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Editorial

Non-Alcoholic Fatty Liver Disease: An Emerging Liver Disease

Non-alcoholic fatty liver disease (NAFLD) is an increasingly recognized cause of liver-related morbidity and mortality, comprising a spectrum of hepatic diseases ranging from simple steatosis to full-blown non-alcoholic steatohepatitis (NASH), cirrhosis and rarely hepatocellular carcinoma¹. It is the most common form of liver disease in the world. Estimates of current prevalence range from 24-42% in western countries and 5-40% in Asian countries. In USA, it is estimated that a third of the general population has NAFLD^{2,3}.

The long-term hepatic prognosis of patients with NAFLD depends on the histological stage of disease at presentation⁴. Among patients with simple steatosis 12-40% will develop NASH with early fibrosis after 8-13 years. For patients presenting with NASH and early fibrosis, around 15% will develop cirrhosis and/or evidence of hepatic decompensation over the same time period, increasing to 25% of patients with advanced precirrhotic fibrosis at baseline. About 7% of subjects with decompensated cirrhosis associated with NAFLD will develop a hepatocellular carcinoma (HCC) within 10 years, while 50% will require a transplant or die from a liver related cause. The risk of HCC in NAFLD-related cirrhosis is comparable to that in cirrhosis associated with alcohol or hepatitis C⁵.

Depending on the etiology, two types of NAFLD are recognized. Most common form is primary NAFLD, associated with metabolic syndrome, now considered the hepatic manifestation of the metabolic syndrome. Less common cause is secondary NAFLD associated with disorders of lipid metabolism, glycogen storage disease, drug toxicity (amiodarone, tamoxifen, methotrexate, corticosteroid and anti retroviral therapy), total parenteral nutrition, weight loss after bariatric surgery and environmental toxicity⁶.

The pathologic process in NAFLD is believed to evolve in two steps, "The two hits theory". The first step involves a discrepancy between the influx and synthesis of hepatic lipid on one side and their oxidation and transport on the other, this discrepancy causing the development of steatosis. The second step is oxidative stress, leading to hepatocytes injury, inflammation and fibrosis⁷.

NAFLD is usually clinically silent. Symptoms, if present, are minimal and non-specific such as fatigue, lethargy and right upper quadrant discomfort. Most patient seeks care because of incidental finding of elevated aminotransferase level or imaging studies suggesting fatty liver and/or hepatomegaly.

NAFLD remains a diagnosis of exclusion of other liver disease. Most cases of unexplained liver enzyme elevation are probably NAFLD, however the vast majority of patient with NAFLD have normal liver blood test⁸. NAFLD should be suspected in all patients with established risk factors including component of metabolic syndrome, polycystic ovary syndrome, obstructive sleep apnoea regardless of liver function test.

In NAFLD, there is usually mild elevation of transaminases. ALT/AST is greater than one unless there is advanced fibrosis in 90% of cases. Alkaline phosphatase is less often elevated but gamma glutamate transferase (GGT) is frequently elevated. Other blood tests are aimed to detect associated condition such as dyslipidemia, diabetes mellitus and to exclude other diseases e.g. viral hepatitis (HbsAg, anti HCV, anti HAV IgM, anti HEV IgM), congenital causes (Wilson disease, haemochromatosis, alpha-1 antitrypsin deficiency) and drug induced liver disease.

Currently available imaging modalities including ultrasound, CT (computed tomography) and MRI (magnetic resonance imaging) are all excellent at detecting steatosis (when more than around a third of liver is affected) but none is reliable in detecting NASH or fibrosis⁹. Newer imaging techniques including MR (magnetic resonance) spectroscopy and transient elastography show promise but require further study prior to routine use for disease staging^{10,11}.

Liver biopsy is the gold standard in term of its accuracy in diagnosis of NAFLD. The main indication of liver biopsy is the accurate staging of the disease as this will help in management planning and give idea about prognosis.

Regarding management, there is no as yet firm evidence-based treatment for NAFLD. Therapy is currently directing at treating components of the metabolic syndrome which may also be beneficial for the liver.

For obesity, diet and exercise is well established in controlling weight and improving insulin resistance and thus improve liver histology. In some studies, bariatric surgery has shown to improve and even resolve metabolic syndrome⁸. Orlistat is a reversible inhibitor of gastric and pancreatic lipase shown encouraging improvement in liver histology in patient in NASH⁸.

Metformin in patient with NAFLD have shown encouraging result in reducing ALT level, liver fat, necroinflammation and fibrosis compared with either vitamin E treatment or weight reducing diet⁸. Vitamin E and pioglitazone can also be used in the treatment of biopsy proven NASH with encouraging result. Ursodeoxycholic acid (UDCA) has no proven value in the treatment of NASH, however in combination with vitamin E encouraging results have recently been reported⁸. Statin is also beneficial in patients of NAFLD with dyslipidemia.

Presently there are no established treatment for NAFLD and therefore an emphasis need to be placed on lifestyle advice with weight loss when necessary, increase in physical activity and improved nutrition in order to decrease liver fat accumulation.

REFERENCES

1. Vuppalanchi R, Chalasani N. Nonalcoholic fatty liver disease and nonalcoholic steatohepatitis: Selected practical issues in their evaluation and management. *Hepatology* 2009; 49(1): 306–17.
2. Amarapurkar DN, Hashimoto E, Lesmana LA, Sollano JD, Chen PJ, Goh KL. How common is non-alcoholic fatty liver disease in the Asia-Pacific region and are there local differences? *J Gastroenterol Hepatol* 2007; 22(6): 788–93.
3. Fan JG, Saibara T, Chitturi S, Kim BI, Sung JJ, Chutaputti A: What are the risk factors and settings for non-alcoholic fatty liver disease in Asia-Pacific? *J Gastroenterol Hepatol* 2007; 22(6): 794–800.
4. Day CP. Natural history of NAFLD: remarkably benign in the absence of cirrhosis. *Gastroenterology* 2005; 129(1): 375–8.
5. Ekstedt M, Franzen LE, Mathiesen UL, Thorelius L, Holmqvist M, Bodemar G, et al. Long-term follow-up of patients with NAFLD and elevated liver enzymes. *Hepatology* 2006; 44(4): 865–73
6. Kneeman JM, Misdraji J, Corey KE. Secondary causes of nonalcoholic fatty liver disease. *Therap Adv Gastroenterol* 2012; 5(3): 199–207.
7. Serafinceanu C, Elian V. Nonalcoholic fatty liver disease- How to manage a “new” cardiovascular risk factor? *Rom J Diabetes Nutr Metab Dis* 2012; 19(3): 225-28.
8. de Alwis NMW, Day CP. Non-alcoholic fatty liver disease: The mist gradually clears. *J Hepatol* 2008; 48(Suppl 1): S104-12.
9. Saadeh S, Younossi ZM, Remer EM, Gramlich T, Ong JP, Hurley M, et al. The utility of radiological imaging in non-alcoholic fatty liver disease. *Gastroenterology* 2002; 123(3): 745–50.
10. Cox I, Sharif A, Cobbold J, Thomas H, Taylor-Robinson S. Current and Future applications of in vitro magnetic resonance spectroscopy in hepatobiliary disease. *World J Gastroenterol* 2006; 12(30): 4773–83.
11. Yoneda M, Yoneda M, Fujita K, Inamori M, Tamano M, Hiriishi H, et al. Transient elastography in patients with non-alcoholic fatty liver disease (NAFLD). *Gut* 2007; 56(9): 1330-1.

Md Habibur Rahman

Professor and Head, Department of Gastroenterology;

Sylhet MAG Osmani Medical College, Sylhet-3100.

E-mail: md_habiburrahman@yahoo.com



Original Article

Sonographic Evaluation of Ovaries in Polycystic Ovary Syndrome Presenting with Infertility

Nigar Sultana¹, Sayed Mizanur Rahman², Kona Ghosh³, Md Moinul Hossain⁴, Aurobindo Roy⁵

^{1,3}Assistant Professor, Department of Radiology and Imaging, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Professor, Department of Radiology and Imaging, Bangabandhu Sheikh Mujib Medical University, Dhaka

⁴Radiologist, Department of Radiology and Imaging, Chittagong Medical College Hospital, Chittagong

⁵Consultant Radiologist, Narayanganj General Hospital, Narayanganj

ABSTRACT

This study was carried out to assess the diagnostic value of sonography in the evaluation of ovaries of polycystic ovary syndrome patients presenting with infertility and to find out more accuracy of transvaginal sonography in this respect. This cross sectional study was carried out in clinically suspected 50 patients of polycystic ovary syndrome (PCOS), presenting with infertility in the department of Obstetrics and Gynaecology of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka in collaboration with the department of Radiology and Imaging from 1st July 2008 to 30th June 2009. Fifty patients were selected purposively and transabdominal sonography (TAS) and transvaginal sonography (TVS) were done in each patient by Siemens Sonoline Adara with 3.5 and 7.5 MHz frequency probe. Findings of TVS were then compared with transabdominal ultrasound and finally with the pathological diagnosis, to show the superiority of TVS to give reliable and accurate informations needed in the diagnosis of PCOS. The difference between the sensitivity of the two imaging modalities of diagnosis was not found to be statistically significant ($p > 0.05$) but TVS was found more sensitive and more effective modality in the evaluation of ovaries in PCOS presenting with infertility. The sonographic assessment of the number and size of the follicles are important in PCOS. Follicular imaging obtained by abdominal scanning may be distorted by echoes from the intestine or by unfavorable location of the ovaries and patients must have a full bladder. Transvaginal sonography showed more follicles than transabdominal sonography because of improved imaging of small follicles in early days of female cycle.

Key words: Transvaginal sonography, Polycystic ovary syndrome, Infertility.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the commonest endocrine disorder affecting 4% to 8% women of reproductive age¹. A significant number of patients have infertility as a presenting feature of polycystic ovary syndrome². It is a disorder of unknown etiology characterized by metabolic abnormalities, chronic anovulation, menstrual

disturbances, hyperandrogenism and obesity. In a recent joint European Society of Human Reproduction and Embryology & American Society for Reproductive Medicine consensus meeting, a refined definition of PCOS was agreed. Namely the presence of two out of the following three criteria: a) Oligomenorrhoea and/or anovulation; b) Hyperandrogenism (clinical and/or biochemical); c) Polycystic ovaries with the exclusion of other etiologies³.

PCOS has increased recently with the realization that this syndrome involves far more than the reproductive system. PCOS is now recognized to be a metabolic syndrome, which may include endometrial cancer, hyperinsulinaemia, hyperlipidemia, diabetes mellitus

Address of Correspondence: Dr Nigar Sultana, Assistant Professor, Department of Radiology and Imaging, Jalalabad Ragib-Rabeya Medical College, Sylhet. E-mail: nsnipa2010@gmail.com; Mobile No: 01191825108

and possibly cardiac disease⁴.

Diagnosis is based on clinical symptoms, haematological findings and sonographic appearances. An imbalance of leutinizing hormone (LH) and follicle stimulating hormone (FSH) results in abnormal estrogen and androgen production⁵. Elevated serum LH level, decreased FSH level and an elevated LH:FSH ratio are characteristic findings of polycystic ovarian disease (PCOD). PCOD is a common cause of infertility⁶.

Polycystic ovaries are commonly detected by transvaginal ultrasonogram with estimates of prevalence in the general population being in the order of 20-33 percent⁷. Abdominal ultrasonography can recognize the increase in volume of the ovaries and presence of few cysts, but the detail morphology of the ovary cannot be demonstrated per abdominally as most of the patients are obese. Transvaginal sonography (TVS) with higher frequency (7.5 MHz) can demonstrate the exact morphology of the ovaries. The USG criteria for the diagnosis of polycystic ovaries are increased number of follicles and the amount of stroma as compared with normal ovaries resulting in increased ovarian volume more than 10 cc⁸. To label one as having PCOS, there should be presence of 12 or more follicles in each ovary, diameters ranging from 2-9 mm and increased ovarian volume (>10 cm³).

Tokagashi studied 30 patients with PCOD and compared the sonographic appearance of polycystic ovaries with patients under going wedge resection. The histological appearances coincided with ultrasonogram findings⁹. The advent of TVS has enabled proper diagnosis of cause of infertility because of its higher resolution and clearer view of developing follicles.

Though abdominal ultrasound is widely used in Bangladesh for the evaluation of patients with polycystic ovaries and as volume of polycystic ovaries can be normal in up to one fourth patients, mere appreciation of volume is not enough in the diagnosis of PCOS⁹. With the introduction of transvaginal ultrasonography, sonographic evaluation of the ovary in women of PCOS presenting with infertility has become very quick, simple, painless, inexpensive and accurate.

This study aimed to evaluate TVS as a diagnostic tool for accurate diagnosis of morphological appearances of ovaries in regards of volume, number of follicles, the average diameter of follicles, the distribution of follicles in comparison to abdominal ultrasound in PCOS presenting with infertility.

MATERIALS AND METHODS

This cross sectional study was carried out on 50 patients with suspected PCOS presenting with infertility in the department of Obstetrics and Gynaecology of BSMMU in collaboration with the department of Radiology and Imaging from 1st July 2008 to 30th June 2009. Patients were clinically evaluated by a detailed history with special emphasis on infertility, menstrual and obstetric history. A thorough clinical examination was done and the emphasis was put on the patient's weight, waist-hip ratio and hair distribution. All the data were meticulously recorded. Serum testosterone, LH and FSH level were analyzed in Clinical Pathology department of BSMMU. Transvaginal and transabdominal sonogram were performed with real time scanner by Siemens Sonoline Adara with 7.5 and 3.5 MHz probe and findings were recorded. At first the patient was adequately prepared in lithotomy position and the transvaginal probe was covered with condom. The probe was oriented from side to side (transverse plane) and also rotated. The transducer in 90° directed the sound beam anteriorly and posteriorly to visualize the uterus and ovary. Ovarian follicles were measured in three dimensions.

RESULTS

This study included 50 patients of infertility with clinically suspected PCOS. The age range was 20-36 years with a mean age of 27.74 (± 3.19) years. The commonest age group was 26 to 30 years (20 patients, 40%) followed by 31 to 36 years group (16 patients, 32%). [Table-I].

Table-I: Distribution of the respondents by age (n=50)

Age (in year)	Number	Percent
16 to 20	02	4
21 to 25	12	24
26 to 30	20	40
≥ 31	16	32
Total	50	100

Table-II: Distribution of patient by primary and secondary infertility (n=50)

Fertility	Number	Percent
Primary infertility	30	60
Secondary infertility	13	26
Sub-fertility	07	14
Total	50	100

Out of 50 patients, 30 (60%) had primary infertility, 13 (26%) had secondary infertility and 7 (14%) had sub-fertility (Table-II).

Table-III: Distribution of signs and symptoms in patients of PCOS with infertility (n=50)

Signs and Symptoms	Number	Percent
Menstrual problem	38	76
Obesity	30	60
Hirsutism	26	52
Acne	10	20
Depression	08	16
Pigmentation	06	12

Among 50 patients of PCOS the commonest other symptoms associated with infertility were menstrual problem 38 (76%) followed by hirsutism 26 (52%) and obesity 30 (60%). Acne and pigmentation were complained by 10 (20%) and 6 (12%) respectively (Table-III). In the current study, 60% presented with three sign symptoms, 16% patients with classical clinical picture of Stein Leventhal syndrome, 16%

patients presented with two sign symptoms and 5% patients presented with one sign symptom.

Table-IV: Distribution of LH: FSH ratio (n=50)

LH: FSH ratio	Number	Percent
1.1	6	12
2.1	10	20
3.1	20	40
4.1	14	28

LH: FSH ratio was ≥ 1.1 in 6 (12%) patients, ≥ 2.1 in 10 (20%) patients, ≥ 3.1 in 20 (40%) patients and ≥ 4.1 in 14 (28%) patients (Table-IV).

Serum testosterone was elevated in 36 patients, which represented about 72% of total number of patients. Significantly higher percentage ($p < 0.01$) of women showed increased concentration of serum testosterone level in Z test.

Table-V: Distribution of ovarian volume by transabdominal and transvaginal sonogram (n=50)

Routes	Right Ovary		Left Ovary		Total volume (Mean)
	Mean	Range	Mean	Range	
Transabdominal	10.52 cm ³	6 – 22 cm ³	11.48 cm ³	5 – 28 cm ³	11.19 cm ³
Transvaginal	11.58 cm ³	6 – 24 cm ³	12.66 cm ³	4 – 30 cm ³	12.41 cm ³

p value is > 0.1 , "t" test of differences in means

The mean (SD) volume of the right ovary calculated by transabdominal route was 10.52 (± 4.15) cm³ and range 6-22 cm³. The mean SD volume of left ovary by abdominal sonogram was 11.48 (± 5.98) cm³ and range 5-28 cm³. The total mean (SD) volume calculated by abdominal sonogram was 11.19 (± 2.8) cm³. The mean volume of left ovary obtained by transvaginal sonogram was 12.66 (± 6.68) cm³ and range was 4-30

cm³ and the mean (SD) right ovarian volume by transvaginal sonogram was 11.58 (± 4.47) cm³ and range 6-24 cm³. The total mean (SD) volume of both ovaries by transvaginal sonogram was 12.41 (± 2.8) cm³. There was no statistically significant difference between the mean volume of the ovaries observed on transabdominal and transvaginal ultrasonogram "t" test of differences in means.

Table-VI: Distribution of number of follicles in abdominal and vaginal sonogram (n=50)

Routes	Right Ovary		Left Ovary		Total (Mean)
	Mean	Range	Mean	Range	
Transabdominal	4.5	2 – 7	5.3	1 – 8	5.21
Transvaginal	10.63	8 - 13	12.29	6 - 16	11.75

p value < 0.01 , 't' test of difference of means

Mean number of follicles observed in both ovaries were 5.21 (± 2.4) by transabdominal route and 11.75 (± 1.4) by transvaginal route. There was statistically significant difference between the mean number of follicles in the ovaries observed in transabdominal and transvaginal ultrasonogram.

Of the 50 patients, the pattern of distribution of follicles could not be evaluated in 34 (68%) patients by

transabdominal ultrasonogram. However, 6 (12%) patients had generalized distribution of follicles (Figure 1) and 10 (20%) had peripheral cystic pattern (Figure 2). By transvaginal route, generalized pattern of distribution were observed in 18 (36%) of patients and 26 (56%) patients had peripheral cystic pattern distribution. In 4 (8%) patients unilateral polycystic ovaries were observed.

In 26 patients of the peripheral cystic pattern, LH:FSH was $\geq 3:1$ and 2 patients of peripheral cystic pattern LH:FSH was $<3:1$. In 16 patients of generalized cystic pattern LH:FSH was $\geq 3:1$ and in 6 patients LH:FSH was $<3:1$. $P < 0.01$ in Chi square test with Yates correlation and 1 df, peripheral patterns were significantly higher than generalized pattern in women who had high ($\geq 3:1$) LH:FSH ratio.



Figure-1: TVS showing generalized distribution of follicles in polycystic ovary



Figure-2: TVS showing peripheral distribution of follicles in polycystic ovary

DISCUSSION

Polycystic ovaries are commonly detected by transvaginal ultrasonogram with estimates of prevalence in the general population being in the order of 20 to 33 percent. Symptoms of polycystic ovary syndrome clearly begins after menarche¹⁰. This is clearly reflected in this study as the highest age group of presentation were 26-30 years and 20 (40%) patients were in this age group. The patients presented with a

wide spectrum of clinical features but most of patients seeking medical help for infertility. The percentage observed in this study was close to the observation made by Goldezieher et al. who studied the percentage of various clinical symptoms in 505 surgically proven cases of polycystic ovary syndrome².

