vitamin D insufficiency inpatients with primary hyperparathyroidism. Am J Med. 1990; 107: 561-7. [PubMed]
4. Beatti GC, Ravi NR, Lewis M, Williams H, Blair AW, Campbell IW, Browning GGP. Rare presentation of maternal hyperparathyroidism. BMJ 2000 Jul 22; 3321(7255): 223-224.
5. Ip p. Neonatal convulsion revealing maternal hyperparathyroidism: an unusual case of late neonatal hyperparathyroidism. Arch Gynecol Obstet 2003 Aug; 268(3): 227-22.

Systemic Sclerosis, a Rare Cause of Connective Tissue Diseases: A Case Report

Siddique MAB[1], Uddin MB[2], Ahmed F[3], Karim MA[4], Hossain MN[5]

Abstract

Systemic sclerosis is a rare autoimmune multisystem connective tissue disease in childhood age group which is characterized by fibrosis of the skin, internal organs and blood vessels. This condition can be systemic and localized. It is more common in female than male. The first specific clinical sign of scleroderma is the swelling on the skin of hands and fingers. It also causes various changes at oral and facial tissues. Before the development specific feature few patients may present with some nonspecific feature like fever, anorexia, weight loss and joint pain which makes difficulties in diagnosis. The aim of this case report is to present A 14 year old girl with systemic sclerosis present with such type of nonspecific features and to raise the awareness early to detect the disease for better outcome.

KuMCJ, June 2017; Vol. 1 (1):33-35

Introduction

Systemic sclerosis is a rare connective tissue disease of unknown etiology characterized by increase deposition of collagen leading to fibrosis and subsequent degeneration of the skin and internal organs[1, 2]. The word scleroderma originates from Greek word 'scleros' meaning hard and 'derma' which means skin[3]. Scleroderma and Raynaud's phenomenon are hallmark of the diseases[4] and the peak age is 30-50 years. Female and male ratio is 4:1. Child represents less than 10% of all cases. Prevalence is 10-20/100000[5]. It has been classified as (a) systemic sclerosis, (b) localized scleroderma, (c) Eosinophilic fascitis, (d) Secondary forms (drug induced, chemical induced) and (e) Pseudoscleroderma[5, 6].

The mechanism of disease appears to be a combination of a vasculopathy, autoimmunity, immune activation and fibrosis[5]. Isolated cases have been reported in which a systemic sclerosis like disease was triggered by exposure to silica dust, vinyl chloride, trimchlomethylene[6]. Triggers, including trauma, infection, and, possibly, subclinical graft versus host reaction from persistent maternal cells (microchimerism), injure vascular endothelial cells, resulting in increased expression of adhesion molecules. These molecules entrap platelets and inflammatory cells, resulting in vascular changes with manifestations such as Raynaud phenomenon and pulmonary hypertension. Inflammatory cells infiltrate the area of initial vascular damage, causing further vascular damage and resulting in thickened artery walls and reduction in capillary numbers. Fibroblasts synthesize excessive amounts of collagen, resulting in fibrosis and subsequent lipoatrophy, loss of sweat glands and hair follicles. In late stages, the entire dermis may be replaced by compact collagen fibers.[5]

The first specific clinical sign of scleroderma is the non-pitting edema on the skin of hand and fingers. Subsequently, the skin becomes shiny and taut, and distal skin creases disappear. The face and neck are usually involved next, with thinning of the lips and radial furrowing[6]. Raynaud's phenomenon is a universal feature and can precede other features by many years. Involvement of small blood vessels in the extremities may cause critical tissue ischaemia, leading to skin ulceration over pressure area[7]. The most

1. Dr. Md. Abu Bakar Siddique, DCH; FCPS (Pediatrics), Assistant Professor, Pediatrics, Kushtia Medical College, Kushtia.
2. Dr. Md. Bashir Uddin, MD (Cardiology), Junior Consultant, Cardiology, Kushtia Medical Collge Hospital.
3. Dr. Firoz Ahmed, MD (Pediatrics), Junior consultant, Pediatrics, Kushtia Medical Collge Hospital.
4. Dr. Md. Abdul Karim, MBBS, Kushtia Medical College.
5. Dr. Md. Nurnobi Hossain, MBBS, Kushtia Medical College.