The other most common symptom of presentation observed in this study was menstrual irregularity and hirsutism. These were present in 76% and 52% of the patients respectively. These observations were within the range observed by Goldzieher² et al. They reported the range of hirsutism were 50-60%, and menstrual irregularity were 65-77% in their study of 505 cases. Thirty patients (60%) complained of obesity. This observation coincides with the observations of the same study² where they found the incidence of obesity to about 60%.

Only 10 (20%) patients complained of acne and 6 (12.5%) patients complained of increased pigmentation. The values observed by Insler¹¹ were 22 (25%) and 10 (15.4%) for acne and pigmentation among 187 patients. The findings in this study are well within findings of Insler.

Among the fifty patients only 8 (16%) patients presented with amenorrhoea, hirsutism, obesity and infertility. This group of women represented the PCOS, as originally described by Stein and Leventhal³. Conway in his study of 556 patients of polycystic ovary syndrome stated that the phenotypic expression of polycystic ovary syndrome was varied¹². In the current study, 60% presented with three sign symptoms, 16% presented with two sign symptoms and 5% presented with one sign symptom. This study also shows the different spectrum of presentation of the patients of PCOS. Indeed it is in agreement with the new definition of androgen excess, as "a heterogeneous syndrome characterizes by chronic anovulatory cycles with signs of androgen excess", by National Institute of Child Health and Development Consensus Conference¹².

Serum testosterone was elevated in 72% patients in this study. According to Rosenfield¹³ total testosterone level is the best single measure of PCOS with sensitivity of 80% and the LH:FSH value can be normal in up to 50% of patients and 20% patients may have values $>3:1$. In this study the patients with LH:FSH ratio was high in 68% patients, and greater than $>3:1$ in 37% of patients. This could be due to the late presentation of patients with PCOS in Bangladesh. In this study the mean (SD) volume observed by transvaginal ultrasonogram was 12.41 (± 2.4) cm^3 with a range 4-30 cm^3 which was very close to the

observations made by Swanson et al¹⁴. They observed the mean ovarian volume to be 12.5 cm³. Jaffe et al¹⁵ found the mean ovarian volume measured by transvaginal sonogram as 16.88 cm³ and range from 8.30-29.37 cm³. Their findings were slightly higher than the findings of this study. In this study, in 23.8% women of PCOS, the mean volume was within 8 cm³ which is upper limit of normal ovaries according to Dodson¹⁶.

Takahashi et al⁹, assessed the morphological findings of 47 women with PCOS clinically by TVS and found an ovarian volume >8.2 cm³, the number of follicles >10 and a diameter of 2-8 mm as the most prominent features of PCOD and 94% patient had at least one of these features. Their findings were similar to the values observed in this study.

The mean number (SD) of the follicles measured by abdominal route was 5.21 (±2.4) and the mean (SD) number of follicles observed by transvaginal ultrasonogram was 11.75 (±2.6). There was statistically significant difference between the mean number of follicles observed by abdominal and transvaginal route ($p<0.01$). Takahashi⁹ et al found the number of follicles >10 in PCOD patients diagnosed by TVS. The observations made in this study were very close to their observations.

Balen et al found that the follicular margins were sharply defined in 90% studies of TVS compared to 40 percent in transabdominal technique⁷. In the study by Balen, the pattern of distribution of the follicles could not be clearly identified in 70% of the transabdominal scans, 8.3% patients had generalized cystic pattern of distribution and 20.8% were observed to have peripheral cystic pattern of follicles. By transvaginal scan 36% patients were observed to have generalized cystic pattern of distribution (Figure-1) and 56% patients had peripheral cystic type of follicle distribution (Figure-2). The number of indistinguishable follicles was statistically significant ($p<0.01$) than others observed by abdominal sonogram. In this study 8.3% patients had unilateral polycystic ovary. This finding is consistent with the findings of Jaffe et al, who found unilateral polycystic ovaries in small number of patients.

In 26 patients of the peripheral cystic pattern LH:FSH ratio was $\geq 3:1$ and 16 patients of generalized cystic pattern LH:FSH was $\geq 3:1$. In statistically significant ($p<0.01$) number of women, the peripheral cystic distribution of follicles were associated with LH:FSH ratio $>3:1$. These observations are similar to the observations made by Takahashi et al⁹, where LH:FSH was significantly higher in peripheral cystic pattern

than generalized cystic pattern.

CONCLUSION

PCOS is now one of the leading causes of infertility. Diagnosis is based on clinical symptoms, laboratory findings and ultrasonographic appearances. Transvaginal ultrasonography can be accepted as a useful modality in the evaluation of ovaries in PCOS patients presenting with infertility. It is a non-invasive and cost effective modality and more informative in comparison to transabdominal ultrasonography.

REFERENCES

- Ehrmann DA, Barnes RB, Rosenfield RL. Hyperandrogenism, Hirsutism and the polycystic ovary syndrome. In: DeGroot LJ, editor, Endocrinology Volume III. 3rd ed. Philadelphia: WB Saunders; 1995. pp.2093-112.
- Goldzieher JW, Axelrod LR. Clinical and biochemical features of polycystic ovarian disease. *Fertile Steril* 1963; 14: 631-53.
- Stein IF, Leventhal ML. Amenorrhoea associated with bilateral polycystic ovaries. *Am J Obstet Gynecol* 1935; 129: 181-91.
- Kumar P, Malhotra N. Jeffercoat's Principles of Gynaecology. 7th ed. New Delhi: Jaypee Brothers Medical Publishers; 2008.
- Vutyavanich T, Khanaiyao V, Wongtra-Ngan S, Sreshthaputra O, Sreshthaputra R, Piromlertamorn W. Clinical, endocrine and ultrasonographic features of polycystic ovary syndrome in Thai women. *J Obstet Gynaecol Res* 2007; 33(5): 677-80.
- Norman RJ, Wu R, Stankiewicz MT. 4: Polycystic ovary syndrome. *MJA* 2004; 180(3): 132-7.
- Balen AH, Tan SI, Jacobs HS. Hyper secretion of luteinising hormone: A significant cause of infertility and miscarriage. *Br J Obstet Gynaecol* 1993; 100(12): 1082-9.
- Rumack CM, Wilson SR, Charbonneau JW, Johnson JAM, editors. Diagnostic ultrasound Volume I. 3rd ed. Philadelphia: Elsevier Mosby; 2005.
- Takahashi K, Okada M, Ozaki T, Uchida A, Yamasaki H, Kitao M. Transvaginal ultrasonographic morphology in polycystic ovarian syndrome. *Gynecol Obstet Invest* 1995; 39(3): 201-6.
- Pache TD, Waldimiroff JW, Hop WCJ, Fauser BM. 1992: How to discriminate between normal and polycystic Ovaries. A transvaginal ultrasound study. *Radiology* 1992; 183(2): 421-3.

11. Insler V, Lunenfeld B. Polycystic ovarian disease. A challenge and controversy. *Gynecol Endocrinol* 1990; 4(1): 51-70.
12. Conway GS, Honour JW, Jacobs HS. Heterogeneity of polycystic ovarian syndrome: Clinical endocrine and ultrasound features in 556 patients. *Clin Endocrinol* 1989; 30(4): 459-70.
13. Rosenfield RL. Ovarian and adrenal function in polycystic ovary syndrome. *Endocrinol Metab Clin North Am* 1999; 28(2): 265-93.
14. Swanson M, Sauerbrei EE, Coopberg PL. Medical implications of ultrasonographically detected polycystic ovaries. *J Clin Ultrasound* 1981; 9(5): 219-22.
15. Jaffe R, Abramowicz J, Eckstein N, Vagman I, Fejgin M, Ayalon D. Sonographic monitoring of ovarian volume in LHRH analog therapy in women of polycystic ovary syndrome. *J Ultrasound Med* 1988; 7(4): 203-6.
16. Dodson MG, editor. *Transvaginal Ultrasonogram*. 2nd ed. New York: Churchill Livingstone; 1995.



Original Article

Rural-Urban Gap in Stroke Incidence and Its Diurnal Variation

Md Abdul Quddus¹, Prodyot Kumar Bhattacharyya², ATMA Jalil³, Ayesha Rafiq Chowdhury⁴, Nurul Hasan Siddiquee⁵, Atanu Bhattacharjee⁶, Almahir Ferdous⁷, Ali Ahsan⁸

^{1,4}Associate Professor, Department of Medicine, Jalalabad Ragib-Rabeya Medical College, Sylhet

^{2,3}Professor, Department of Medicine, Jalalabad Ragib-Rabeya Medical College, Sylhet

^{5,6}Asst. Registrar, Department of Medicine, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

⁷Registrar, Department of Orthopedics, North East Medical College Hospital, Sylhet

⁸Indoor Medical Officer, Department of Medicine, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

ABSTRACT

This prospective study was done in the Department of Medicine, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet from February, 2010 to January, 2011. Objective of the study was to find out geographical distribution and diurnal variation of stroke patients. Total 104 patients were included in the study. Patients were divided into two groups, infarction type (Group-I) and haemorrhagic type (Group-II). It was observed that stroke incidence was more in rural community (59.6%). It was also observed that stroke in Group-I patients were more at the early hours of the day (56.4%) and that in Group-II patients were more at the late hours of the day (61.5%).

Key words: Stroke, Place and time of occurrence.

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INTRODUCTION

Cerebrovascular diseases (CVD) include some of the most common and devastating disorders: ischaemic stroke, haemorrhagic stroke and cerebrovascular anomalies such as intracranial aneurysms and arteriovenous malformations (AVMs). Stroke causes approximately 200,000 deaths each year in the United States and is a major cause of disability¹. Stroke is the third commonest cause of death in developed countries after ischaemic heart disease and malignant diseases. It predominates in middle and late years of life. At least 50 percent of neurologic disorders in general populations are of this type². About 50 percent of admitted patients in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BMMU), Dhaka, are stroke patients. About 70% of deaths among hospital patients are due to stroke³. Various studies were done on geographical distribution of stroke patients⁴⁻⁸. Some studies were done on diurnal variation of stroke^{9,10}. We decided to do this

study in a tertiary care hospital to observe these patterns in Sylhet region.

MATERIALS AND METHODS

This randomized prospective study was done on stroke patients of both sexes admitted in Medicine ward, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet from February 2010 to January 2011. A total number of 104 stroke patients were included. The patients with history of recurrent stroke were excluded from this study. Age of the patients ranged from 35 to 100 years (mean age 62.96±13.2 years). Male and female ratio was 1.74:1 (male 66, female 38).

After admission, all patients were evaluated by history and physical examination. In the history patients were specially asked whether they were hypertensive, diabetic, smokers and about other relevant risk factors. They were also evaluated for any family history of hypertension, diabetes mellitus, ischaemic heart disease or stroke. In general examination, special attention was paid on pulse, BP along with other parameters. Neurological and cardiovascular examinations were done completely. In neurological examination level of consciousness, motor, reflexes,

Address of Correspondence: Dr MA Quddus, Associate Professor, Department of Medicine, Jalalabad Ragib-Rabeya Medical College, Sylhet. Mobile:01715294592.

sensory, cerebellar functions, cranial nerve functions were evaluated. In cardiovascular examination it was observed whether they had any arrhythmia, hypertension, evidence of valvular heart disease or carotid bruit. CT scan of brain, ECG, X-ray chest, fasting blood sugar, fasting lipid profile, serum creatinine, complete blood count and urine for routine examination were carried out in every patient.

The patients were categorized into two groups on the basis of CT scan, group-I infarction type and group-II haemorrhagic type. The patients were evaluated to find out rural-urban gap in stroke incidence and its diurnal variation in these two groups.

All data were collected and recorded in pre-designed data collection sheet. After entry into computer, chi-square test was followed with the help of SPSS programme.

RESULTS

A total of 104 patients were included in this study and the patients were categorized into two groups on the basis of CT scan. The patients with infarct were considered as group-I and with haemorrhage considered as group-II patients. Out of 104 patients, 78

(75%) patients had cerebral infarct and 26 (25%) had cerebral haemorrhage. The mean age of the patients was 62.96 ± 13.2 years ranging from 35 to 100 years. Male and female ratio was 1.74:1 (male 66, female 38). Main risk factors for stroke were smoking and/or tobacco consumption (71.2%), hypertension (63.5%) and diabetes mellitus (35.9%).

Analysis of rural-urban gap in stroke patients showed 62 patients were rural and 42 patients were urban (Table-I).

Table-I: Distribution of patients according to place.

Group	Rural	Urban	Total	P-Value
Group-I	44	34	78	
Group-II	18	8	26	0.024
Total	62	42	104	

P-Value reached from Pearson chi-square test. Rural urban difference was significant ($p < 0.05$).

Analysis of the diurnal variation showed that in group-I, 44 patients and in group-II, 10 patients stroke occurred at early hours of the day (AM) and 34 patients in group-I and 16 patients in group-II stroke occurred at late hours of the day (PM) (Table-II).

Table-II: Distribution of patients by time of occurrence.

Occurrence	Group-I		Group-II		Total		P-Value
	No (n=78)	Percent	No (n=26)	Percent	No (n=104)	Percent	
AM	44	56.4	10	38.5	54	51.9	0.03
PM	34	43.6	16	61.5	50	48.1	0.05

P value reached from Pearson chi-square test. It was found that occurrence of stroke in infarction type group-I was higher in the morning than evening, the difference was significant ($p < 0.05$) and stroke were more at evening than morning in haemorrhagic type group-II. Difference was just significant ($p = 0.05$).

DISCUSSION

The study was done to find out rural-urban gap and diurnal variation of stroke. In our study it was found that stroke were higher in rural than urban area. Difference was significant ($p < 0.05$). Bejot Y et al in their study in Europe found more stroke in rural community⁸. Pearson et al found in their study more stroke in American rural people¹¹. Adams PF et al found in their study more strokes in rural people in USA¹². Zhang XH et al found in their study in China more strokes in rural area¹³. Correia M et al found in Portugal more strokes in rural residents¹⁴. Kitamura A et al found in their study in Japan more stroke incidence in rural community¹⁵. On the other hand in a

study by Motiur Rahman et al on 105 stroke patients in Sylhet MAG Osmani Medical College Hospital found 61.9% cases were from urban areas and 38.1% from rural areas¹⁶. In our country and also in other countries rural stroke incidence rate is higher likely due to lack of medical awareness regarding risk factors of stroke (smoking, hypertension, diabetes mellitus), less access to specialized treatment, inadequate treatment of hypertension, diabetes mellitus etc. in rural people^{17,18}. In our study it was found that occurrence of stroke in infarction type group-I was higher in the morning than evening, the difference was significant ($p < 0.05$). It was found that stroke were more in the evening than morning in haemorrhagic type group-II. Difference was just significant ($p = 0.05$). Tsementzis SA et al found in their study on 557 patients infarction type is more during sleep and early morning and haemorrhagic stroke is more at the late hours of the day⁹. Inagawa T et al in a study on 350 patients in Japan found infarction type more during sleep and early morning and haemorrhagic stroke more at the late hours of the

day¹⁰. Rahman et al in their study on 105 stroke patients found 47.62% in morning, 38.09% in day time and 14.29% in the evening¹⁶. This diurnal variation is likely, due to in lying posture more chance of thromboembolism due to slow blood flow in group-I and variation of blood pressure (more rise of blood pressure in the evening) due to day time activities leads to intracerebral haemorrhage in group-II patients. Other study by Tsementzis et al also support this finding⁹.

CONCLUSION

Stroke is the commonest neurological disorder and is more prevalent in rural areas. It was also found that infarction type stroke more at early hours of the day and haemorrhagic type stroke more at late hours of the day. As the patients were selected from Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet and patients of Sylhet division came to this hospital, it more or less represents place and time of occurrence of stroke in this part of the country. After stroke, most patients lead crippled life. So it is important to prevent stroke by creating awareness among rural and urban people to modify life style, change in dietary habit, avoid smoking, proper and timely treatment of hypertension and diabetes mellitus, as these are usually related to development of stroke.

REFERENCES

- Smith WS, English JD, Johnston SC. Cerebrovascular diseases. In: Fauci AS, Kasper DL, Longo DL, Brandwall E, Hauser SL, Jameson JL, editors. *Harrison's Principles of Internal Medicine Volume II*. 17th edition. New York: McGrawHill Company; 2008. pp. 2513-4.
- Adams RD, Victor M, Rooper AH, editors. *Principles of Neurology*. 6th ed. New York: McGraw-Hill Companies Inc; 1997.
- Haque A, Ullah AKMA, Kabir I, Khah MRK. Morbidity and mortality pattern among the hospitalized patients in the department of Neurology, IPGMR, Dhaka: a one-year study, *Bangladesh J Neuro Sci* 1995; 11:1-5.
- Japan Health and Welfare Statistics Association. *Trends of Nation's health*. *J Health & Welfare Statistics* 2007; 54: 412-3.
- Gillum RF, Ingram DD. Relation between residence in the southeast region of the United States and stroke incidence. The NHANES I Epidemiologic Follow Up Study. *Am J Epidemiol* 1996; 144(7): 665-73.
- Morris RW, Whincup PH, Emberson JR, Lampe FC, Walker M, Shaper AG. North-south gradients in Britain for stroke and CHD: Are they explained by

the same factors? *Stroke* 2003; 34(11): 2604-9.

- Havulinna AS, Paakkonen R, Karvonen M, Salomaa V. Geographic patterns of incidence of ischemic stroke and acute myocardial infarction in Finland during 1991-2003. *Ann Epidemiol* 2008; 18(3): 206-13.
- Bejot Y, Benatru I, Rouaud O, Fromont A, Besancenot JP, Moreau T, et al. Epidemiology of stroke in Europe: Geographic and environmental differences. *Journal Neurol Sci* 2007; 262(1): 85-8.
- Tsementzis SA, Gill JS, Hitchcock ER, Gill SK, Beevers DG. Diurnal variation of and activity during the onset of stroke. *Neurosurgery* 1985; 17 (6): 901-4.
- Inagawa T. Diurnal and seasonal variations in the onset of primary intracerebral hemorrhage in individuals living in Izumo City, Japan. *J Neurosur* 2003; 98 (2): 326-36.
- Pearson TA, Lewis C. Rural epidemiology: insights from a rural population laboratory. *Am J Epidemiol* 1998; 148(10): 949- 57.
- Adams PF, Hendershot GE, Marano MA. Current estimates from the National Health Interview Survey, 1996. *Vital Health Stat* 10. 1999; 200:1-203.
- Zhang XH, Guan T, Mao J, Liu L. Disparity and its time trends in stroke mortality between urban and rural populations in China 1987 to 2001: changing patterns and their implications for public health policy. *Stroke* 2007; 38(12): 3139-44.
- Correia M, Silva MR, Matos I, Magalhaes R, Lopes JC, Ferro JM, et al. Prospective community-based study of stroke in Northern Portugal: incidence and case fatality in rural and urban populations. *Stroke* 2004; 35(9): 2048-53.
- Kitamura A, Sato, Kiyama M, Imano H, Iso H, Okado T, et al. Trends in incidence of coronary heart disease and stroke and their risk factors in Japan, 1964 to 2003: The Akita-Osaka study. *J Am Coll Cardiol* 2008; 52(1): 71-9.
- Rahman MM, Mosharraf AKM, Patwary MIH. Risk factors for stroke- A clinical study. *Bangladesh J Medicine* 2000; 11(2): 90-4.
- Joubert J, Prentice LF, Moulin T, Liaw ST, Joubert LB, Preux PM, et al. Stroke in rural areas and small communities. *Stroke* 2008; 39(6):1920-8.
- Leira EC, Hess DC, Torner JC, Adams HP Jr. Rural-urban differences in acute stroke management practices: a modifiable disparity. *Arch Neurol* 2008; 65(7):887-91



Original Article

An Autopsy Study of 357 Cases of Organophosphorous Compound Poisoning

MA Salam¹, Md Abdul Hye², AA Adiluzzaman³, Md Abul Mansur⁴

¹Assistant Professor, Department of Forensic Medicine, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Lecturer, Department of Forensic Medicine, Sylhet MAG Osmani Medical College, Sylhet

³Assistant Professor, Department of Forensic Medicine, Shere-E-Bangla Medical College, Barishal

⁴Assistant Professor, Department of Forensic Medicine, Sylhet MAG Osmani Medical College, Sylhet

ABSTRACT

This retrospective study was performed to find out the age and sex incidence as well as the nature of death in case of OPC (organophosphorus compound) poisoning. The study was carried out among 357 victims of OPC poisoning whose autopsy were done in the Department of Forensic Medicine, Sylhet MAG Osmani Medical College, Sylhet from January 2007 to December 2010. There were 119 (33.33%) male and 238 (66.66%) female and the male-female ratio was 1:2. Of 119 males victims the highest 33.33% were in age group of 21-30 years followed by 21.85% in age group of 11-20 years, the least affected age groups were > 60 (00%) years and 51-60 (2.52%) years respectively. Among the female victims, the highest 45.38% were in age group of 21-30 years followed by 27.31% in age group of 11-20 years. Here the least affected age groups were in 51-60 (0.42%) years and 0-10 (3.36%) years. The causes of death were asphyxia. The nature of death were mainly suicide (96.64%) followed by accident (3.08%). Only 1 case (0.28%) was homicidal in nature. In suicidal case, male-female ratio was 1:2 (male 33.33% and female 66.66%) which revealed that the incidence of suicide in female was more than that of male. Accidental cases were more in male than female. Homicidal case was only 1.

Key words: Organophosphorous compound, Homicide, Suicide, Acetylcholinesterase.

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INTRODUCTION

In the last half century there has been a technological revolution in farming methods, which has been spread to almost all parts of the world. The technology besides its usefulness created new problems for users which are accidental or intentional poisoning by any one of the compounds. The developing nations (who in terms of area of land usage and proportion of the population, who work on that land, are numerically greatest at risk) suffer most and this is sometimes exacerbated by less stringent controls and less safe working methods that exist in those countries. In this context lack of proper usage and preservation of agrochemical substances may cause social problems like homicidal, suicidal and

accidental casualties.

The potent chemicals used in agriculture may harm persons by accidental exposure, either during their application to crops, or due to incorrect or careless storage. The major source of human poisoning by these chemicals is through self administration, when the easily available substances are used for suicide¹. In Sri Lanka many thousands of hospital admissions each year are for agrochemical poisoning with over a thousand deaths annually. Of these, about-three quarters were self-administered, the remainder being accidental and occupational².

The commonly used agricultural chemicals are the organophosphorus compounds, viz, alkyl and aryl phosphate³, the chlorinated hydrocarbons, the carbamates, the coal-tar products, a few metallic compounds such as zinc phosphide, aluminum phosphide etc. Organophosphorus compounds were initially developed as chemical warfare agent, e.g.

Address of Correspondence: Dr MA Salam, Assistant Professor, Department of Forensic Medicine, Jalalabad Ragib-Rabeya Medical College, Sylhet. Mobile: 01712-140717

sarin. Although they have numerous complex actions, their principal effect is inhibition of cholinesterase enzymes, particularly acetyl cholinesterase (AChE). This leads to accumulation of acetylcholine at masticatory receptors (cholinergic effector cell), nicotinic receptors (skeletal neuromuscular junctions and autonomic ganglia), and in the CNS³. Acetyl cholinesterase is important in the maintenance of impulse transfer between nerves and muscle cells. Organic phosphate inhibits AChE in all parts of the body, due to which acetylcholine accumulates at the parasympathetic, sympathetic and somatic sites, and transfer of nerve impulse across the synapse at the autonomic ganglia and at the neuromuscular junction is prevented⁴.

The inactivation of cholinesterase enzyme becomes irreversible after 24 to 36 hours⁴. Symptoms appear in both the sympathetic and parasympathetic nervous system as well as in the somatic system. Mild poisoning usually occurs when cholinesterase activity is 20 to 50% of normal; moderate poisoning occurs when activity is 10 to 20% of normal, and severe poisoning when activity is less than 10% of normal⁴. Fatalities begin to occur after ingestion of 125-175 mg, though ingestion of much larger amount can survive. Death can occur in less than an hour after ingestion, though usually several hours elapse in those who are not going to survive¹. Cause of death is asphyxia resulting from paralysis of respiratory muscles, respiratory arrest due to failure of respiratory centre, or intense bronchoconstriction⁴. Clinical features can be minimized by use of antidotes like pralidoxime, diacetyl monoxime (DAM) etc.

MATERIALS AND METHODS

This autopsy study was carried out among 357 victims of OPC poisoning in the morgue of the Department of Forensic Medicine, Sylhet MAG Osmani Medical College, Sylhet from January 2007 to December 2010. The data were collected from the inquest reports, challans, and post mortem reports. The chemical analysis reports of preserved viscera furnished by the chief chemical examiner, Mohakhali, Dhaka were also collected. The viscera preserved for chemical analysis were stomach with its contents and the proximal 50 cm of small intestine, portion of liver (500gm), and half of each kidney. Preservatives used were rectified spirit/saturated solution of sodium chloride with all precautions.

RESULTS

Table-I reveals that, out of 357 autopsy cases, 119

(33.33%) were male and 238 (66.66%) were female victims, and the male-female ratio was 1:2. It also reveals that among 119 male victims, the highest incidence of OPC poisoning were in the age group of 21-30 years, and among female the highest incidence were also in the age group of 21-30 years. The least incidence in both sexes was in the age group of 51-60 years and above.

Table-I: Age group and sex of the victims (n=357)

Age distribution	Male	Female	Total
0-10 years	11	08	19
11-20 years	26	65	91
21-30 years	40	108	148
31-40 years	23	37	60
41-50 years	16	10	26
51-60 years	03	01	04
Above 60 years	00	09	09
Total	119(33.33%)	238(66.66%)	357(100%)

Male female Ratio=1:2

Table-II shows the male-female distribution in the context of the nature of death. It reveals that out of 357 cases, only 1 (0.28%) was homicidal in nature and it was in the male group; 345 were suicidal (96.64%) of whom 111 (32.17%) were male and 234 (67.83%) were female victims, the ratio of which was 1:2.11. Out of 357 deaths only 11 (3.08%) were accidental in nature, of whom 7 were male and 4 were female.

Table-II: Nature of death (n=357)

Nature of death	Male	Female	Total	Percent
Homicide	01	00	01	0.28%
Suicide	111	234	345	96.64%
Accident	07	04	11	3.08%
Total	119	238	357	100%

Table-III: Distribution of suicidal death according to age (n=345)

Age distribution	Male	Female	Total
0-10 years	06	05	11
11-20 years	24	65	89
21-30 years	40	108	148
31-40 years	22	37	59
41-50 years	16	10	26
51-60 years	03	01	04
Above 60 years	00	08	08
Total	111	234	345

Male female ratio in suicide 1:2.11

Table-IV: Distribution of accidental death according to age (n=11)

Age distribution	Male	Female	Total
0-10 years	05	03	08
11-20 years	02	00	02
21-30 years	00	00	00
31-40 years	00	00	00
41-50 years	00	00	00
51-60 years	00	00	00
Above 60 years	00	01	01
Total	07	04	11

Table-V: Distribution of homicidal death according to age (n=01)

Age distribution	Male	Female	Total
0-10 years	00	00	00
11-20 years	00	00	00
21-30 years	00	00	00
31-40 years	01	00	01
41-50 years	00	00	00
51-60 years	00	00	00
Above 60 years	00	00	00
Total	01	00	01

Table III, IV and V showing death due to suicide, accident and homicide respectively according to the age group and sex of the victims.

DISCUSSION

Among the agrochemicals, the esters of phosphoric acid⁴ i.e. organophosphorous compounds are important because they are used in vast quantities in most parts of the world. They are particularly common and therefore particularly dangerous in rural and agricultural communities. World wide, there are million of admissions to hospitals and more than 250000 deaths each year as a result of ingestion of agrochemicals⁵. Parathion is the most toxic organophosphorous compound, though others, such as malathion, dithion demeton, dimefox, paraoxon, chlorhion, methyl parathion, diazinon are also poisonous. They are absorbed by inhalation, through the skin, mucous membrane and the gastrointestinal tract. Metabolism occurs in the liver. Detoxification occurs via cytochrome P₄₅₀ monooxygenase. Excretion of metabolites occur in the urine⁴.

Richard Shepherd mentioned that most cases of OPC poisoning are the result of either accidental or environmental exposure⁵. Suicide is second in position and least is homicide. But our study revealed that the highest incidence of OPC poisoning were suicidal in

nature and it was 96.64% of total cases. Accidental causes were only 3.08%. The reduction of incidence of accidental poisoning may be due to awareness about cautious and careful handling of agrochemical agents by the users. On the other hand much increased incidence of suicidal poisoning is due to the ease with which OPC can be obtained. Besides this, as a developing nation familial disharmony, financial constraints, frustration, over burden of children, dowry system, sometimes failure of love provoke suicidal destruction of self, especially the female by taking OPC which is very much available in every parts of the country. The study also revealed that the vast majority of victims were of tender age. At that age every individual may more or less be emotional and they can not understand the consequence of taking OPC by ingestion. On the other hand, poison is usually present in their house for agricultural purpose which the victim can easily avail of, and these cause the increased incidence of suicidal poisoning among the young adults in our country.

CONCLUSION

As OPC poisoning is hazardous and harmful to social health, its incidence should be reduced to a remarkable level to attain a healthy society. Workers engaged in spraying OPC in the agricultural fields, packers and manufacturers are at high risk of accidental poisoning. So adequate precautionary knowledge should be given to the people who are engaged in those works. As this compound has specific antidote other than atropine, it is strongly recommended that it should be made available at district level hospitals for proper use at the earliest. OPC should be strictly preserved at home so that it could not be found easily by the children or any susceptible person who may misuse it.

REFERENCES

1. Saukko P, Knight B. Knight's Forensic Pathology, 3rd ed. Hachette Liver UK; 2004. pp.566
2. Parikh CK. Parikh's Text Book of Medical Jurisprudence, Forensic Medicine and Toxicology. 6th ed. Delhi: CBS; 2002. pp.10.41
3. Haslett C, Chilvers ER, Boon NA, Colledge NR, editors. Davidson's Principles and Practice of Medicine. 19th ed. London: Churchill Livingstone; 2002. pp.1122
4. Reddy KSN. The Essentials of Forensic Medicine and Toxicology, 24th ed. Hyderabad: Om Sai Graphics; 2005. pp.428
5. Shepherd R. Simpson's Forensic Medicine, 12th ed. London: Arnold; 2003. pp.179



Original Article

Postoperative Wound Infections in Surgical Practice

Nurul Quayum Mohammad Musallin¹, Md Nazmul Islam², Shajia Chowdhury³, Parveen Aktar⁴, Angel Shubhagata Baidya⁵, Apurba Kishore Paul⁶, Abdul Mukaddem Mashud⁷

^{1,4,5}Assistant Professor, Department of Surgery, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Professor, Department of Surgery, Jalalabad Ragib-Rabeya Medical College, Sylhet

³Assistant Professor, Department of Pathology, Jalalabad Ragib-Rabeya Medical College, Sylhet

⁶Registrar, Department of Surgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

⁷Assistant Registrar, Department of Surgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

ABSTRACT

This study was conducted in the Department of Surgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet. Total 50 cases of wound infection that developed during January 2010 to July 2011 in different units of Surgery were included. Along with clinical findings, routine hematological investigations, culture and sensitivity of discharges were done in all cases. Out of 50 patients with wound infections 23 (46%) were male and 27 (54%) were female. Most of the infections belonged to the age group of 50-60 years. Nineteen (38%) infections developed in elective surgery in contrast to 31 (62%) cases in emergency surgery. Wound infection developed in 29 (58%) cases with dirty wound, 12 (24%) from contaminated, 7 (14%) from clean-contaminated and 2 (4%) from clean wounds. Anaemia, diabetes mellitus, obesity, smoking and prolonged surgery were common risk factors. The usual time of presentation was within three weeks following surgery and most patients presented with abscess and cellulites. The common organisms were E. coli, others were Staph aureus, Pseudomonas, Klebsiella and Proteus. Result of this study was mostly consistent with other published literatures.

Key words: Wound infection, Surgical site infection.

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INTRODUCTION

Post operative wound infection; now a days called surgical site infection (SSI), results from bacterial contamination during or after a surgical procedure. For this to happen, a sufficient number of pathogens must enter the tissue, overcome the host resistance and multiply¹. Wound infection is the commonest and most troublesome disorder of wound healing².

The purpose of this study was to evaluate the frequency of different types of wound infections, clinical presentation, common risk factors and different organisms involved in postoperative wound infections. An important requirement in the prevention of SSI is the availability of correct and recent data i.e. surgical

audit and wound surveillance. Unfortunately there is deficiency of this topic in our local literature, and often we have to refer to the Western studies, though the nutritional status of their patients, operation theatre facilities, nursing care and management of wound infection are entirely different there.

Infection in a wound is a manifestation of disturbed host-bacteria equilibrium that favors bacterial growth. This not only elicits a systemic septic response but also inhibit the multiple processes that are involved in the wound healing i.e. each of these processes is affected when bacteria proliferate in a wound².

Microorganisms are normally prevented from causing infection in tissues by intact epithelial surface. In addition to this mechanical barrier, there are other protective mechanisms, such as, low gastric pH, antibodies, complements. All of these natural mechanisms may be compromised by surgical intervention and treatment. Reduction of resistance to infection has several causes, such as: metabolic

Address of Correspondence: Dr Nurul Quayum Mohammad Musallin, Assistant Professor, Department of Surgery, Jalalabad Ragib-Rabeya Medical College, Sylhet. Mobile: 01711-735766; E-mail: mdmusallin@gmail.com

(malnutrition, diabetes, uraemia, jaundice) disseminated disease (cancer, AIDS), smoking, and iatrogenic (radiotherapy, chemotherapy, steroids etc)². Major SSI is defined as a wound that discharges significant quantities of pus spontaneously or needs a secondary procedure to drain it. The patient may have systemic signs such as tachycardia, pyrexia and a raised white cell count. Minor wound infections may discharge pus or infected serous fluid but is not associated with excessive discomfort, systemic signs or delay in return home³.

Microbiological factors that influence the establishment of wound infection are the bacterial inoculums, virulence, and the effect of the microenvironment. The usual pathogen on skin surface is gram positive cocci (notably *staphylococci*); however gram-negative aerobes and anaerobic bacteria contaminate skin in the groin/perineal areas. The contaminating pathogens in gastrointestinal surgery are the multitude of intrinsic flora, which includes gram-negative bacilli (eg. *Escherichia coli*) and gram positive microbes, including *Enterococci* and anaerobic organisms⁵.

Wound infections usually appear between the fifth and tenth day after surgery, but they may appear as early as the first postoperative day or even years later. The first sign is usually fever, and post operative fever may require inspection of the wound. The patient may complain of pain at the surgical site. The wound rarely appears severely inflamed, but oedema may be obvious as the skin sutures appear tight¹.

The absolute prevention of surgical wound infection seems to be an impossible goal⁶. It is the second commonest nosocomial infection that causes patient discomfort, prolongs hospital stay, more days off work and increases cost of therapy⁷.

MATERIALS AND METHODS

A total 50 cases of surgical wound infection developed after different elective and emergency surgical procedure, e.g. cholecystectomy, choledocolithotomy, appendectomy, repaired duodenal ulcer perforation, resection anastomosis of small gut, pyelolithotomy, nephrolithotomy, retropubic prostatectomy and suprapubic cystolithotomy were included in this study. Wound infection after laparoscopic, anal and perianal surgery were excluded in this study.

All patients of wound infections were classified as different age group and sex. Then they were labeled as elective and emergency procedure group and classified according to types of surgical wound. All risk factors were identified and tabulated. Discharges from infected wound were collected and sent for culture and

sensitivity test. The results of culture and sensitivity and routine hematological tests were collected and tabulated. Statistical data were analyzed by using SPSS 17.

RESULTS

In this study 50 cases of postoperative wound infection were included, of which 32 (64%) developed minor wound infection and 18 (36%) developed major wound infection. The highest numbers of patients were seen in 5th decade followed by 4th decade. Female had slightly higher infection rate than male. The results are summarized in Table-I.

Out of 50 cases of wound infection 19 (38%) developed after elective surgery and 31 (62%) after emergency surgery. Two (4%) infections occurred in clean wound, 7 (14%) in clean contaminated wound, 22 (44%) in contaminated wound and remaining 19 (38%) infections developed in dirty infected wound. The results are summarized in Table-II.

Surgical wound infections were more common in patient that had low hemoglobin level pre-operatively, 26 (52%) wound infections were related to anaemia. Other factors which were found to be related to wound infection were malnutrition 22 (44%), diabetes mellitus 19 (36%), jaundice 15 (30%), smoking 10 (20%) and obesity 9 (18%).

Most of the wound infections presented as simple wound abscess which was in 27 (54%) cases. Localized cellulites found in 13 (26%) cases, spreading cellulites in 4 (8%) cases and wound dehiscence developed in 6 (12%) cases.

Thirteen (26%) cases of surgical wound infection had preoperative haemoglobin level 9-10 gm/dl, 5 (10%) and 7 (14%) had haemoglobin level <8 gm/dl and 8-9 gm/dl respectively.

Pus or swab culture showed *E. coli* infection in 18 (36%) cases, *Staph. aureus* 11 (22%), *Pseudomonas* 4 (8%) cases. Thirteen (26%) cases shown no growth. All the isolates showed resistance to cloxacillin, amoxicillin and cephradine.

Table-I: Showing distribution of wound infection according to age and sex

Age Group	Male	Female	Total	Percent
0-10 years	0	1	1	2
11-20 years	1	1	2	4
21-30 years	1	2	3	6
31-40 years	3	3	6	12
41-50 years	6	7	13	26
51-60 years	8	10	18	36
60+ years	4	3	7	14
Total	23	27	50	100

Table-II: Showing distribution of wound infection according to types of surgery and nature of wound

Nature of wound	Elective surgery	Emergency surgery	Total	Percent
Clean	0	2	2	4
Clean- contaminated	3	4	7	14
Contaminated	10	12	22	44
Dirty	6	13	19	38
Total	19	31	50	100

Table-III: Showing distribution of culture organism in wound infection

Name of Organism	No (%)
<i>E. coli</i>	18 (36%)
<i>Staph. aureus</i>	11 (22%)
<i>Pseudomonas</i>	04 (8%)
<i>Klebsiela</i>	03 (6%)
<i>Proteus</i>	01 (2%)
No Growth	13 (26%)
Total	50 (100%)

DISCUSSION

Despite advances in the operative techniques and better understanding of the pathogenesis of wound infection, the postoperative wound infection continues to be a major source of morbidity for patient undergoing operative procedure. Patients with age of 50 years and above had a higher incidence of (36%) of postoperative wound infection in this study as compared to 2% in patients having age less than 10 years (Table-I). Ahmed described the incidence of surgical wound infection as 25% in 5th decade⁸.