Address of Correspondence: Dr. Md. Abu Bakar Siddique, Assistant Professor, Pediatrics, Kushtia Medical College, Kushtia, Mobile: 01731-435045, E.mail: absiddique45@gmail.com

common clinical feature of musculoskeletal involvement is arthralgia; less frequent features are arthritis, flexion contractures, stiffness of fingers, wrists and ankles, proximal muscle weakness and tendon sheath involvement[8]. Up to 8% of systemic sclerosis patients develop severe gastrointestinal tract symptoms[9] like oesophagitis with dysphagia, haematemesis, malaena and occasionally outlet obstruction. Pulmonary involvement consists most often of interstitial fibrosis and pulmonary vascular disease leading to pulmonary arterial hypertension (PAH)[10]. Renal crisis occurs in systemic sclerosis[11] and Hypertensive renal crisis is much more likely to occur in dcSS than in lcSS, and in patients with topoisomerase 1 antibodies. ESR is usually elevated and raised levels of IgG but CRP is normal. ANA is positive in about 70%, and approximately 30% of patients with dcSS have antibodies to topoisomerase 1[12]. About 60% of patients with CREST syndrome have anticentromere antibodies. No treatments are available that halt or reverse the fibrotic changes that underlie the disease. The focus of management, therefore, is to ameliorate the effects of the disease on target organs.

The aim of this case report is that systemic sclerosis may present with nonspecific signs and symptoms for prolonged period of time before establishment of specific diagnosis.

Case Report

Rashmi, 14 years, school going girl of a poor family presented in the hospital (KuMCH) with the complaints of Fever, anorexia, weight loss for 2 years, multiple joint pain for 1 year and Skin changes for 1 month. Actually she was reasonably well about 2 years back; then she developed continuous low grade fever with maximum recorded temperature (100ºF), not associated with chills and rigor and reduced by taking paracetamol and sponging. Fever is associated with anorexia and within last 1 year her weight has reduced markedly. that she could not move, sit or walk but not associated with joint swelling, morning stiffness and it is slightly relieved by taking analgesics. She has no previous medical and surgical events at all. Her father and younger brother suffered from tuberculosis about 10 and 7 years back respectively and treated with appropriate Anti-TB drug regimen under category 1.

She has no allergy to drugs or foods. From the very beginning of her disease, she was treated by many local physicians as well as specialist doctors. But they couldn't come to the diagnosis despite of doing many investigations and longtime observations but got treatment of pyrexia of unknown origin for 2 years without any improvement. Recently few changes of skin especially in trunk and distal part of limb are noticed and skin became dry, scaly and slightly taught.

She was undernourished, mildly anemic, somewhat depressive but intelligent and well co-operative. Joint tenderness is present without any swelling and movement is restricted, no lymphadenopathy, jaundice, cyanosis, no detected systemic abnormality. Skin is smooth, shiny in face and it is dry, scaly, slight tout with somewhere salt and pepper appearance in the trunk. Alopecia is also present. Her investigations, those have been done recently reveals all general reports including ANA and Anti ds DNA are normal but ENA profile shows SmD1 and Scl70 positive.