In this series, wound infections occurred in 19 (38%) cases after elective surgery and 31 (62%) cases after emergency surgery. Sorensen LT et al. reported that in emergency operative procedures surgical wound infections occurred about 2.5 times more than that of elective procedures⁹.

In this study the common risk factors for development of surgical wound infections were anaemia, malnutrition, diabetes, operation in contaminated and dirty field, obesity and smoking. Singhal et al. showed the common risk factors are malnutrition, obesity, diabetes mellitus, steroids etc. which correlates to this study⁵. Anaemia itself is not an established factor for postoperative wound infection. However, a higher incidence of wound infection was noted with initial low haemoglobin level. It can be due to the effect of blood transfusion, which was given preoperatively to bring the haemoglobin level up to 10 mg/dl. Ford et al. postulated this in 1993¹⁰.

Diabetes mellitus was related to 19 (38%) cases of wound infection in this study. This is significantly

higher, though blood sugar level was controlled preoperatively by giving insulin. Ahmed et al⁸ showed the rate was 33.33%. The increased susceptibility to infection in diabetes is an established risk factor¹¹.

Increased rate of surgical site infection (20%) was also noted amongst smokers. Several studies have shown a higher incidence of wound infection amongst smokers than non-smokers. In Ahmed et al⁸ study the rate was 25%.

The commonest presentation in this study was wound abscess 27 (54%). Localized cellulites was found in 13 (26%) cases, 4 (8%) cases had spreading cellulites and 6 (12%) cases were wound dehiscence. No cases of septicaemia or necrotizing fasciitis were noted. Ahmed et al reported that the common presentations were simple wound abscess and local cellulitis in his study⁸.

In this study the common single pathogen involved in postoperative wound infection was *E. coli* in 18 (36%). The others were *Staphylococcus aureus* 11 (22%), *Pseudomonas* 4 (8%). Most of the studies showed that the commonest organism was *Staphylococcus aureus*^{8,12,13}. In our series organism were isolated from 37 (74%) cases. Most of which were sensitive to cefuroxim and amikacin but almost all were resistant to amoxycilin, ampicilin and flucloxacillin. Siguan et al showed the organisms of surgical wound infections were most sensitive to ciprofloxacin, cefuroxim and ceftazidime¹⁴.

CONCLUSION

Surgical wound infections are common and consume a considerable portion of health care facility. Despite modern surgical and sterilization techniques, and prophylactic use of antibiotics, wound infection remains a major contributory factor of patient's morbidity. A reduction in the infection rate to a minimal level, however, can be achieved by meticulous surgical technique, proper sterilization, judicious use of antibiotics, improvement of operation theater and ward environment, control of malnutrition and obesity, treatment of infective foci and diseases like diabetes mellitus, and avoidance of smoking helps to control the morbidity of surgical wound infection.

REFERENCES

1. Lawrence WW, Gerard MD. Current Surgical Diagnosis & Treatment. 12th Edition. New York: McGraw-Hill; 2007. pp.106-7.
2. Robson MC. Wound infection. A failure of wound healing caused by an imbalance of bacteria. Surg Clin North Am 1997; 77(3): 637-50.
3. David JL. Surgical infection. In: Norman S. Williams, Cristopher J.K. Bulstrode, P. Ronan O'connel. Bailey & Love's Short Practice of Surgery. 25th edition. London: Arnold; 2008. pp.32-48.
4. Nichols RL. Wound infection rates following clean operative procedures: can assume them to be low? Infect Control Hosp Epidemiol 1992; 13(8): 455-6.
5. Singhal K, Kaur K, Zammit C. Wound Infection. Emedicine 2008 August. <http://www.emedicine.com/MED/topic2422.htm>.
6. Martone WJ, Garner JS. Proceeding of the Third Decennial International Conference on Nosocomial Infections. Am J Med 1991; 91(3B): 1S-333S.
7. Steven M, Steinberg J. Investigation and Treatment of Surgical infections. In: Cuscheri A, Steele RJC, Moosa AR, editors. Essential Surgical Practice. 3rd ed. London: Butterworth Heinemann; 1995. pp.20.
8. Ahmed M, Alam NS, Manzar SO. Post Operative Wound Infection: A Surgeon's Dilemma. Pak J Surg 2007; 23(1): 41-7.
9. Sorensen LT, Hemmingsen U, Kallehave F, Wille-Jørgensen P, Kjærgaard J, Møller LN, et al. Risk factors for tissue and wound complications in gastrointestinal Surgery. Ann Surg 2005; 241(4): 654-8.
10. Ford CD, VanMoorleghem G, Menlove RL. Blood transfusions and postoperative wound infection. Surgery 1993; 113(6): 603-7.
11. Collin J. Diabetic skin and soft tissue Infections. Curr opin Infect Dis 1994; 7(2): 214-8.
12. Akthar S, Ali AA, Karim F, Aslam N, Gondal KM, Chaudhry AM. Duration of surgery: Does it contribute to post-operative wound infection? Pak J Surg 2001; 17(3): 35-0.
13. Centers for Disease Control and Prevention. National nosocomial infections surveillance (NNIS) report, data summary from October 1986-April 1996, issued May 1996. A report from the National Nosocomial Infections Surveillance (NNIS) system. Am J Infect Control 1996; 24(5): 380-8.
14. Siguan SS, Ang BS, Pala IM, Baclig RM. Aerobic Surgical Infection: A Surveillance on Microbiological Etiology and Antimicrobial Sensitivity Patterns of Commonly used Antibiotics. Phil J Microbiol Infect Dis 1990; 19(1): 27-33.



Original Article

Histopathological Pattern of Ovarian Lesions

Suchanda Ray¹, Md Amzad Hossain Khan², Md Abed Hossain³, Md Sohorab Hossain Bhuiyan⁴, Jahan Ara Shirin⁵, Shajia Chowdhury⁶

^{1,6}Assistant professor, Department of Pathology, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Professor, Department of Pathology, Sylhet M.A.G. Osmani Medical College, Sylhet

³Professor, Department of Pathology, Jalalabad Ragib-Rabeya Medical College, Sylhet

^{4,5}Associate Professor, Department of Pathology, Jalalabad Ragib-Rabeya Medical College, Sylhet

ABSTRACT

The purpose of the study was to observe the histopathological pattern of ovarian lesions. This study was carried out in the department of Pathology, Sylhet M.A.G. Osmani Medical College during the the period of July 2007 to June 2008. A total of 80 cases who had ovarian tumor and tumor like lesions on the basis of clinical findings were included. Among the histopathologically confirmed diagnosed cases of 80 ovarian lesions, 7 cases were non-neoplastic, 57 cases were benign tumors, two were borderline tumors and 14 cases were malignant tumors. Among all malignant ovarian lesions, Serous cystadenocarcinoma was the most common malignant tumor (35.71%). The Others were mucinous cystadenocarcinoma (21.45%), endometrioid carcinoma (14.28%), dysgerminoma (14.28%), yolk sac tumor (7.14%) and metastatic tumors (7.14%). This study was mostly consistent with other published literatures.

Key words: Ovarian lesions, Serous cystadenocarcinoma.

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INTRODUCTION

Ovarian neoplasm is one of the complex areas of gynaecology because of its greater ranges and varieties than any other organ in the body. The comprehensive global cancer statistics from the International Agency for Research on Cancer point out that 2,04,000 new cases of ovarian cancer were diagnosed worldwide and 1,25000 deaths in the year 2002¹.

Ovarian cancer is one of the common causes of tumor related death in women and the most lethal gynaecological malignancy². Ovarian cancer is the fifth leading cause of cancer death among women of United States and has the highest mortality rate of all gynaecologic cancers³. In India, incidence of ovarian tumor amongst the gynaecological admission varies from 1-3%⁴, and in Bangladesh ovarian tumor constitutes 6.11% of total gynaecological admission⁵.

Approximately 80% of ovarian tumors are benign with cystic, solid or mixed characteristic having favorable

Address of correspondence: Dr Suchanda Ray,
Assistant professor, Department of Pathology, JRRMC,
Sylhet-3100.

prognosis and the remaining 20% constitute malignant tumors⁶. In a study by Choudhury in Bangladesh, 77.2% of ovarian tumors were found benign and 22.8% were malignant⁷. Almost similar results were found in other studies conducted in different region of Bangladesh, India and Pakistan^{5,8,9}.

Although frequently viewed as a single disease, ovarian cancer represents a group of related but distinct tumor types including epithelial tumors, germ cell tumors, sex cord stromal tumors and metastatic tumors¹⁰. Of these different types, cancers of epithelial origin are the most common of all ovarian malignancies. Epithelial cancers can inturn be divided into multiple histologic subtypes, such as serous, mucinous, endometrioid, clear cell, undifferentiated, squamous cell and transitional¹¹.

Most of the ovarian tumor's type cannot be easily distinguished from one another on the basis of their clinical or gross characteristics alone. Therefore histologic interpretation of ovarian neoplasms is important in distinguishing different types of ovarian tumors.

MATERIALS AND METHODS

This comparative cross sectional study was carried out in the department of Pathology, Sylhet M.A.G. Osmani Medical College. A total 80 patients of ovarian tumor and tumor-like lesions who were treated in inpatient department of Obstetrics and Gynaecology during the period of July 2007 to June 2008 were consecutively selected in this study. The patients were selected on the basis of history, physical examination and willingness to undergo operative treatment.

Data were collected from the enrolled patients by using a standardized questionnaire. All the relevant clinical informations of the patients under study were obtained before surgical intervention and subsequently specimen was sent for histopathological examination. Both clinical history and histopathological findings were recorded in a predesigned patient's profile made for the study.

Ethical clearance was obtained from the ethical review committee of SOMC. Informed written consent of the patient was taken. All the data were evaluated by the standard statistical method.

RESULTS

A total of 80 patients were included in the study. Their age ranged from 13 to 75 years. Considering the decade as a group, patients were divided into seven groups. The peak incidence 26 (32.5%) was seen in third decade. Out of 80 patients, the highest number 26 (32.5%) of cases belonged to the age group 21-30 years, 17 (21.25%) were 41-50 years, 14 (17.5%) were 11-20 years, 10 (12.5%) were 31-40 years, 8 (10%) were 51-60 years, 4 (5%) were 61-70 years and 1 (1.25%) were 71-80 years, which is shown in Table-I.

Table-I: Age distribution of study subjects

Age group	Frequency	Percent (%)
11-20	14	17.50
21-30	26	32.50
31-40	10	12.50
41-50	17	21.25
51-60	8	10.00
61-70	4	5.00
71-80	1	1.25

Distribution of age group with histopathological diagnosis:

Among the histopathologically confirmed diagnosed cases of 80 ovarian lesions, 7 cases were non-neoplastic lesions, 57 cases were benign tumors, 2 were borderline tumors and 14 cases were malignant tumors. Of the non-neoplastic lesions, 1 case was found in 11-20 age group, 3 cases were found in 21-30 age

group, 2 cases were found in 31-40 age group and 1 case was found in 41-50 age group.

Laterality distribution of ovarian lesions in the study subject:

Most of the benign lesions were unilateral and malignant tumors were bilateral. Among the benign lesions (57 benign tumors and 7 non neoplastic cases), 49 (76.56%) cases were unilateral and 15 (23.44%) cases were bilateral.

Mobility of the lesions:

Among 80 cases of ovarian lesions, 65 cases were mobile and 15 cases were fixed with underlying structures. In these 65 mobile cases, 57 cases were benign lesions, 2 cases were borderline tumors, 6 cases were malignant tumors as diagnosed by histopathological examination.

Consistency of the lesions:

The consistency of ovarian lesions were cystic, solid and mixed. Among the 80 cases, 54 were cystic, 15 were solid and 11 were mixed. In 54 cystic cases, histopathology showed 51 were benign, 1 was borderline tumor and 2 cases were malignant tumors.

Clinical diagnosis of the lesions:

As per clinical diagnosis 51 cases were benign ovarian tumor, 12 were malignant ovarian tumor and 17 were adnexal mass. Later on final histopathological diagnosis was obtained. Among clinically diagnosed 51 benign tumors, histopathologically 44 were benign tumors, 7 were non-neoplastic cyst. Among clinically diagnosed 12 malignant tumors, 8 were malignant tumors, 1 was borderline tumor and 3 were benign tumors as diagnosed histopathologically.

Histopathological diagnosis of ovarian lesions:

Histopathological examination was done in all 80 cases of ovarian lesions. The results of histopathological examination show 7 (8.75%) non-neoplastic lesions, 57 (71.25%) benign tumors, 2 (2.5%) borderline tumor and 14 (17.5%) malignant tumors. These findings are depicted in the Table-II.

Table-II: Histopathology of ovarian lesions

Histopathology	Frequency	Percent (%)
Non-neoplastic lesions	7	8.75%
Benign tumor	57	71.25%
Borderline tumor	2	2.50%
Malignant tumor	14	17.50%
Total	80	100.00%

Among the histopathologically confirmed 14 malignant ovarian tumors, 5 (35.71%) cases were serous cyst adenocarcinoma, 3 (21.45%) cases were mucinous cystadenocarcinoma, 2 (14.28%) were dysgerminoma,

1 (7.14%) was yolk sac tumor, 2 (14.28%) were endometrioid carcinoma, 1 (7.14%) was metastatic carcinoma. These findings are depicted in the Table-III.

Table-III: Histopathological diagnosis of malignant ovarian lesions

Histopathological diagnosis	Frequency	Percent
Serous cystadenocarcinoma	5	35.71
Mucinous cystadenocarcinoma	3	21.45
Dysgerminoma	2	14.28
Endometrioid carcinoma	2	14.28
Yolk sac tumor	1	7.14
Metastatic carcinoma	1	7.14

Most of the benign lesions were unilateral and malignant tumors were bilateral. Among the benign lesions, 49 (76.56%) cases were unilateral and 15 (23.44%) cases were bilateral.

Among 80 cases of ovarian lesions, 65 were mobile and 15 were fixed with underlying structures. The consistency of ovarian lesions was cystic, solid and mixed. Among the 80 cases, 54 were cystic, 15 were solid and 11 were mixed.

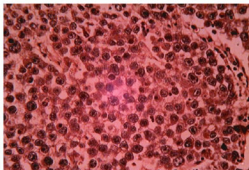


Figure-1: Dysgerminoma



Figure-2: Serous cystadenocarcinoma

DISCUSSION

Epithelial neoplasms are the most common forms of ovarian tumors. The single most malignancy of the ovary is serous adenocarcinoma, while its benign counterpart is considered as the most common benign ovarian tumor¹². In a study of Hogdall et al, among 584 cases of ovarian cancer highest percentage 62% (362) cases were diagnosed as serous adenocarcinoma¹³.

This study revealed that, serous cystadenocarcinoma is the most common malignant tumor among all ovarian malignancy. Tushar et al carried out a study of 67 cases of ovarian tumor, malignant serous tumor accounted for 17.94%, which closely resembles our study¹⁴. It was found in our study that malignant ovarian tumors were common in 3rd and 4th decades. This result closely resembles to studies by Negri et al¹⁵. Smith and OI found that epithelial cancers of ovary occur most frequently in the peri and post menopausal group with a median age of 50 yrs¹⁶. Ries et al carried out a study of ovarian cancer in which the median age at diagnosis is 63 yrs, which does not correlate with the present study¹⁷.

In the present study, most of the benign lesions were unilateral and malignant tumors were bilateral. Among the benign lesions, 49 (76.56%) cases were unilateral and 15 (23.44%) cases were bilateral.

In our study mucinous cyst adenocarcinoma is the second most malignant ovarian tumor. In a study of 42 cases of ovarian carcinoma, Karen et al accounted only 9 (21.42%) cases as mucinous cyst adenocarcinoma, which is lower than the present study¹⁸. There were 2 (14.28%) cases diagnosed as endometrioid carcinoma among all malignant ovarian cancers in our study, which is higher than the study done by Peter and Wenxin where endometrioid carcinoma accounted for 4.65%¹⁹.

In the present study, most of the benign lesions were unilateral and malignant tumors were bilateral. Among the benign lesions, 49 cases (76.56%) were unilateral and 15 cases (23.44%) cases were bilateral. In malignant tumors including metastatic adenocarcinoma, 9 cases (64.29%) were bilateral and only 5 cases (35.71%) cases were unilateral. Among the borderline tumors, 1 case (50%) was unilateral and another 1 case was bilateral. Among 80 ovarian lesions, 65 cases were mobile and 15 cases were fixed with underline structure. The consistency of ovarian lesion were cystic, solid and mixed. Among the 80 cases, 54 cases were cystic, 15 cases were solid and 11 cases were mixed.

CONCLUSION

It can be concluded from this study that serous cystadenocarcinoma is the most common pattern of malignant ovarian tumor.

REFERENCES

1. Sankaranarayanan R and Ferley J. Worldwide burden of gynaecological cancer: the size of the problem. *Best Prac Res Clin Obstet Gynaecol* 2006. 20: 207-25.
2. Greenlee RT, Murray T, Boldne S, Wingo PA. Cancer statistics. *CA Cancer J Clin*. 2000. 50: 7-33.
3. American cancer society. *Cancer Facts and Figures 2006*. Atlanta : American Cancer society ; 2006.
4. Konar Hiralal .2005. Dutta's Textbook of gynaecology, 6th edition, New Central Book Agency, Kolkata, 271.
5. Begum S. Clinical presentation of benign and malignant ovarian tumor-A comparative study, FCPS Dissertarion, BCPS, 1996. Dhaka, Bangladesh.
6. Kumar V, Abbas A K and Fausto N. 2006. Robbins and Cotran Pathological Basis of Disease, 7th edn. W.B. Saunders Company, Philadelphia, pp.1093.
7. Choudhury S, Mohiuddin AS, Ahmed AU , Ahsan S. Preoperative discrimination of benign and malignant ovarian tumors using color Doppler sonography and its correlation with histopathology, *Bangladesh Med Res Counc Bull*. 2005. 31: 21-6.
8. Pilli GS, Suneeta KP, Dhaded A V and Yenni VV. Ovarian tumors: a study of 282 cases. *J Indian Med Assoc*. 2002. 100: 420,423-4,447.
9. Saha NK and Hena SNB. Ovarian tumors-A five year retrospective study. *Sylhet Med J*. 2003. 24: 33-6.
10. Moss C, Kaye SB. Ovarian cancer, progress and continuing controversies in management. *Eur J Cancer*. 2002. 38: 1701-7.
11. Young RH, Clement PB, Scully RE. The ovary. In: Sternberg SS, ed. *Diagnostic Surgical Pathology*. Philadelphia, PA: Lippincott, Williams and Wilkins, 1999: pp.2307-94.
12. Coffey D, Kaplan A L, Ramzy. Intraoperative Consultation in Gynecologic Pathology, *Arch Pathol Lab Med*. 2005. 129: 1544-7.
13. Hogdall EV, Christensen L, Kjaer SK, Blaakaer J, Kjaerby-Thygesen A, Gayther S et al. CA 125 expression pattern, prognosis and correlation with serum CA 125 in ovarian tumor patients. *Gynecologic Oncology*. 2007. 104: 508-15.
14. Tushar K, Asaranti K, Mohapatra PC. Intraoperative cytology of ovarian tumors. *J Obstet Gynecol India*. 2005. 55: 345-9.
15. Negri E, Franceschi S, Tzonou A. Pooled analysis of 3 European case-control studies. Reproductive factors and risk of epithelial ovarian cancer. *Int J Cancer*. 1991; 49: 50-6.
16. Smith LH, OI RH. Detection of malignant ovarian neoplasms: a review of the literature. I. Detection of the patient at risk; clinical, radiological and cytological detection, *Obstet Gynecol Surv*. 1984. 39 (6): 313-28.
17. Ries LAG, Eisner MP, Kosary CL. SEER cancer statistics review, 1975-2002.
18. Karen H. Lu, Andrea P. Patterson, Lin Wang. Selection of potential marker for epithelial ovarian cancer with gene expression arrays and recursive descent partition analysis. *Clinical Cancer Research*. 2004. 10: 3291-00.
19. Peter E Schwartz and Wenxin Zheng. Neo adjuvant chemotherapy for advanced ovarian cancer: the role of cytology in pretreatment diagnosis. *Gynecologic oncology*. 2003. 90: 644-50.