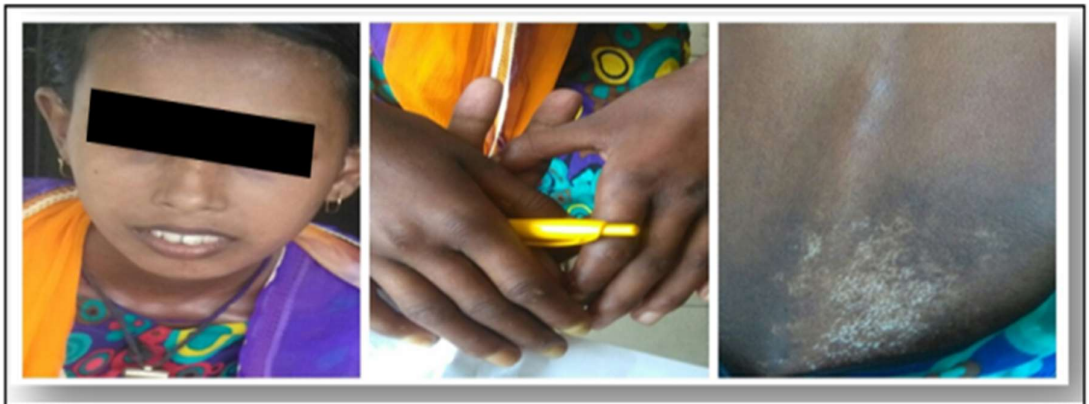


Figure 1: showing shinny face with alopecia, non-specific finding of finger, salt and pepper appearance in the trunk.

Discussion

Systemic sclerosis is rare connective tissue disease and very rare in childhood. Prevalence is about 10-20/100000[5]. The etiology of SSc is unknown. It is the one of the most important rheumatic diseases in childhood. Fibrosis of the skin, subcutaneous tissues, and internal organs are some of the characteristic outcomes. Therefore, early detection of the severity of the disease may play a significant role in establishing the most effective therapeutic regimen[12-14]. In all sclerodermic subjects, while 10% shows the disease symptoms before the age of 20, only 1 to 2% shows the symptoms before the age of 10. The mean age of onset is 8.1 years, and the peak age is between 10 and 16 years[15,16]. In our patient, the symptoms started at the age of twelve. The girl, Rashmi was early manifested by fever, anorexia, weight loss with others non-specific clinical findings. So it is difficult to diagnosis earlier on the clinical basis and she was treated about 2 years as pyrexia of unknown origin. She was thought TB patient because of positive family history that her father and younger brother were suffering from TB about 10 and 7 years ago respectively, also with low grade fever, anorexia, but investigation shows MT test, sputum for AFB, gene xpart negative. By this time, she passed long time with non-specific findings of systemic sclerosis.

Multiple organ involvement in children at the time of diagnosis may be less common than in adults. The majority of children with SSc presents with skin changes (tightening, thinning, atrophy) of the hands and face, and/or the Raynaud's phenomenon. A multicenter retrospective study of 153 children with JSSc revealed that the Raynaud's phenomenon was the most frequent symptom in these patients (75%). Other involvements of the skin can be less frequent such as sclerodactyly, edema, and calcinosis[17]. Difficulty in opening the mouth, tightness and slimness on the lip skin may be seen. Similarly, our patient suffered from characteristics skin changes weight loss, arthritis and but she had no Raynaud's phenomenon and difficulty in opening her mouth.

Finally Rashmi admitted in KuMCH and was examined and suspected as a case of connective tissue disease with differential diagnosis SLE and Systemic sclerosis. Then investigation was done according to the plan and ultimately diagnosed as systemic sclerosis. Now she is under supervision.

Conclusion

Systemic sclerosis is rare life threatening disease of unknown etiology involving all most all organs and initially presents with non-specific signs and symptoms. Physician should keep in mind that if non-specific symptoms do not match with the common diseases then should focus on rare diseases to early diagnosis for prolongation of survival, preservation of optimal function and substantially improvement of quality of life.

References

1. Sunumu BO., Atlan G., Kose TE., Erdem TL., Qzcan I; Scleroderma: A Case Report. J Istanbul Univ Fac Dent 2015; 49(2): 31-34.
2. Sinnathurai P, Schrieber L. Treatment of Raynaud phenomenon in systemic sclerosis. Intern Med J. 2013 May; 43(5): 476-83.
3. Albilia JB, Lam DK, Blanas N, Cloakie CM, Sándor GK. Small mouths big problems? A Review of Scleroderma and its oral health implications, J Can Dent Assoc, 2007; 73(9): 831-836.
4. Hinchcliff M. and Varga J., Systemic sclerosis/scleroderma: a treatable multisystem disease. Am Fam Physician. 2008 Oct 15; 78(8): 961-968
5. Mater HAV and Rabinovich CE. Scleroderma and Raynaud Phenomenon. In: Kliegman RM, Stanton BF, Schor NF, Geme III JWS, Behrman RE, editors. Nelson Text Book of Pediatrics Nelson Textbook of Pediatrics, First south Asia edition. Vol. 1, India: Elsevier Inc; 2016. P 1187.
6. Ralston SH, McInnes IB. Rheumatology and Bone disease. In: Walker BR, Colledge NR, Ralston SH, Penman ID, editors. Davidson's Principles and Practices of Medicine, 22rd ed. London: Churchil Livingston Elsevier; 2014. P 1037.
7. Sunderkötter C, et al. Hautarzt. Raynaud phenomenon in dermatology: Part 2: therapy. Zeitschrift fur Dermatologie, Venerologie, und Verwandte Gebiete; 01 Oct *2006;* 57(10): 927-38.
8. Randone SB, Guiducci S, Cerinic MM. Musculoskeletal involvement in systemic sclerosis. Best Pract Res Clin Rheumatol. 2008 Apr; 22(2): 339-50.
9. Kumar S. Singh J. and Rattan S. et al. Review article: Pathogenesis and Clinical Manifestations of Gastrointestinal Involvement in Systemic Sclerosis. Aliment Pharmacol Ther. 2017 Apr; 45(7): 883–898.
10. Shahin AA. Pulmonary involvement in systemic sclerosis. Treat Respir Med. 2006; 5(6): 429-36.
11. Steen VD.Renal involvement in systemic sclerosis. Clin Dermatol. 1994 Apr-Jun; 12(2): 253-8.
12. David M. A case of scleroderma mentioned by Hippocrates in his aphorisms. Korot 1981; 8: 61-3.
13. Rodnan GP. When is scleroderma not scleroderma? The differential diagnosis of progressive systemic sclerosis. Bull Rheum Dis 1981; 31: 7-10.

14. Preliminary criteria for the classification of systemic sclerosis (scleroderma). Subcommittee for scleroderma criteria of the American Rheumatism Association Diagnostic and Therapeutic Criteria Committee. Arthritis Rheum 1980; 23: 581-90.
15. Scalapino K, Arkachaisri T, Lucas M, Fertig N, Helfrich DJ, Londino AV Jr, et al. Childhood onset systemic sclerosis: classification, clinical and serologic features, and survival in comparison with adult onset disease. J Rheumatol 2006; 33: 1004-13.
16. Martini G, Foeldvari I, Russo R, Cuttica R, Eberhard A, Ravelli A, et al. Systemic sclerosis in childhood: clinical and immun ologic features of 153 patients in an international database. Arthritis Rheum 2006; 54: 3971-8.
17. Pope JE. Musculoskeletal involvement in scleroderma. Rheum Dis Clin North Am 2003; 29: 391-408.

Editorial policy & Instruction to Authors

KuMCJ, June 2017; Vol. 1 (No. 1): 37-40

Kushtia Medical college journal is published twice in a year in the month of June and December. Manuscript written in English related to medical science will be received throughout the year and will be considered for publication provided that the article has not been already published or been submitted elsewhere for publication.

Articles submitted for publication may be in the form of original articles, case reports, review articles, recent advances, new techniques, book review on clinical or medical education, adverse drug reactions or a letter to the editor. Author should obtain & submit written permission from appropriate authority if the manuscript contents any table, data or illustrations from any previously published article of any journal.

Submission of article

Authors are requested to send three (03) hard copies and one soft copy (a CD or DVD or an email to kumcjournal.bd@gmail.com) to the editor in chief. Authors must keep a hard & soft copy with them as reference. Manuscript must be accompanied by a covering letter addressing the Editor in Chief and signed by principal author declaring that (a). It has neither been previously published nor being submitted elsewhere for publication, (b). The manuscript has been read and approved by all the authors and (c). The order of authorship is the joint decision of principal author and all the co-authors.