Original Article

Ocular Changes during Pregnancy in Tribal Women of Sylhet

Nandan Kushum Das¹, Mushahid Thakur², SA Muktad Razu³, Syeda Fatima Jinat⁴, Nasrin Sultana⁵, Md Altaf Hossain⁶

¹Assistant professor, Department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Professor, Department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College, Sylhet

³Associate professor Department of Ophthalmology, TMSS Medical College, Bogra

^{4,5}IMO, Department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

⁶Ex. Registrar, Department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

ABSTRACT

Pregnancy induces a variety of physiological, anatomical, metabolic and endocrine changes in women. During pregnancy, like other organs there are significant physiological and pathological changes that occur in the eyes. Moreover, pre-existing ocular conditions may worsen during pregnancy. The purpose of this study was to find out the ocular changes during pregnancy in tribal women of Sylhet. This was a cross-sectional observational study carried out in the Department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet from 1st January 2009 to 31st December 2009. One hundred Manipuri pregnant women were included in this study. Purposive sampling (non-randomized) technique was applied to select sample. Ocular changes were evaluated by measurement of intraocular pressure (IOP), corneal sensitivity testing, retinoscopy, direct ophthalmoscopy, slit lamp examination and visual acuity testing. The age ranged from 18 to 45 years with mean age 29.9 (SD±5.8) years. Myopia was the most common refractive error (75%). The intraocular pressure was found within normal physiological limit. Corneal sensitivity was decreased in 71% cases. Pathological ocular changes were central serous retinopathy (2), macular oedema (2), ptosis (2) and pregnancy induced hypertension [hypertensive retinopathy (1)]. In tribal pregnant women, ocular changes during pregnancy were not significantly different from the ethnic group of population. All physiological and pathological ocular changes during pregnancy were resolved by 6 weeks of delivery. This study put forward some message about the effect of pregnancy on eyes and need for special attention specially during refraction, measurement of intraocular pressure, assessment of corneal sensitivity and management of diabetic retinopathy.

Key words: Ocular changes, Pregnancy, Tribal women, Intraocular pressure, Pregnancy induced hypertension, Diabetic retinopathy.

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INTRODUCTION

There are progressive anatomical and physiological changes during pregnancy that occur not only in the genital organs but also in other systems of the body, including the eyes^{1,2}. The ocular changes are most often transient in nature, though occasionally may become permanent^{3,4,5,6}.

The pregnancy induced ocular changes may be divided into physiological and pathological changes or

modification of pre-existing conditions^{2,3,5,7}. The physiological changes in the eyes include decrease corneal sensitivity, decrease intra-ocular pressure and change in refraction^{2,6,7,8,9}. Pathological changes include central serous retinopathy (CSR), optic neuropathy, macular oedema, serous retinal detachment, ptosis and pregnancy induced hypertension (PIH)^{2,3,6,7,8}.

The most common pre-existing ocular condition modified by pregnancy is diabetic retinopathy^{5,6,10} and other conditions modified by pregnancy are non infectious uveitits, pituitary adenoma, meningioma etc^{2,8}.

Address of Correspondence: Dr Nandan Kushum Das, Assistant Professor, Department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College, Sylhet-3100.

At least 350 million people worldwide are considered to be indigenous and in Bangladesh about two million indigenous people of 45 different distinct communities are living throughout the country¹¹. These people with distinctive social and cultural practices, languages and customs are commonly known as 'Adibasy' by themselves¹². They live in the Chittagong hill tracts, some regions of Mymensing, Sylhet and Rajshahi. They differ in their social organization, marriage customs, birth and death rates, food and other social customs from the people of the rest of the country. Manipuris are living densely in rural areas of Komalganj upazila of Moulvibazar district and scattered in all four districts of Sylhet division. They had migrated from Manipur, a state of India, in late seventeenth century^{13,14,15}.

The pregnant Manipuri women seek health care services (antenatal checkup) from Thana Health Complex at Komalganj and from private and government hospitals of Sylhet city. Ophthalmic care facilities in those areas where they live are inadequate. In addition, the pattern of ophthalmological problem in a small ethnic group in Bangladesh should be screened out as they are diverse from the main steam of population.

Monitoring the ocular changes during pregnancy among tribal women may provide some informations that can be utilized to create awareness, to prevent and manage the ocular changes in pregnancy in respective tribal groups.

MATERIALS AND METHODS

This was a cross-sectional observational study, conducted in the department of Ophthalmology, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet during 1st January 2009 to 31st December 2009. Total 100 tribal pregnant women who attended the departments of Ophthalmology & Obstetrics & Gynecology of this hospital, and at Komalganj Thana Health Complex of Moulvibazar, fulfilling the inclusion criteria (Tribal pregnant women between 18-45 years) and exclusion criteria (History of hypertension) were enrolled in this study. Purposive sampling (non-randomized) technique was applied to select sample.

Ocular examination: Ocular evaluation of patient was conducted in the department of Ophthalmology Jalalabad Ragib-Rabeya Medical College Hospital and Sylhet MAG Osmani Medical College Hospital, Sylhet and special ophthalmological examination unit of Komalganj health complex with the help of torch light, binocular loupe, slit lamp biomicroscope, Schiotz

tonometer, Snell's chart, trial box, retinoscope, direct ophthalmoscope, cotton threads etc. Data were processed manually and analyzed with the help of SPSS. Informed written consent was taken from each patient before taking any interview.

RESULTS

A total 100 pregnant Manipuri women attended the different study areas during the period. Their age ranged from 18 to 45 years with mean age 29.9 (SD \pm 5.8) years. Young pregnant women attended more in different health care facilities (72%). Among the cases 64% were multipara, 34% were primi and 2% were grand multipara. Out of 100 cases, 16 had refractive errors; among them myopia was the most common refractive error (75%). The others were hypermetropia (12.5%) and astigmatism (12.5%).

Table-I: Distribution of patients by intraocular pressure (n=100)

Intraocular pressure	Number of patients	Percent
10-14 mm of Hg	74	74
15-21 mm of Hg	26	26
> 21 mm of Hg	00	00
Mean (SD)	12.5 (SD \pm 2.1)	

The intraocular pressure of participants ranged from 10 to 20 mm of Hg with the mean 12.5 (SD \pm 2.1) mm of Hg. None of the participants had raised intraocular pressure.

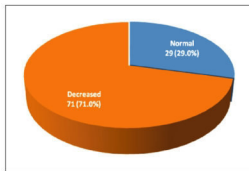


Figure-1: Distribution of patients by corneal sensitivity

Figure-1 shows the changes in corneal sensitivity among the participating pregnant tribal ladies. Corneal sensitivity was decreased in 71% cases.

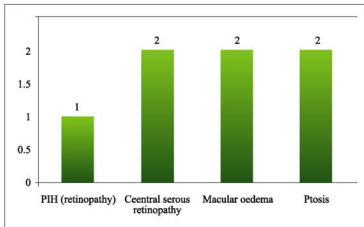


Figure-2: Distribution of cases according to pathological changes

Figure-2 presents the pathological ocular changes. Out of 100 pregnant women, pathological ocular changes were found in 7 women. The changes were central serous retinopathy (2), macular oedema (2), ptosis (2) and pregnancy induced hypertension [Hypertensive retinopathy (1)].

Table-II: Preexisting ocular condition changed during pregnancy

Conditions	During 1 st trimester	During 2 nd trimester	During 3 rd trimester
Diabetic retinopathy	Non proliferative	Non proliferative	Proliferative
Glaucoma (POAG) (Without medicines)	Optic disc change (Cup disc ratio 0.5)	Optic disc change (Cup disc ratio 0.5)	Optic disc change (Cup disc ratio 0.5)
	IOP: 20 mm of Hg	IOP: 16 mm of Hg	IOP: 14 mm of Hg

POAG: Primary Open Angle Glaucoma

Table-II shows the changes of pre-existing ocular conditions detected during different trimester of pregnancy. In 3rd trimester of pregnancy, non-proliferative diabetic retinopathy progressed to proliferative diabetic retinopathy. In the patient with primary open angle glaucoma (POAG), no significant changes were detected in respect to optic disc change and IOP.

DISCUSSION

This study revealed a significant number of physiological and pathological ocular changes occurred during pregnancy in Manipuri women. In addition, a significant change in pre-existing ocular condition was also found in this series. The changes were comparable to the findings of other studies in different settings. However, lack of data on Bangladeshi pregnant women hindered us to compare data with local population.

Among the tribals of Sylhet division, Manipuris are predominant tribal group and only Manipuris were enrolled in our study.

Pregnancy is associated with changes in corneal sensitivity and thickness². In this series, corneal sensitivity was decreased in 71% of Manipuri women. The result was comparable with the findings of Riss et al¹⁶. Decrease in corneal sensitivity in pregnancy may be due to accumulation of fluid in different ocular structures. In this study, within 6 weeks of delivery the corneal sensitivity of some patients came back to normal.

During measuring visual acuity, refractive errors were newly diagnosed in 16 cases which were subsequently confirmed by retinoscopy. Myopia was the most common type found in 12 cases. Change in thickness of cornea due to fluid accumulation may alter the refractive index of cornea, which may change the refraction. The curvature of lens may increase causing a myopic shift in refraction^{9,17}. The refractive errors in

pregnancy is generally reversible⁹.

The ocular complication such as diabetic retinopathy was evident in one patient. Diabetic retinopathy was associated with type II diabetes mellitus. This patient was followed up according to the American Academy of Ophthalmology Guidelines¹⁸, from 1st to last trimester of pregnancy as pregnancy is a major risk factor for the progression of diabetic retinopathy¹⁹. Several previous studies indicated the degree of retinopathy progression during the course of pregnancy. Axier-Seigel et al²⁰ found 26% out of 65 patients without retinopathy at conception developed mild non-proliferative diabetic retinopathy (NPDR) during the course of pregnancy. Twenty two percent of patients with initial NPDR progressed to proliferative retinopathy²⁰. The pathogenesis for the acceleration of diabetic retinopathy during pregnancy is unclear. Several investigators have studied retinal circulatory changes in diabetes and control subjects during pregnancy. Chen et al²¹ found an increase in retinal blood flow in pregnant women who had worsening of their diabetic retinopathy. In contrast Schochet et al²² noted that retinal venous diameter and retinal blood flow decreased to a greater degree in diabetic mothers compared with nondiabetic mothers. Thus they proposed that the decrease in blood flow might exacerbate retinal ischemia and hypoxia leading to the acceleration of diabetic retinopathy. In this series, despite taking the preventive measures, the NPDR type transformed to proliferative one. Irregularity in taking insulin as well as some social taboos may be the causes behind it. Social taboos such as in Manipuri society, food intake increased during pregnancy may hinder the preferred glycaemic control of the patient.

In this study, IOP was found within normal limit including one patient of glaucoma. The patient was followed up regularly in all the trimester of pregnancy without anti glaucoma medications and no significant change was observed in optic disc. IOP decreases in both normal and glaucomatous eyes during pregnancy. Pregnancy decreases about 19.6% and 24.4% IOP in normal eye and glaucomatous (ocular hypertension) eye respectively²³. The results of this study correspond with the findings of the above studies. Several mechanisms have been implicated for decrement of IOP in pregnancy. Such mechanisms include an increase in aqueous outflow, a decrease in systemic vascular resistance, leading to decreased episcleral venous pressure, generalized increased tissue elasticity leading to decreased scleral rigidity and generalized acidosis during pregnancy^{3,5}.

The pathological ocular changes detected in the

Manipuri pregnant women were CSR (2), macular oedema (2), mild ptosis (2) and ocular changes due to PIH (1). CSR is predominantly a disorder of middle aged adult male but it may occur in all three trimester of pregnancy²⁴. There may be no racial predominance in the development of CSR in pregnancy³. Subretinal fibrous exudates are found in 90% patients of CSR associated with pregnancy compared to 20% in nonpregnant female and male²⁵. Subretinal fibrous exudates are diagnosed by fluorescein angiography which is not done in this study due to lack of facilities. In this study, CSR was diagnosed clinically which is the easy and reliable method of CSR diagnosis. The etiology of CSR in pregnancy is unknown, though hormones, coagulation and hemodynamic changes may play a role⁵.

Bilateral macular edema was found in 2% women. Macular edema with or without proliferative retinopathy may occur during pregnancy^{3,26}. In this study, macular edema was not associated with diabetes mellitus and hypertension. In one patient, macular edema resolved spontaneously after four weeks of delivery. And in other patient, it did not resolve rather diminished the vision.

Mild ptosis was present unilaterally in right upper eyelid in 2% of cases. Ptosis has been reported to occur during and after normal pregnancy and is usually unilateral. The mechanism is thought to be due to defects that develop in levator aponeurosis from fluid, hormonal and other changes from the stress of pregnancy⁸. However, ptosis recovered after two weeks of delivery.

A patient of eclampsia attended with diminished vision with occasional convulsion at full term pregnancy. Ophthalmoscopic examination showed arteriolar narrowing, retinal hemorrhage and hard exudates. The patient was primi and a healthy male baby was delivered through an emergency caesarean section. The mother was followed up weekly and her ocular symptoms improved gradually and visual acuity became 6/9 at 6th postpartum week. Ophthalmoscopic examination at 6th week showed recovery of pathological findings of retina and there was no retinal detachment. Like findings of other studies^{27,28}, this patient had almost complete recovery.

CONCLUSION

From our study it can be concluded that there occur some ocular changes in tribal pregnant women which are generally reversible but proper ophthalmic care should be taken to prevent permanent damage to the eyes.

REFERENCES

- Dutta DC. Physiological changes during pregnancy. In: Konar H (editor). Textbook of Obstetrics including perinatology and contraception. 6th ed. Kolkata: New Central Book Agency (P) Ltd; 2004, p 46-56.
- Somani S. Pregnancy, special considerations. eMedicine. Updated: Dec 1, 2011. Available from: <http://emedicine.medscape.com/article/1229740>. Accessed on 8 May 2012.
- Omoti AE, Waziri Ermeh JM, Okeiybemen VW. A review of the changes in the ophthalmic and visual system in pregnancy. African Journal of Reproductive Health 2008; 12: 185-96.
- Kubicka-Trzaska A, Karska-Basta I, Kobylarz J, Romanowska-Dixon B. Pregnancy and the eye. Klin Oczna 2008; 110: 401-4.
- Sheth BP, Mieler WF. Ocular complication of pregnancy. Current Opinion in Ophthalmology 2001; 12: 455-63.
- Barbaze A, Pizzarello LD. Ocular changes during pregnancy. Comp Ophthalmol update 2007; 8:155-67.
- Vitale BF, Nuzzi R, Fea A. The eye and pregnancy. Minerva Ginecol 1991; 43: 141-67.
- Bhatia J, Sadiq MN, Chaudhury TA, Bhatia A. Eye changes and risk of ocular medications during pregnancy and their management. Pak J Ophthalmol 2007; 23: 20-4.
- Pizzarello LD. Refractive changes in pregnancy. Grafer Arch Clin Exp Ophthalmol 2003; 241: 484-8.
- Pilas PM, Czajkowski J, Ozzukowski P. Ocular changes during pregnancy. Gynecol Pol 2005; 76: 655-60.
- International Work Group on Indigenous Affairs. 2001 Indigenous issues. Retrieved from: <http://www.iwgia.org/sw155.asp>. Accessed on September 5, 2006.
- Chakma S. Bangladesh: Racial Discrimination against Indigenous Peoples. An alternative report to the CERD Committee. Geneva, Switzerland. 1999.
- Sheram AK. Manipuri (Meitei) settlement in Bangladesh. In: Sanajaoba N (editor) Manipur: Past and Present: The Ordeals and Heritage of a civilization. Vol.-IV (Pan-Manipur in Asia and Autochthones). New Delhi: Mittal Publications; 2005.
- Sattar A. Aaranya Janapade (Research on tribal peoples of Bangladesh). 2nd edition. Dhaka: NASAS; 1975.
- Bangladesh Bureau of Statistics. Tribal households and population by tribe, 1991. In: Statistical Pocket Book of Bangladesh 2005. Bangladesh Bureau of Statistics, Dhaka; 2006.
- Riss B, Riss P. Corneal sensitivity in pregnancy. Ophthalmologica 1981; 183: 57-62.
- Dinn RB, Haris A and Marcus PS. Ocular changes in pregnancy. Obstetrical and Gynaecological Surgery 2003; 58: 137-44.
- Diabetic Retinopathy. Preferred Practice Patterns. The American Academy of Ophthalmology 1998; 13: 28.
- Sneth B. Does pregnancy accelerate the rate of progression of diabetic retinopathy? Curr Dia Rep 2002; 2: 327-30.
- Axier-Seigel R, Hod M, Fink-Cohen S, Kramer M, Weinberger D, Schindel B, et al. Diabetic retinopathy during pregnancy. Ophthalmol 1996; 104: 1806-10.
- Chen HC, Newsom RS, Patel V, Cassar J, Mather H, Kohner EM. Retinal blood flow changes during pregnancy in women with diabetes. Invest Ophthalmol Vis Sci 1994; 35: 3199-208.
- Schocket LS, Grunwald JE, Tsang AF, DuPont J. The effects of pregnancy on hemodynamics in diabetic versus nondiabetic mothers. Am J Ophthalmol 1999; 128: 477-84.
- Qureshi IA. Intraocular pressure and pregnancy: a comparison between normal and ocular hypertensive subjects. Arch Med Res 1997; 28: 397-400.
- Sunness JS, Haller JA, Fine SL. Central serous retinopathy and pregnancy. Arch Ophthalmol 1993; 111: 360-4.
- Gass JD. Central serous chorioretinopathy and white subretinal exudation during pregnancy. Arch Ophthalmol 1991; 109: 677-81.
- Sinclair SH, Nesler C, Foxman B, Nichols CW, Gabbe S. Macular edema and pregnancy in insulin dependent diabetes. Am J Ophthalmol 1984; 97: 154-67.
- Somfai GM, Mihaltz K, Tulassay E, Rigo J. Dignosis of serous neuroretinal detachments of the macular in severe preeclamptic patients with optical coherence tomography. Hypertens Pregnancy 2006; 25: 11-20.
- Prado RS, Figueiredo EL, Magalhaes TV. Retinal detachment in preeclampsia. Arq Bras Cardiol 2002; 79: 183-6.