Editorial Procedure

All manuscripts on receipt are initially screened by editors and registered. The manuscripts are sent to two or more reviewers for masking peer review. Thereafter a feedback is provided to the principle author. One masked copy of the proofs may be sent to principal author for careful correction of errors if any. The ultimate authority to accept or reject the article rest with the Editorial Board. Editorial board may ask any relevant explanation of any part or whole of the article form the author. Failure to comply or explanation will reject the article for publication. No article is rejected unless similar comments are received from at least two reviewers. Ethical aspect and conflict of interest are evaluated by the editorial board at the time of review of the article. Any conflict related to Helsinki declaration of 1975 as revised in 1983 will eliminate the article for publication. On acceptance of manuscript submitted for publication it implies transfer of copyright from author to publisher.

Authorship

Authorship credit should be based only on substantial contribution to:

a. Conception and design or analysis and interpretation of data.
b. Drafting the article or revising it critically for important intellectual content.
c. Final approval of the version to be published. “Conditions (a), (b) & (c) must all be met.” Only funding, collection of data or general supervision of a research group is not sufficient for authorship.Editors may ask authors to describe what each author contributed.

Technical requirements for manuscripts

a. Double-space all parts of manuscripts.
b. Begin each section or component on a new page.
c. Review the sequence for original article- Title page, Abstract and key words, text, acknowledgments (not mandatory), references, tables (each on separate page) and legends.
d. Illustrations (unmounted prints) should be no larger than 203 X 254 mm (8 X 10 inch).
e. Keep copies of everything submitted.
f. Font is to be “Times new Roman”, font size is to be “12”.
g. Serial number of the tables is to be in Roman style (e.g. Table – I, Table – II, Table – III ect.
h. Serial number of the figures is to be in Arabic style (e.g. Figure – 1, Figure – 2, Figure – 3 ect.
i. Paper should be of good quality A_4 size with one inch margins on the top, bottom, right and left sides and articles should be typed on one side of the paper.
j. Number pages consecutively beginning with title page. Put the number on upper or lower right hand corner of each page.

Preparation of the manuscript

The text of observational and experimental article is usually (but not necessarily) divided into sections with the headings – introduction, materials and methods, results and discussion. Long articles may need subheadings within some sections (especially results and discussions) to clarify their content. Other types of articles such as case reports, review articles and editorials can be in other formats. The tittle page should be the first page of all manuscripts.

Title page

Title page should carry

i. Title of the article which should be concise, informative, self explanatory and catchy.
ii. Article type
iii. Full name for each author including highest degree and affiliation.
iv. Contact information for the principal / corresponding author (Name, Address, Mobile and Email).

Format of manuscript

The format of the manuscript depends on the type of the article , Each component should begin in a new page in a sequential order and contain relevant information.

A) Original articles (Length: 2000 ± 500 words)

The following sequence of headings should be followed for original articles
i. Abstract, ii. Introduction, iii. Materials and Methods, iv. Results, v. Discussion, vi. Acknowledgments, vii. References.

i. Abstract

The second page should carry an abstract (of no more than 150 words for unstructured abstract or 250 words for structured abstract).Unstructured abstract is preferred. The abstract should state the purposes or aims of the study, basic procedures (selection of study subjects; observational and analytic methods), main findings (specific data and their statistical significance, if any) and the principal conclusions. Below the abstract authors should provide and identify 3 to 10 key words or short phrases. Terms from the medical subject headings (MeSH) list of index Medicus" should be used: if suitable are not yet available for recently introduced terms, present terms may be used.

ii. Introduction

Provide the wider context that generated the study. This should summarize the rationale for and purpose of the study. Give only strictly pertinent references and do not include data or conclusions form the study being reported.

iii. Materials and Methods

This section details the study design to ensure reproducibility of the research. It will include case selection, place and time of study, sampling technique, data collection, ethical aspect and the statistics. Statistical soft wire if used should be specified Studies that involve human subjects must be approved by an institutional ethical review committee. Mention precisely all drugs and chemicals if used including generic name(s) and route(s) ofadministration. Reports of randomized clinical trials should present information on all major study elements including the protocol (study population, interventions or exposures, outcomes and the rationale for statistical analysis), assignment of intervention (methods of randomization, concealment of allocation to treatment groups), and the method of masking (blinding).