Original Article

CAG Profile and Procedural Outcome of 200 Cases Performed in a Peripheral Cath Center of Bangladesh

M Nurul Afsar¹, Kamal Ahmed², Sufia Rahman³

¹Assistant Professor, Department of Cardiology, North East Medical College, Sylhet

²Assistant Professor, Department of Medicine, North East Medical College, Sylhet

³Professor (Emeritus), Department of Cardiology, North East Medical College, Sylhet

ABSTRACT

This was a cross sectional study focusing the prevalence of coronary artery disease in people of Sylhet Division and also to determine the procedural outcome by observing the procedural success and complications related to the procedure performed in this newly setup cardiac center. Percutaneous coronary angiography (CAG) was performed in 200 consecutive patients with suggestive of coronary artery disease (CAD) during the period of January 2009 to February 2012. The population consisted 150 (75%) male and 50 (25%) female. Age distribution in this study was (12) 6.08% belongs to 30-39 years, (35) 17.57% to 40-49 years, (100) 50% to 50-59 years and (53) 26.35% belongs to age group > 60 years. Clinically (70) 35% patients had history of myocardial infarction (MI), while (17) 8.5% had unstable angina, (105) 52.5% stable angina and (8) 4% non-specific chest pain. Risk factors of CAD showed smoking (140) 70%, diabetes mellitus (DM) (120) 60%, hypercholesterolemia (110) 55% and hypertension (HTN) (100) 50%. We observed positive angiographic lesion sites and number of vessel(s) involvement, average percentage of stenosis, procedure related complications & outcome. CAG findings showed (60) 30.40% had normal coronary arteries, while (46) 22.97% single vessel disease (SVD), (58) 29.05% double vessel disease (DVD), (34) 16.89% triple vessel disease (TVD) and diffuse lesions (02) 1%. Among SVD; left anterior descending (LAD) stenosis was in (24) 52.17%, right coronary artery (RCA) stenosis in (13) 28.26% & left circumflex (LCX) stenosis was in (9) 19.57%. Among DVD; LAD + LCX stenosis were in (21) 36.20%, LAD + RCA in (22) 38% and RCA + LCX stenosis in (15) 25.86%. After CAG (62) 31% patients were advised for medical treatment which include normal and diffuse lesion, (104) 52% of SVD & DVD for PTCA and (34) 17% for CABG especially patient with TVD. Procedural success was 100% & no complication was noted.

Key words: Coronary angiography, Myocardial infarction, Angina.

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INTRODUCTION

The reported prevalence of coronary artery disease 1.6% to 4.1% have been observed among the general population^{1,2}. With the development of effective treatment options for coronary artery disease (i.e. bypass surgery or angioplasty), diagnostic coronary angiography (coronary arteriography) has become one of the primary components of cardiac catheterization. It is estimated that more than 1,20,000 coronary

angiographic procedures (roughly 400 per 100,000 population) are performed each year in United States with a procedure related mortality rate of 0.1%^{3,4}. The objective of CAG is to examine the entire coronary tree (both native vessels & any surgically reconstructed bypass grafts), recording details of coronary anatomy that include the individual pattern of arterial distribution, anatomic or functional pathology (atherosclerosis, thrombosis, congenital anomalies, or focal coronary spasm), and the presence of intercoronary & intracoronary collateral connections. Despite invention of non-invasive techniques as magnetic resonance angiography (MRA), fast

Address of correspondence: Dr. M. Nurul Afsar, Assistant Professor, Department of Cardiology, North East Medical College, Sylhet.

computed tomography (FCT), intravascular ultrasound & angiography as screening tests for coronary artery disease; the use of coronary angiography (CAG) remains the clinical "gold standard" for evaluating coronary anatomy².

Though CAG is an invasive procedure but now a days it is being practiced as out-patient basis in many developed centers. In our country facilities of this amazing investigation is confined within few centers. To date, very few studies, have attempted to evaluate the CAG finding in peripheral Cath Lab centre in our country. Performing the CAG in the North East Medical College Hospital (NEMCH cath lab), Sylhet Bangladesh is a breakthrough in the field of modern treatment of coronary artery disease outside Dhaka. It brings the opportunity to have better management of CAD with the local facilities at a cost effective rate.

MATERIALS AND METHODS

Two hundred cases of males and females, who presented in the department of Cardiology, North East Medical College Hospital (NEMCH) Sylhet, from January 2009 to February 2012 were included in this study. Patients were selected on the basis of inclusion and exclusion criteria as mentioned below. The patient's personal data including age, sex, clinical diagnosis, risk factors of CAD & types of MI were recorded. Details angiographic findings including vessels involvement & lesion's morphology were recorded & studied. To determine the procedural outcome, all possible relevant complications were observed & recorded accordingly. The study was approved by the review committee of the medical college.

1. Study population

1.a. Inclusion criteria:

All patients clinically diagnosed or documented to have CAD, who required coronary angiography (CAG) were taken as study population. Informed consent was taken from all patients.

1.b. Criteria for coronary artery disease (CAD) & Coronary Angiography (CAG):

1. Chronic stable angina pectoris with positive E.T.T (with or without previous MI).
2. Unstable angina.
3. Atypical chest pain (non specific chest pain).
4. After acute MI (with or without persistent angina).

1.c. CAD risk factors were defined as:

1. Hypertension: on anti hypertensive therapy & or BP >140/90 mm of Hg.
2. DM: on hypoglycemic medication and/or patient

who fulfilled the diagnostic criteria for DM recommended by the World Health Organization (WHO) in 2005.

3. Dyslipidaemia (hypercholesterolemia): Serum cholesterol >200 mg/dl; Serum LDL >115 mg/dl.

1.d. Exclusion criteria:

1. Patient with hypertrophic or dilated cardiomyopathy.
2. Patient with valvular heart disease.
3. Patient with congenital heart disease.

2. Coronary Angiographic (CAG) Procedure

CAG were done in all patients by standard Judkin's technique through femoral approach by modified Seldinger's technique using non ionic dye. Multi angled standard views were recorded for analysis. A comprehensive analysis of coronary angiogram (CAG) was done; severity & extent of arterial disease were measured by eye estimation. The pre requisites for CAG were followed according to the hospital protocol and then morphological characteristics of lesions were analyzed.

- a) Positive CAG: was taken when coronary artery stenosis 50%
- b) Negative CAG: was taken when coronary artery stenosis < 50%
- c) According to branches of coronary artery involvement:
 - i. single vessel disease (SVD): one coronary artery involved
 - ii. double vessel disease (DVD): two coronary artery involved
 - iii. triple vessel disease (TVD): three coronary artery involved
 - iv. diffuse lesion: diffusely involved one or more coronary artery

3. Statistical analysis

After processing all available informations, statistical analysis and their significance were done. All parametric values were expressed as mean and nonparametric values were expressed in percentage (%).

RESULTS

Total 200 patients of 30-80 years of age were included in this study. Out of these 6.08% belonged to 30-39 years, 17.57% were 40-49 years, 50% were 50-59 years & 26.35% belonged to age group > 60 years which is shown in Figure 1. There were 150 (75%) male and 50 (25%) female patients.

Pre procedure clinical diagnosis shows (70) 35% patients had history of myocardial infarction (MI), (17) 8.5% had unstable angina, (105) 52.5% had stable

angina & (8) 4% patients had non-specific chest pain as illustrated in Figure 2. Regarding risk factors of coronary artery disease we observed that smoking (140) 70%, DM (120) 60%, hypercholesterolemia (110) 55% & HTN constitute (100) 50% cases as shown in Figure 3.

Coronary angiographic finding revealed (60) 30.40% normal coronary arteries, (46) 22.97% single vessel disease (SVD), (58) 29.05% double vessel disease (DVD), (34) 16.89% triple vessel disease (TVD) and diffuse lesions were in (02) 1%. This findings are shown in Figure 4.

Among the cases of SVD; left anterior descending (LAD) stenosis (24) 52.17%, right coronary artery (RCA) stenosis (13) 28.26% & left circumflex (LCX) stenosis were (9) 19.57% as shown in Figure 5. Among DVD; LAD + LCX stenosis (21) 36.20%, LAD + RCA (22) 38% & RCA + LCX stenosis (15) 25.86% were noted.

After the procedure (62) 31% patients were advised for medical treatment which included normal & diffuse lesions (104) 52% of SVD & DVD for PTCA & (34) 17% for CABG (Figure 6). Procedural success were 100% & no complication noted.

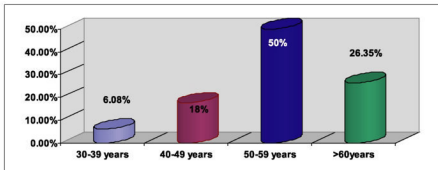


Figure-1: Showing age distribution

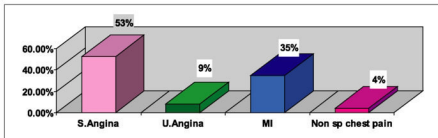


Figure-2: Showing clinical diagnosis before CAG

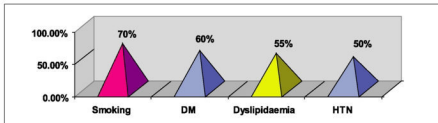


Figure-3: Showing risk factors of CAD

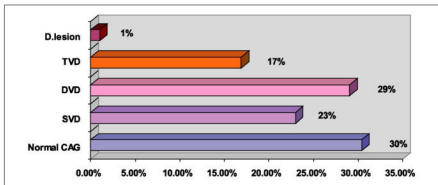


Figure-4: Showing CAG findings

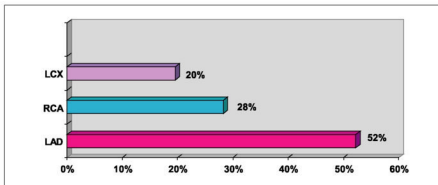


Figure-5: Showing vessels involvement among SVD (n=46=100%)

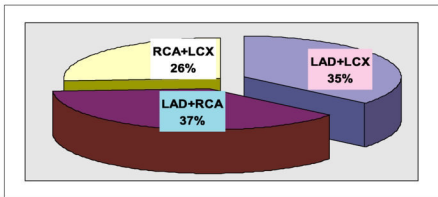


Figure-6: Showing vessels involvement among DVD (n=58=100%)

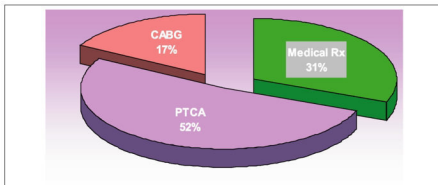


Figure-7: Showing recommendation after CAG

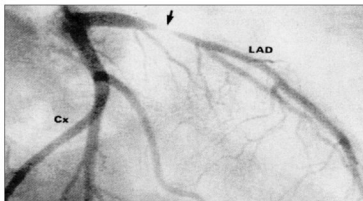


Figure-8: SVD (Left anterior descending artery-LAD)

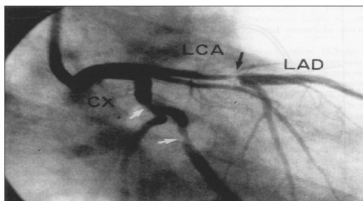


Figure-9: DVD (Left anterior descending & circumflex artery)

DISCUSSION

Coronary angiography (CAG) usually performed in patient with documented ischemic chest pain for evaluating coronary anatomy and pathology for planning the modalities of treatment. This study design was done to demonstrate the details of angiographic findings including vessels involvement & lesions morphology in suspected CAD patients. The prevalence of coronary artery disease (CAD) more prevalent (50%) in 50-59 years age group. This finding almost similar with MAK Akanda et al⁶ & Khan AR et al⁷ series. Two hundred patients undergone CAG, among them 52% had chronic stable angina, 35% had history of MI. Khan AR et al study⁷ conducted in Chittagong Medical College Hospital showed chronic stable angina as 22% & history of MI 34%. In our study chronic stable angina was more prevalent, it may be due to early health seeking behavior of people in this region. Literature review about risk factors suggest that, in documented CAD smoking constitute the highest frequency (72%) followed by family history 62%, hypercholesterolemia 60%, HTN 55% & DM 38%. Our study reveals almost similar result except DM, which occupied the 2nd position for risk of CAD. This may due to DM is gradually increasing in our society now a days and play a major role in CAD as mentioned in another study⁸.

Figure 4 showing CAG findings constitute 30% had normal coronary arteries, 23% had single vessel disease (SVD), and 29% double vessel disease (DVD), 17% triple vessel disease (TVD) and diffuse lesions in 1%. This finding is almost similar to an international study done by Johansen, AH et al⁹. According to CAG finding finally 31% patients were advised for medical treatment, 52% for PTCA & 17% for CABG. Carasso S, Markiewicz W¹⁰ finding revealed that, among 160 cases of stable angina pectoris 55% were advised for PTCA, 33% for both medical and surgical treatment. In our study procedural success was 100% and no complication was noted. This finding is similar to other studies done in our national level^{6,7}.

CONCLUSION

Though further study is needed in large scale but it can be concluded that CAG can safely be done in this newly set-up cath-lab of NEMCH, Sylhet. It is also seen that there is significant number of normal CAG inspite of clinically documented IHD.

LIMITATION OF THE STUDY

This was a small scale study & does not represent the whole population. So, a large scale study is warranted

for evaluating the safety & overall finding of CAG.

ACKNOWLEDGEMENTS

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REFERENCES

1. Epstein FH, Ostrander LD Jr, Johnson BC. Epidemiological studies of cardiovascular disease in a total community: Tecumseh, Michigan. *Ann Intern Med.* 1965; 62:1170-1187.
2. Kannel WB, Dawber TR, Kagan A, Revotskie N, Stokes J. Factors of risk in the development of coronary heart disease: six-year follow-up experience: the Framingham Study. *Ann Intern Med.* 1961; 55:33-50.
3. Dorel S, Baim & William Grossman. Coronary angiography. Grossman's cardiac catheterization, angiography & intervention. 6th ed. Lipincott Williams & Wilkins, 2006: pp.221-250.
4. Luchi RJ, Scott SM, Dupree RH. Comparison of medical & surgical therapy for unstable angina pectoris. *N Engl J Med.* 1987; 316: 977-984.
5. Frier B.M, Fisher M. Diabetes mellitus. In: Boon NA, Colledge NR, Walker BR, editors. Davidson's principles and practice of medicine. 20th ed. Edinburgh: Churchill Livingstone; 2006: pp.580-816.
6. Akanda MAK, Ali MA, S Zaman M. Correlation of major risk factors with severity of angiographically documented coronary artery disease in 55 male patient of Bangladesh. *J Bangladesh Coll Phy Surg.* 1996; 14 (3): 94-97.
7. Khan AR, Morsed M, Al-Mamun SH. Coronary angiographic profile & Procedural Outcome of 50 cases performed in the Cath-lab of Chittagong medical college hospital. *Bangladesh heart journal.* 2008; 23 (1): 38-39.
8. Ahmed K, Rahman S, Afsar MN. A comparative study of Coronary Angiographic (CAG) findings between diabetic and nondiabetic Patients. *North East Medical College Journal.* 2008; 3 (1): 8-12.

9. Johansen AH, Poulsen TS, Hoiland-Carlsen PF. Myocardial perfusion imaging and coronary angiography in patients with known or suspected stable angina pectoris. *Dan Med Bull.* 2001 May; 48(2): 80-3.
10. Carasso S, Markiewicz W. Medical treatment of patients with stable angina pectoris referred for coronary angiography: failure of treatment or failure to treat. *Clin Cardiol.* 2002 Sep; 25 (9): 436-41.



Review Article

Prevalence of Postoperative Wound Infection by MRSA

Syeda Rafiqen Nessa¹, Taufiq Elahi Chowdhury², Mohammed Sakir Ahmed³, Kazi Zana Alam⁴

¹Assistant Professor, Department of Microbiology, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Assistant Registrar, Department of Surgery, Sylhet MAG Osmani Medical College Hospital, Sylhet

³Resident, Department of Cardiology, Sylhet MAG Osmani Medical College Hospital, Sylhet

⁴Resident Surgeon, Sylhet MAG Osmani Medical College Hospital, Sylhet

ABSTRACT

Surgical site infection (SSI) is a serious complication of hospitalization, occurring in 2%-5% of patients who undergo surgery. Staphylococcus aureus is the most common cause of hospital acquired wound infections, most of the stains are methicillin resistant posing one of the greatest challenges for modern antimicrobial therapy. Infections caused by MRSA can be expensive in terms of antibiotic therapy, isolation facilities and materials and length of hospital stay. According to a World Health Organization literature, the global financial burden because of MRSA infection has been worked out to be \$20,000 to \$ 114,000 for outbreaks and from \$28,000 to \$1600,000 for endemic infections per year. Prompt diagnosis of MRSA infection is important for patients, health care providers and for epidemiological purposes. Surveillance of MRSA related infections especially in the hospital set up is required. Furthermore evolving changes in drug resistance in various communities have forced the importance to reassessment of local empiric choices of antibiotic prophylaxis. The causative agent and spectrum of its antimicrobial susceptibilities of surgical site infections according to local epidemiology is essential, since this spectrum may vary among geographical locations, hospitals and also in different age groups, each institution should carefully plan their antibiotic prophylaxis. Otherwise multi drug resistant pathogens will travel not only locally but also globally and newly introduced pathogens will spread rapidly in susceptible host. For better decision, physicians need more information about local susceptibility pattern of microorganism causing surgical site infections. This review article may be helpful in planning empirical antibiotic prophylaxis in elective surgical patients to prevent surgical site infections.

Key words: Surgical site infection, MRSA, HCAs.

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INTRODUCTION

Despite recent advances in the operative techniques and better understanding of the pathogenesis of wound infections, infection control strategies, postoperative wound infections continues to be a major source of morbidity and mortality for patients undergoing operative procedures. Infections that occur in the wound created by an invasive surgical procedure are

generally referred to as surgical site infections (SSIs). SSIs accounted for 16% of all hospital acquired infections, making them the third most frequent type of nosocomial infections in developed countries. The rates were higher in developing countries, and vary from 7% to 40%. The infections also contribute greatly to the economic costs of surgical procedures and the estimated range is 1.47 to 19.1 billion Euro. This is a great problem, especially in resource poor countries^{1,2}. Infection of surgical wound is considered as nosocomial infection if it occurs within 30 days after surgery, or within one year in case of implant. Infection is defined as discharge of pus from the wound, or a clinical suspicion of wound infection, based on

Address of Correspondence: Dr Syeda Rafiqen Nessa, Assistant Professor, Department of Microbiology, Jalalabad Ragib-Rabeya Medical College, Sylhet-3100. Mobile : 01718-047580; E-mail : srnessa0101@gmail.com

inflammatory signs such as raised temperature, redness and tenderness of the wound. Surgical wound infections are prolonging the duration of hospitalization, increase the risk of poor cicatrization and increase morbidity and mortality.

Staphylococcus aureus has been recognized for a long time as one of the leading causes of hospital infections all over the world. Most of its strains are opportunistic pathogens that can colonise individuals, without symptoms, for either short or extended period of time, causing disease when the immune system becomes compromised².

Before the antibiotic era diseases caused by *Staphylococcus aureus* had high mortality rates. The introduction of benzyl penicillin into chemotherapy in the early 1940s found *Staphylococcus aureus* fully susceptible and several of the first successes of penicillin therapy were related in the cure of formerly untreatable staphylococcal diseases. But in 1950s, the number of *Staphylococcus aureus* clinical isolates with resistance to penicillin increased rapidly.