iv. Results

Should be presented in logical sequence in the text, tables and illustrations. The order of presentation of results should parallel the order of the materials and method section. Do not repeat in the text all data in the tables or illustrations; emphasize only important observations.

a) Tables

Type or print out each table with double spacing on a separated sheet of paper, be numbered consecutively using Roman numerals in order of their first citation in the text and supply a brief title for each in the appropriate headers. Do not use internal horizontal and vertical rules. Be sure that each table is cited in the text. Place explanatory matter in foot notes not in the heading. Explain in footnotes all non standard abbreviations that are used in each table.

b) Illustrations (Figures)

Figures include graphs, charts and photographs. Figures should be professionally drawn and photographed; freehand or type written lettering is unacceptable. Instead of original drawings, x-ray film and other materials, send sharp, glossy coloured photographic prints, usually 127 X 178 mm (5 X 7 inch) but no larger than 203 X 254 mm (8 X 10 inch). Figures should be numbered consecutively according to order in which they have been first cited in the text. Each figure should be accompanied by a legend at the bottom. Do not write on the back of the figure or scratch or mar them by using paper clips. Do not

bend figures or mount them on cardboard. Written permission is needed when photographs of people are used and if person is unidentifiable then written permission is not mandatory.

v. Discussion

Emphasize the new and important aspects of the study and conclusions that follow from them. This section should include implications of the findings and their limitation including implications for future research. The section should not merelyrecapitulate the results but compare and contrast the result with other relevant studies. Do not make statement on economic benefits and costs unless the manuscript includes economic data and analyses.

vi. Conclusion

A brief concluding paragraph presenting the implications of the study results and possible new research directions on the subject.

vii.Acknowledgments (not mandatory)

Institution, organization, sponsor, individuals or any bodies may be acknowledged in the for financial, material, technical or any form of assistance or contribution to the work with their consent.

B) Case reports (Length: 1000 ± 200 words)

This should cover uncommon and / or interesting cases with appropriate confirmation process. The number of case may be one or two. Case report should consist of an unstructured abstract (100± 200 words) ,introduction, case study including clinical presentation, diagnosis and outcome as well as discussion that includes brief review of relevant literatures, one table and not more than two figures.

C) Review articles (Length: 3000 ± 500 words)

Submission of reviews and perspectives covering topics of current interest are encouraged. Review articles should be focused on topic of health interes and up to date. Components should include: unstructured abstract (250 ± 50 words),context, objective, data sources , study selection, data extraction, results, analysis and conclusion.

D) Letters to Editor (Length: 300 ± 50 words)

Letters in response to articles published in Kushtia Medical College Journal are welcome. The letter will be followed by comments from original author (where appropriate) of the article in question

E) References:

References should be arranged according to VANCOUVER system which is based on an American National Standards Institute (ANSI) style for its database adapted by the National Library of Medicine (NLM) and used in index medicus. In the text references should be numbered by Arabic numeral in the superscript consecutively in the order in which they are first mention in the text. In the reference list it should be numbered sequentially as it appears in the text. Names and initials of the authors are listed if there are six or fewer, otherwise first six and than et al is added. Authors are responsible for the accuracy of the references cited, these should be checked against original documents before the article is submitted.

Examples of references

i) Standard journal article

Vega KJ, Pina I, Krebsky B.Heart transplantation is associated with increased risk for pancreatobiliary disease. Ann Intern Med 1996; 124: 980-3.

ii) Books

a) Personal Author(s)

Ringsven MK, Bond D. Gerentology and leadership skills for nurses. 2nd ed Albany(NY): Delmar publishers; 1996. p 28-35.

b) Editor(s), compiler(s) as author

Norman IJ, Redfan SI, editors. Mental health care for elderly people. 2nd ed.New York: Churchil Livingston; 1996 p 125-129.

c) Chapter in a book

Philips SJ,Whisnant JP. Hypertension and stroke. In : Laragh JH, Brenner BM, editors. HYpertension: pathophysiology,diagnosis and management 2nd ed.New York:Raven press; 1995. p465-78.

iii). Electronic material

(Standard journal article on the internate.)
Kaul S, Diamond GA. Good enough:a primer on the analysis and interpretation of noninferiority trials. Ann Intern Med (internet. 2006 jul 4 (cited 2007 jan4); 145 (1): 662-9 Available from:http:/www.annals.org/cgi/reprint/145/1/62.pdf.