Methicillin, originally called celbenine, is a semisynthetic derivative of penicillin chemically modified to withstand the degradative action of penicillinase². The drug was introduced into the therapy in Europe in 1959 for combating against hospital strains of penicillinase producing *Staphylococcus aureus*. However, resistance to methicillin was noted shortly thereafter. Methicillin resistance in *Staphylococcus aureus* isolates first appeared in the 1960s. Today the major nosocomial pathogen worldwide is Methicillin resistant *Staphylococcus aureus* (MRSA). Recent surveillance studies in hospitals in various parts of the world indicate a varying incidence of MRSA strains depending on the country and the hospital.

MRSA do not generally appear to be more virulent than sensitive strains but, because of their resistance patterns, they are more difficult to treat if infection occurs. Increased frequency of MRSA in hospitalized patients and possibility of vancomycin resistance requires rapid and reliable characterization of isolates and control of MRSA spread in the hospital².

Surgical site infection (SSI):

Definitions:

Superficial incisional SSI: Infection involves only skin and subcutaneous tissue of incision.

Deep incisional SSI: Infection involves deep tissues, such as fascial and muscle layers. This also includes infection involving both superficial and deep incision

sites and organ/space SSI draining through incision.

Organ/space SSI: Infection involves any part of the anatomy in organs and spaces other than the incision, which was opened or manipulated during operation.

Superficial incisional SSI is more common than deep incisional SSI and organ/space SSI. Superficial incisional SSI accounts for more than half of all SSIs for all categories of surgery³.

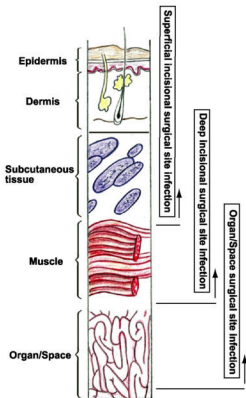


Figure-1: Definitions of surgical site infection (SSI)³

Physical

According to a report by the NNIS⁴ program, surgical site infections are defined as follows:

Superficial incisional SSI

Occurs within 30 days after the operation.
Involves only the skin or subcutaneous tissue.

At least one of the following:

- * Purulent drainage is present (culture documentation not required).

- * Organisms are isolated from fluid/tissue of the superficial incision.
- * At least one sign of inflammation (e.g. pain or tenderness, induration, erythema, local warmth of the wound) is present.
- * The wound that is deliberately opened by the surgeon.
- * The surgeon or clinician declares the wound is infected.

Note: A wound is not considered a superficial incisional SSI if a stitch abscess is present; if the infection is at an episiotomy, a circumcision site, or a burn wound; or if the SSI extends into fascia or muscle³.

Deep incisional SSI

Occurs within 30 days of the operation or within 1 year if an implant is present.

Involves deep soft tissues (eg, fascia and/or muscle) of the incision.

At least one of the following:

- * Purulent drainage is present from the deep incision but without organ/space involvement.
- * Fascial dehiscence or fascia is deliberately separated by the surgeon because of signs of inflammation.
- * A deep abscess is identified by direct examination or during reoperation, by histopathology or by radiologic examination.
- * The surgeon or clinician declares that a deep incisional infection is present³.

Organ/space SSI

Occurs within 30 days of the operation or within 1 year if an implant is present.

Involves anatomical structures not opened or manipulated during the operation.

At least 1 of the following:

- * Purulent drainage is present from a drain placed by a stab wound into the organ/space.
- * Organisms are isolated from the organ/space by aseptic culturing technique.
- * An abscess in the organ/space is identified by direct examination, during reoperation or by histopathologic or radiologic examination.
- * A diagnosis of organ/space SSI is made by the surgeon or clinician³.

SSIs are one of the most important causes of healthcare-associated infections (HCAIs). A prevalence survey undertaken in 2006 suggested that approximately 8% of patients in hospital in the UK have an HCAI.

SSIs are associated with considerable morbidity and it

has been reported that over one-third of postoperative deaths are related, at least in part, to SSI⁵. However, it is important to recognise that SSIs can range from a relatively trivial wound discharge with no other complications to a life-threatening condition. Other clinical outcomes of SSIs include poor scars that are cosmetically unacceptable, such as those that are spreading, hypertrophic or keloid, persistent pain and itching, restriction of movement, particularly when over joints and a significant impact on emotional wellbeing⁶.

SSI can double the length of hospital stays of a patient and thereby increase the costs of health care. The main additional costs are related to re-operation, extra nursing care and interventions, and drug treatment costs. The indirect costs, due to loss of productivity, patient dissatisfaction and litigation, and reduced quality of life, have been studied less extensively.

Epidemiology of postoperative wound infection

Surgical site infections are not an extinct entity; they account for 14.9% of all infections reported during 1990 through 1992 by the 80 NNIS system hospitals in the United States⁶.

Internationally, the frequency of SSI is difficult to monitor because criteria for diagnosis might not be standardized. A survey sponsored by the World Health Organization demonstrated a prevalence of nosocomial infections varying from 3-21%, with wound infections accounting for 5-34% of the total⁷. The 2002 survey report by the Nosocomial Infection National Surveillance Service (NINSS)⁸, which covers the period between October 1997 and September 2001, indicates that the incidence of hospital acquired infection related to surgical wounds in the United Kingdom is as high as 10% and costs the National Health Service in the United Kingdom approximately 1 billion pounds (1.8 billion dollars) annually.

Causes

All surgical wounds are contaminated by microbes, but in most cases, infection does not develop because innate host defenses are quite efficient in the elimination of contaminants. A complex interplay between host, microbial, and surgical factors ultimately determines the prevention or establishment of a wound infection.

Microbiology

Microbial factors that influence the establishment of a wound infection are the bacterial inoculum, virulence, and the effect of the microenvironment. When these

microbial factors are conducive, impaired host defenses set the stage for enacting the chain of events that produce wound infection.

Most SSIs are contaminated by the patient's own endogenous flora, which are present on the skin, mucous membranes, or hollow viscera. The traditional microbial concentration quoted as being highly associated with SSIs is that of bacterial counts higher than 10,000 organisms per gram of tissue (or in the case of burned sites, organisms per cm² of wound)³.

Gram-positive organisms, particularly *staphylococci* and *streptococci*, account for most exogenous flora involved in SSIs. Sources of such pathogens include surgical/hospital personnel and intraoperative circumstances, including surgical instruments, articles brought into the operative field, and the operating room air.

The most common group of bacteria responsible for SSIs are *Staphylococcus aureus*. Methicillin resistant *Staphylococcus aureus* (MRSA) is proving to be the scourge of modern day surgery. Problems arise in the treatment of overt infections with MRSA because antibiotic choice is very limited. MRSA infections appear to be increasing in frequency and are displaying resistance to a wider range of antibiotics.

Of particular concern are the vancomycin intermediate *Staphylococcus aureus* (VISA) strains of MRSA. These strains are beginning to develop resistance to vancomycin, which is currently the most effective antibiotic against MRSA. This new resistance has arisen because another species of bacteria, called *enterococci*, relatively commonly express vancomycin resistance³.

Table-1: Pathogens commonly associated with wound infections and frequency of occurrence³

Pathogen	Percent
<i>Staphylococcus aureus</i>	20
Coagulase-negative staphylococci	14
Enterococci	12
<i>Escherichia coli</i>	8
<i>Pseudomonas aeruginosa</i>	8
<i>Enterobacter species</i>	7
<i>Proteus mirabilis</i>	3
<i>Klebsiella pneumoniae</i>	3
Other streptococci	3
<i>Candida albicans</i>	3
Group D streptococci	2
Other gram-positive aerobes	2
<i>Bacteroides fragilis</i>	

Mechanisms of Resistance to Antimicrobial Agents:

Penicillin is inactivated by β -lactamase, a serine protease that hydrolyzes the β -lactam ring. Less than 5 percent of isolates remain sensitive to penicillin. Resistance to methicillin confers resistance to all penicillinase-resistant penicillins and cephalosporins. This high level of resistance requires the presence of the *mec* gene that encodes penicillin-binding protein 2a. The *mec* genes probably originated from a different species of *Staphylococci*. Although many methicillin-resistant strains appear to be descendants of a limited number of clones, some appear to be multiclonal in origin, suggesting the horizontal transfer of *mec* DNA. Other staphylococcal genes, including *bla* (for-lactamase) and *fem* (for factors essential for methicillin resistance), affect the expression of resistance. The expression of resistance to methicillin is often heterogeneous, and the percentage of a bacterial population that expresses the resistance phenotype varies according to the environmental conditions. Antimicrobial-sensitivity testing has been modified to enhance the detection of the resistance phenotype⁹. There has been increasing concern about the possible emergence of vancomycin-resistant *S. aureus* strains. Resistance to vancomycin has been reported in clinical isolates of *S. haemolyticus*, a coagulase-negative species. The enterococcal plasmid-bearing gene for resistance to vancomycin has been transferred by conjugation to *S. aureus* in vitro. Four recent case reports (one from Japan and three from the United States) have documented the isolation of clinical strains with intermediate sensitivity to vancomycin (minimal inhibitory concentration, 8 mg per milliliter). The mechanism of resistance in these isolates is not known but is not due to the *van* genes present in *enterococci*. Both increased cell-wall synthesis and alterations in the cell wall that prevent vancomycin from reaching sites of cell-wall synthesis have been suggested as mechanisms⁹.

MRSA Infection Diagnosis:

To ascertain the correct antimicrobial therapy for treatment of staphylococcal infections, it is imperative that methicillin resistance is detected as rapidly as possible. Most screening is still carried out using plate-based methods, but alternative methods including broth culture, chromogenic media, rapid screening kits, molecular assays and automated systems are increasingly being used.

Classical selective media that have been employed variously contain inhibitors such as NaCl and/or antibiotics to aid selection, with a pH indicator to

highlight presumptive colonies. Examples include: mannitol salt agar containing 7% NaCl with either 4mg/L methicillin or 2mg/L oxacillin; "oxacillin-resistant screening agar base" with 5.5% NaCl and 2 mg/L oxacillin; Baird Parker medium with 8mg/L ciprofloxacin; and Mueller-Hinton agar with 4% NaCl and 6 mg/L oxacillin¹⁰. Growth after 24 hours incubation is variable and incubation for 48 hr. is often required for an accurate result.

The definitive laboratory studies to diagnose that a person is infected with MRSA are straightforward. *S. aureus* is isolated and identified from the patient by standard microbiological techniques (growth on Baird-Parker agar plates and a positive coagulase test). The coagulase test is a laboratory test based upon the ability of *S. aureus* to produce the enzyme coagulase, that ultimately leads to the formation of a blood clot. After *S. aureus* bacteria are isolated, the bacteria are then cultured in the presence of methicillin (and usually other antibiotics). If *S. aureus* grows in the presence of methicillin, the bacteria are termed MRSA¹¹.

A number of more specific molecular methods have been developed, offering rapid detection of MRSA. Many of these use multiplexed PCR primers to detect genes specific to *S. aureus* (for example *nuc*, *fem* and *coa*) together with the *mecA* gene for methicillin resistance. CytAMP® (British BioCell International) detects *mecA* in combination with *coa* (coagulase) using signal-mediated amplification of RNA technology¹². IDI-MRSA (Cepheid) is a multiplex, qualitative real-time PCR assay for direct detection of MRSA from swabs. It is sensitive and specific for the rapid detection of colonization by MRSA, but cultures may still be needed to confirm positive results¹³.

Multiplex PCR systems have also been developed for rapid detection of MRSA isolates carrying the PVL genes, with discrimination between MRSA and methicillin-susceptible *S. aureus* (MSSA) by simultaneous amplification of *mecA*, *nuc* and *pvl* gene,^{14,15} and SCCmec typing¹⁶. Such methods may be important in rapid detection of CA-MRSA and to follow the local epidemiology of MRSA.

MRSA Infection Treatment:

The standard treatments for serious methicillin-resistant staphylococcal infections, such as septicemia, are the glycopeptides, vancomycin or teicoplanin. Vancomycin should be preferred to teicoplanin for infections due to CONS owing to superior in vitro activity. Other alternative antibiotics for MRSA infections depend predominantly on the site of infection and on the antimicrobial sensitivity of the

particular microorganism. Traditional alternatives to the glycopeptides may include fusidic acid, rifampicin and the tetracyclines, used in various combinations or with a glycopeptide¹¹.

MRSA Infection Prevention:

The best way to avoid MRSA infection is not making direct contact with skin, clothing, or any items that come in contact with either MRSA patients or MRSA. This is often not possible because MRSA-infected individuals or MRSA carriers are not immediately identifiable. A first step is excellent hygiene practices (for example, hand washing with soap after personal contact or toilet use, washing clothes potentially in contact with MRSA patients or carriers, and using disposable items such as gloves when treating MRSA (MRSA patients))¹¹. Another prevention method is to treat and cover (for example, antiseptic cream and a Band-Aid) any skin breaks.

CONCLUSION

Vancomycin and imipenem are very costly antibiotics and it would not be always possible to use by the people of poor resource country like Bangladesh. On the other hand the most commonly used and cheaper antibiotics were resistant to MRSA. So, if we have to fight against MRSA we have to concentrate more on preventing the MRSA infection. For this reason we have to adopt strict principles of asepsis, need to apply barrier nursing in all steps of patients care and also regular surveillance of SSIs including monitoring antibiotic sensitivity pattern of MRSA and formulation of definite antibiotic policy may be helpful for reducing the incidence of MRSA infection. Otherwise we will face a major therapeutic challenge in the future.

REFERENCES

1. Leaper DJ, Van Goor H, Reilly J, Petrosillo N, Geiss HK, Torres AJ, et al. Surgical site infection; European perspective of incidence and economic burden. *Int Wound J* 2004; 1(4): 247-73.
2. Šiširak M, Zvizdi A, Huki M. Methicillin resistant *Staphylococcus aureus* (MRSA) as a cause of nosocomial wound infections. *Bosn J Basic Med Sci* 2010; 10(1): 32-7.
3. Singhal H. Wound Infection. eMedicine [online]. Last up dated Jan 6, 2012. Retrieved from: <http://emedicine.medscape.com/article/188988>. Accessed on 9 March 2012.
4. Emori TG, Gaynes RP. An overview of nosocomial infections, including the role of the microbiology

- laboratory. *Clin Microbiol Rev* 1993; 6(4): 428-42.
5. Astagneau P, Rioux C, Golliot F, Brückner G. Morbidity and mortality associated with surgical site infections: results from the 1997-1999 INCISO surveillance. *J Hosp Infect* 2001; 48(4): 267-74.
 6. Bayat A, McGrouther DA, Ferguson MW. Skin scarring. *BMJ* 2003; 326(7380): 88-92.
 7. Mayon-White RT, Duclou G, Kereselidze T, Tikomirov E. An international survey of the prevalence of hospital-acquired infection. *J Hosp Infect* 1988; 11(Suppl A): 43-8.
 8. National Nosocomial Infections Surveillance System. National Nosocomial Infections Surveillance (NNIS) system report, data summary from January 1992 to June 2002, issued August 2002. *Am J Infect Control* 2002; 30(8): 458-75.
 9. Lowy FD. Staphylococcus aureus infections. *N Engl J Med* 1998; 339(8): 520-32.
 10. Casey AL, Lambert PA, Elliott TS. Staphylococci. *Int J Antimicrob Agents* 2007; 29 (Suppl 3): S23-32.
 11. Davis C. MRSA Infection (Methicillin-Resistant Staphylococcus aureus) Infection. Last Editorial Review: 3/7/2011. Retrieved from: http://www.emedicinehealth.com/mrsa_infection/article_em.htm. Accessed on 9 March 2012.
 12. Levi K, Bailey C, Bennett A, Marsh P, Cardy DL, Towner KJ. Evaluation of an isothermal signal amplification method for rapid detection of methicillin-resistant Staphylococcus aureus from patient-screening swabs. *J Clin Microbiol.* 2003; 41(7): 3187-91.
 13. Desjardins M, Guibord C, Lalonde B, Toye B, Ramotar K. Evaluation of the IDI-MRSA assay for detection of methicillin-resistant staphylococcus aureus from nasal and rectal specimens pooled in a selective broth. *J Clin Microbiol* 2006; 44(4): 1219-23.
 14. McDonald RR, Antonishyn NA, Hansen T, Snook LA, Nagle E, Mulvey MR, et al. Development of a triplex real-time PCR assay for detection of Pantone-Valentine leukocidin toxin genes in clinical isolates of methicillin-resistant staphylococcus aureus. *J Clin Microbiol.* 2005; 43(12): 6147-9.
 15. McClure JA, Conly JM, Lau V, Elsayed S, Louie T, Hutchins W, et al. Novel multiplex PCR assay for detection of the staphylococcal virulence marker Pantone-Valentine leukocidin genes and simultaneous discrimination of methicillin-susceptible from-resistant staphylococci. *J Clin Microbiol* 2006; 44(3): 1141-4.
 16. Zhang K, McClure JA, Elsayed S, Louie T, Conly JM. Novel multiplex PCR assay for characterization and concomitant subtyping of staphylococcal cassette chromosome mec types I to V in methicillin-resistant Staphylococcus aureus. *J Clin Microbiol* 2005; 43(10): 5026-33.



Case Report

Intraabdominal Lymphangioma (Mesenteric Lymphangioma): A Case Report

Parveen Aktar¹, Md Nazmul Islam², Apurba Kishore Paul³, Nurul Quayum Mohammad Musallin⁴, Abdul Mukaddem Mashud⁵

^{1,4}Assistant Professor, Department of Surgery, Jalalabad Ragib-Rabeya Medical College, Sylhet

²Professor, Department of Surgery, Jalalabad Ragib-Rabeya Medical College, Sylhet

³Registrar, Department of Surgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

⁵Assistant Registrar, Department of Surgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

ABSTRACT

A diagnostic dilemma arose when a 28 years old woman reported with pain in right hypochondrium and painless mass in lower abdomen. Clinical examination and investigations showed a case of chronic calculous cholecystitis with a gut related mass which compressed the lower descending colon from outside. At operation, cholecystectomy and excision of mesenteric growth along with resection of affected small gut and end to end anastomosis was carried out. The aim of reporting this case was to show whether their is any relation of cholelithiasis with that rare pathology of mesenteric lymphangioma both are or is a separate entities or a coincidental chronic calculous cholecystitis and it is a congenital malformations that was expressed in adult age.

Key words: Mesenteric lymphangioma, Intra abdominal lymphangioma.

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INTRODUCTION

Lymphangiomas are benign lesions of vascular origin that show lymphatic differentiation. These occur in many anatomic locations and may have a paediatric or adult clinical presentation. Most (95%) occur in the neck and axillary regions, the remaining 5% are located in the mesentery, retroperitoneum, abdominal viscera, lung, and mediastinum¹. However, intra abdominal lymphangiomas are rare, usually slow growing and manifest in early adulthood². The clinical signs and symptoms are nonspecific. A significant proportion of these patients present with acute surgical abdomen requiring emergency surgery³. We present a case of intraabdominal lymphangioma in an adult with chronic calculous cholecystitis which developed rather early and presented as a diagnostic dilemma.