.

Units of Measurement:

Measurement of length, weight and volume be reported in metric units. Measurement of temperature in Celsius, blood pressure in mm of Hg. All hematological and clinical chemistry measurement be reported in SI units. Editors may request non-S1 units be added by authors before publication.

Letter to the Editor in Chief

To

The Editor in Chief
Kushtia Medical College Journal
Kushtia, Bangladesh.

Subject: Submission of an article for publication.

Dear Sir,

I, the undersigned, give an undertaking to the following with regard to our original article / case report / review article titled "..... ...." submitted for publication in your journal.

The article mentioned above has neither been previously published nor being submitted elsewhere for publication.
The manuscript has been read and approved by all the authors.
The order of authorship is the joint decision of principal author and all the co-authors.

Thanking you in anticipation.

Best Regards.

Name & highest academic qualification
Designation:
Department:
Institution:
Mobile No:.
E-mail:

Attachment:
1. Article- Three Copies
2. Figures and Tables – Three copies.
3. Soft copy of the article in "Word" & "Excel" format.

Title Page

Title of the article: .....

Authors:

Principal Author

1. Name
   Highest Academic Qualification
   Designation & Department
   Institution

Co-Authors:

2. Name
   Highest Academic Qualification
   Designation & Department
   Institution

3. Name
   Highest Academic Qualification
   Designation & Department
   Institution

4. Name
   Highest Academic Qualification
   Designation & Department
   Institution

5. etc.

Address for correspondance-
Name of the correspondence
Designation & Department:
Institution:
Mobile No.:
E-mail:

(Signature of Principal Author)

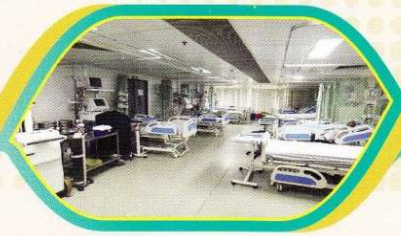
BRB HOSPITALS LIMITED

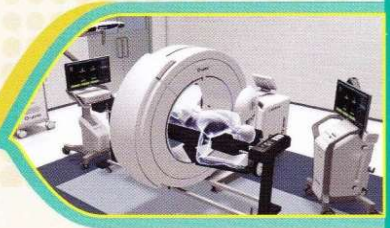
CENTER OF EXCELLENCE

- Gastro Liver Center
- Hepato Biliary Pancreatic Surgery Center
- Mother & Child Care Center
- Brain & Spine Center
- Nephrology & Urology Center
- Bone & Joint Center

SPECIALIZED SERVICES

Internal Medicine	Hematology/Transfusion Medicine
Cardiology	ENT, Head & Neck Surgery
Respiratory Medicine	Plastic & Reconstructive Surgery
Medical Oncology	Physiotherapy & Rehabilitation
ICU, NICU & HDU	Dermatology
Endocrinology & Diabetology	Hemodialysis
General & Laparoscopic Surgery	Food & Nutrition





For Appointment: 01777 764 800-3

For Details: 01709 63 58 63

BRB HOSPITALS LIMITED

77/A, East Rajabazar, West Panthapath, Dhaka-1215., Tel: 9140333, 9140346
E-mail: brbhospitals@gmail.com, Web: www.brbhospital.com

a concern of BRB

Printed by: Asian Colour Printing, 130 DIT Extension Road, Fakirerpool, Dhaka-1000
Phone : 49357726, 58313186, E-mail: asianclr@gmail.com