CASE REPORT

A 28 year old female presented with right upper abdominal pain for 5 years and lower abdominal lump for 3 months. Her presenting complaints started 5 years back since then she felt recurrent pain in the right upper abdomen which was colicky in nature and had radiation to the back, associated with bouts of vomiting. Pain was aggravated by taking fatty food. Three months back, she noticed a painless lower abdominal mass. She had no complaints about her bowel and bladder habit or of any weight loss or rise of temperature. She was amenorrhoeic for 3 months after taking inj. Depot provera. On examination, her vital status was within normal limit. Abdominal examination revealed: a palpable rounded mass felt in the left lower abdomen extending from umbilicus to left iliac fossa, measuring about 12x10 cm, non tender with ill defined margin, firm in consistency and freely mobile, there was no visible peristalsis or any sign of gut obstruction. Ultrasonography of abdomen showed cholelithiasis and gut related mass or high up ovarian mass. Barium enema showed segmental narrowing of the lower descending colon but no filling defect or mucosal

Address of Correspondence: Dr Parveen Aktar, Assistant Professor Department of Surgery, Jalalabad Ragib-Rabeya Medical College, Sylhet-3100. Mobile : 0 1 7 1 1 4 6 1 6 3 1 ; E - mail: aktar.parveen@yahoo.com

irregularity was seen; probably due to compression of the contrast filled bowel loops from outside the colon, intraperitoneal mass other than bowel mass. An exploratory laparotomy through midline incision revealed a large ovoid mass in the mesentery which was about 10x8 cm, dusky red, freely mobile, involving the small gut. No lymphadenopathy or ascites were detected. The growth involved the small gut, 30 cm distal to the D-J junction. Other organs of abdomen like liver, spleen, and colon were healthy. Complete excision of involved small gut along with the growth was done and end to end anastomosis carried out. Then cholecystectomy was done. Wound closed in layers with keeping a drain tube in place. Resected specimen was sent for histopathological examination in which the tumour revealed mesenteric lymphangioma and features of chronic cholecystitis in the gall bladder. Patient resumed oral feeding on third post-operative day and drain tube was removed on 5th postoperative day. Patient's recovery was uneventful, and discharged on 8th post operative day after removal of stitches with necessary advices.



Figure-1: Intra operative picture of mesenteric lymphangioma

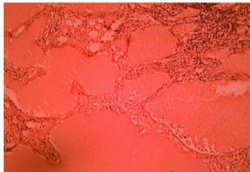


Figure-2: Histopathology shows multiple cystic spaces lined by flat low lying epithelium with surrounding scattered lymphoid cells.

DISCUSSION

Intraabdominal cystic lymphangioma of the mesentery is a rare congenital lesion with a relatively low growth potential⁴. Benign mesenteric lymphangiomas are thought to account for 1 in 1,00,000 to 1 in 250,000 of hospital admission^{5,6}. It is typically found in young adults and presents with chronic features which correspond with our patient's age. Abdominal lymphangiomas are reported to occur most commonly in the mesentery, followed by the omentum, mesocolon and retroperitoneum⁷. Many patients with mesenteric lymphangioma eventually present with symptoms based on the location and size of the cyst. The clinical spectrum includes abdominal pain, nausea, vomiting or change in bowel habits⁸. These benign lesions usually present as painless abdominal distension or with complications like volvulus, infection, intracystic haemorrhage and intestinal obstruction⁹. In our case there was only asymptomatic lower abdominal mass which was noticed for 3 months. The preoperative diagnosis is difficult without a high index of suspicion¹². Laparotomy may be necessary if signs of an acute abdomen develop. In rare circumstances, the cyst can expand considerably and present as a gigantic mass in the abdomen¹⁰. Plain abdominal films may identify displacement of small bowel loops by a soft tissue mass. This test is not diagnostic but does exclude simple intestinal obstruction¹¹. Radiological investigations using ultrasound or computed tomography (CT) usually confirms the presence of a mass and may help to exclude other causes of intraabdominal masses but is usually insufficient to provide a definite diagnosis. MRI has also been proposed as a suitable investigation for defining the origins of the mass and thus aid diagnosis¹⁰. In our case ultrasonography made some confusion towards gut related mass or high up left ovarian mass. Barium enema X-ray also showed segmental narrowing of gut. However surgical exploration made the conclusion of the origin of mass. The diagnosis of mesenteric lymphangioma is usually made histologically following surgical resection of the cyst. There are no blood tests which confirm the diagnosis. However, Jain et al report using multi-slice spiral CT confidently to establish a diagnosis prior to surgery¹². Cystic lymphangioma can be enucleated completely without compromising the bowel vascularity. However, in cases of infiltrative lymphangioma, resection of the involved part of the mesentery along with the bowel is recommended. Laparoscopic excision of mesenteric cystic lymphangioma has the advantages of more detailed dissection of mesenteric vessels, lower

vascular trauma, less postoperative pain, and shorter hospitalization¹³. Partial excision or marsupialization is associated with instillation of sclerosing factors within the cystic cavity with agents such as alcohol, dextrose, bleomycin, or protein, has not been used sufficiently to allow definitive conclusions to justify its use¹⁴. However, there has been some success with factor OK-432, a protein. A common postoperative complication is persistent lymphatic leak with or without infection. Other modalities of treatment for unresectable intraabdominal lymphangiomas include sclerotherapy with doxycycline or alcohol^{15,16}. Our patient's surgery was well-tolerated, and she was discharged without complications or further incidents.

CONCLUSION

Lymphangiomas are uncommon benign lymphatic lesions that may occur virtually at any anatomic location in the abdomen. Knowledge of the imaging and pathologic spectrum of abdominal lymphangiomas is necessary when evaluating pediatric and adult patients with intra abdominal cystic masses. It is inconclusive in a single case, wide spectrum of evaluation of cases is required to see any correlation with concomitant pathologies.

REFERENCES

- Lugo-Olivieri CH, Taylor GA. CT differentiation of large abdominal lymphangioma from ascites. *Pediatr Radiol* 1993; 23: 129-30.
- Fernandez HI, Bregante J, Mulet JFJ, Moron CJM. Abdominal cystic lymphangioma. *Cir Pediatr* 1998; 11: 171-3.
- Okur H, Ozokutan BH, Durak AC, Kazez A and Ozkan K. Mesenteric, omental and retroperitoneal cysts in children. *Eur J Surg* 1997; 163: 673-7.
- De Perrot M, Rostan O, Morel P, Le Coultre C. Abdominal Lymphangioma in adults & children. *Br J Surg* 1998; 85: 395-7.
- Chim H, Chuwa E, Chau Y-P, Chow PK. Gastrointestinal: Mesenteric cystic lymphangioma. *J Gastroenterol Hepatol* 2006, 21: 916. PubMed.
- Campbell WJ, Irwin ST, Biggart JD. Benign lymphangioma of the jejunal mesentery : an unusual cause of small bowel obstruction. *Gut* 1991, 32: 1568. PubMed.
- Ko SF, Ng SH, Shieh CS, Lin JW, Huang CC, Lee TY. Mesenteric cystic lymphangioma with myxoid degeneration: unusual CT and MR manifestations. *Pediatr Radiol* 1995; 25: 525-7.
- Takiff H, Calabria R, Yin L, Stabile BE. Mesenteric cysts and intra-abdominal cystic lymphangiomas. *Arch Surg*. 1985; 120: 1266-9.
- Merrot T, Chaumoitre K, Simeoni-Alias J. Abdominal lymphangiomas in children. Clinical, diagnostic and therapeutic aspects: apropos of 21 cases. *Ann Chir* 1999; 53: 494-9.
- Losanoff EJ, Richman BW, El-Sherif A, Rider DK, Jones WJ. Mesenteric Cystic Lymphangioma. *J Am Coll Surg* 2003, 196(4): 598-603.
- Konen O, Rathaus V, Dlugy E, Freud E, Kessler A, Shapiro M. et al. Childhood abdominal cystic lymphangioma. *Pediatr Radiol* 2002; 32: 88-94.
- Jain S, Upreti L, Bhargava SK, Gupta R, Gupta PK. Mesenteric Lymphangioma: diagnosis by multislice spiral CT[Letter]. *Indian J Radiol Imaging* 2002, 12: 580-2.
- Vara-Thobeck C, Toscano MR, Herriaz HR. Laparoscopic resection of a giant mesenteric cystic lymphangioma. *Eur J Surg* 1997; 163: 395-6.
- Luzzatto C, Midrio P, Tschapirassian Z, Guglielmi M. Sclerosing treatment of lymphangiomas with OK-432. *Arch Dis Child* 200; 82: 316-8.
- Mabrut JY, Grandjean JP, Henry L. Mesenteric and mesocolic cystic lymphangiomas. Diagnostic and therapeutic management. *Ann Chir* 2002; 127: 343-9.
- Stein M, Hsu RK, Schneider PD. Alcohol ablation of a mesenteric lymphangioma. *J Vasc Interv Radiol* 2000; 11: 247-50.



Case Report

Wilson Disease: A Case Report

Mst Ruzina Rahman¹, Naznin Akther², Afroza Jinnat³, Ashith Chandra Das⁴, Archana Deb⁵, Md Tarek Azad⁶

^{1,2,3,4}Indoor Medical Officer, Department of Paediatrics, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet

⁵Assistant Professor, Department of Paediatrics, Jalalabad Ragib-Rabeya Medical College, Sylhet

⁶Associate Professor, Department of Paediatrics, Jalalabad Ragib-Rabeya Medical College, Sylhet

ABSTRACT

Wilson Disease is a rare autosomal recessive disorder of copper metabolism having wide spectrum of clinical manifestations. Affected children may be entirely asymptomatic or may present with various hepatic, neurogenic or renal manifestations. This work reports a case of 10 years old boy presented with acute hepatic failure, but there was no neurological involvement.

Key words: Copper metabolism, K-F ring, Ceruloplasmin.

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INTRODUCTION

Wilson disease is a disorder of copper metabolism in which copper accumulate in tissues leading to liver disease, degenerative changes in brain and Kayser-Fleischer ring in the cornea¹. The condition is due to mutation of gene, localized to the long arm of chromosome 13 (13q14.3) that encodes a copper transporting P-type ATPase, ATP7B¹. Absence or malfunction of ATP7B, result in accumulation of copper in liver cells and subsequently to other tissues¹. Disease manifestations are due to toxic effects of copper in various organs¹. It affects different system at different age. The younger the patient, the more likely hepatic involvement will be the predominant manifestation. Various hepatic forms like acute hepatitis, chronic hepatitis, acute fulminant hepatic failure, cirrhosis of liver can occur in early childhood². After about 20 years, neurological symptoms predominate¹. Disease presentations are variable, with a tendency to familial patterns. The incidence is 1/5,00,000 to 1/1,00,000 live births¹. There is no male female preponderance. Wilson disease is named after Samuel Alexander Kinnier Wilson (1878-1937), the British neurologist who first described the condition in 1912.

Address of Correspondence: Dr Ruzina Rahman, Indoor Medical Officer, Department of Paediatrics, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet-3100

CASE REPORT

A 10 years old boy (Figure-1), 3rd issue of consanguineous parents got admitted in Jalalabad Ragib-Rabeya Medical College Hospital, on 11th December 2011 with the complaints of yellow discoloration of skin and mucous membrane associated with anorexia and nausea for last 4 months, abdominal distention and constipation for 15 days. The patient had no history of abnormal movement or speech difficulty. No history of haematemesis, melena, haemoptysis and blood transfusion in the past. He did not take any offending drugs or had no history of contact with TB patient and neither had any positive family history regarding to the disease.

On examination, the boy was conscious, mildly pale, deeply icteric, oedema was present in both lower limbs, vital signs were normal, anthropometric measurement was WAZ= -1.5SD, WHZ= -2.3SD, HAZ= -2.7SD. On systemic examination ascites and mild hepatomegaly but no splenomegaly found. Nervous system examination revealed normal. On slit lamp examination K-F ring was found in both eyes. His other sibs were also examined for K-F ring and among rest 4 sibs, 2 were found having K-F ring in eyes.

A clinical diagnosis of acute liver failure due to Wilson disease was made. Biochemical and serological laboratory investigations finalized the diagnosis. On laboratory investigations we found, TC-8.7×10⁹/L,

Hb%- 9.9g/dl, platelets count- $160 \times 10^9/L$, S.bilirubin 5.7mg/dl, SGPT 140 IU/L, PT was 30 seconds, serum albumin 3gm/dl, serum ceruloplasmin 10 mg/L, serum electrolytes were normal.



Figure-1: Boy presented with Wilson disease

The patient was managed by both supportive and specific treatment in our hospital. Salt and water were restricted. As oedema was not improved and serum albumin was decreased, injection 20% albumin given. Low copper diet advised. For regular bowel habit lactulose was given. As specific treatment D-penicillamine 10mg/kg was started and gradually dose was increased to 20mg/kg body wt. Oral zinc supplement 75 mg daily was continued. With all these treatment clinical improvement was seen after 13 days of hospital stay. Then the child was discharged with counseling for regular follow up and informed them about the side effects of D-penicillamine. After 1 month when the patient came to us, there was expected clinical improvement.

DISCUSSION

Wilson disease is also known as hepatolenticular degeneration as it mainly involve liver and brain¹. The precise mechanism of Wilson disease is not known but

basic mechanism relates to decrease excretion of biliary copper due to lysosomal defect in liver cells. There is diffuse accumulation of copper in cytosol of hepatocytes. When liver cells become overloaded, copper is redistributed to other tissues like brain, cornea and kidneys. More than 250 mutations have been identified. Due to multiple mutation in the gene of Wilson disease, presentations are variable. Milder mutation leads to late manifestation of the disease¹. Acute or fulminant liver failure can be the first manifestation of Wilson disease. Acute or fulminant hepatitis are seen among children and adolescents, and are sometimes accompanied with haemolytic anaemia as a result of circulatory free copper ions that causes haemolysis. This condition, which is usually manifested by haemoglobinuria and renal failure, accounts as one of the indication for urgent liver transplantation³. Neurological involvement usually occurs after 2nd decade but earlier in Indian children⁴. It has insidious onset with intention tremor, dystonia, dysarthria, poor school performance, behavioral changes, in-coordination and neuro-psychiatric manifestations (35%)⁵. Kayser-Fleischer ring is present in 99% of patient presenting with neurogenic form and 30-50% of patient with hepatic form⁴. On slit lamp examination, Kayser- Fleischer ring was found in our patient. On familial screening, it was identified among two sibs out of four. K-F ring is due to copper deposition in descemet layer of cornea and is best seen with slit lamp. This ring may disappear with treatment⁶.

Unusual presentation of Wilson disease include arthritis, cardiomyopathy, endocrinopathy specially hypothyroidism, Sunflower cataract, renal tubular acidosis. Diagnosis of Wilson disease is based on clinical evaluation along with biochemical, neuroimaging and histopathological study of liver specimen. Biochemical studies reveal a low serum ceruloplasmin level ($< 20\text{mg/dl}$) and increased urinary copper excretion ($>100\text{mg/day}$), hepatic copper estimation of more than 250 gm/gm of dry tissue is the most definitive method of diagnosis^{7,8}. In this case serum ceruloplasmin was 10mg/L and urinary copper could not be done. In equivocal cases the response of urinary copper output to chelation may be of diagnostic help¹.

Management strategy for Wilson disease is dietary restriction of copper $<1\text{mg/day}$ ¹. Food such as liver, nut, chocolate, shellfish should be avoided. Chelation therapy is best managed with oral administration of D-Penicillamine in a dose of 20mg/kg/day¹. This drug remove copper from tissue. Toxic effect of D-

Penicillamine occur in 10-20% patient and consist of fever, skin rash, arthralgia, pancytopenia, nephritic syndrome, hypersensitivity reaction (Good Pasture Syndrome, SLE, Polymyocitis), aplastic anaemia¹. Triethyline tetraamine dihydrochloride is alternative chelating agent if child doesn't tolerate D-Penicillamine due to its side effects¹. Ammonium tetrathiomolybdate acts by preventing absorption of copper by gut is under trial for patient with neurological disease but experience in children is limited¹. Zinc 25mg 3 times daily has also been used for maintenance therapy and for treatment of asymptomatic sibs^{1,2}. Treatment of fulminant Wilson hepatitis by plasma exchange, has been reported with diverse outcomes. Most studies could not express any relationship between copper ion removal from circulation and any clinical outcome^{9,10}. Wilson disease is progressive and fatal if untreated, however effective treatment is available and early initiation of therapy is gratifying.

CONCLUSION

Wilson disease is not uncommon in Bangladeshi children. Early diagnosis and family screening may help the patient and family to prevent dreadful outcome.

REFERENCES

1. Carey RG, Balistreri WF. Metabolic Diseases of the Liver. In: Nelson's Textbook of Pediatrics. Eds. Kliegman RM, Behrman RE, Jenson HB, Stanton BF. 18th Ed, Philadelphia, W.B. Saunders Company, 2008; pp. 1677-8.
2. Bhavé Sa, Purohit GM, Pradhan AM, Pandit AN. Hepatic Presentation of Wilson Disease. *Indian Pediatr* 1987; 24: 385-93.
3. Zandman Goddard G, Weiss P, Avidan B, Bar-meir S, Shoenfeld Y. Acute Varicella Infection Heralding Wilsonian Crisis. *Jclin Gastroenterol*.1994; 18(3): 265-6.
4. Ganguli S, Samanta T; Wilson Disease, IAP Teaching Module 2005.
5. Muller T, Koppikar S, Taylor RM, et al. Re-evaluation of the Penicillamine Challenge Test in the Diagnosis of Wilson Disease in Children. *J Hepatol*.2007; 47(2): 270-6.
6. Kalra Veena, Practical Paediatric Neurology 2nd Edition Arya Publication, 2008; 205-6.
7. Von Wassenaer- Van Hall HN, Vanden Henvel AG, Janson GH, Hoogenraad TU, Mali WPTM. Cranial MR in Wilson Disease; Abnormal White Matter in Extra Pyramidal and Pyramidal Tract. *AJNR* 1995; 16: 2021-7.
8. Youssef ME. Wilson Disease. *Mayo Clin Proc* 2003; 78: 1126-36.
9. Lee JJ, Kim HJ, Chung IJ, et al. Acute Hemolytic Crisis with Fulminant Hepatic Failure as the First Manifestation of Wilson Disease: a Case Report. *J Korean Med Sci*.1998; 13(5): 548-50.
10. Scheinberg IH, Sternlieb I. Wilson Disease and the Concentration of Ceruloplasmin in Serum. *Lancet*.1963; 1(7296): 1420-1.



Miscellaneous

Campus News

Postgraduate Training Recognized by BCPS

A high powered inspection team consisted of eight members from Bangladesh College of Physicians and Surgeons (BCPS) Dhaka, headed by Professor Syed Mokarrom Ali, visited the Jalalabad Ragib-Rabeya Medical College and Hospital on 27-12-2010. On the recommendations of the inspection team, the Council of Bangladesh College of Physicians and Surgeons (BCPS) has renewed recognition to the departments of Paediatrics, Ophthalmology, Otolaryngology, Psychiatry, Pathology (Histopathology) and Orthopaedic Surgery for imparting training to the resident doctors provisionally for a period of five years with effect from 21-09-2009. The Council has granted recognition to the department of Paediatric Surgery for imparting training to the resident doctors provisionally for a period of five years with effect from 13-02-2010. The training will be accepted for appearing in the FCPS, MD, MS Part-II and diploma examinations in these specialties. The postgraduate training imparted from the departments of Surgery, Medicine and Obstetrics & Gynaecology were recognized by Bangladesh College of Physicians and Surgeons (BCPS) earlier in 2003.

Programmes

- The 5th indoor games competition was inaugurated on 1st January 2012 by Danobir Mr Ragib Ali, founder chairman of the governing body of Jalalabad Ragib-Rabeya Medical College & Hospital. The principal, teacher incharge of the sports committee, other teachers and students of the college were present on the occasion.
- A high powered inspection team led by Professor Dr AK Azad Chowdhury, Chairman University Grant Commission visited Jalalabad Ragib-Rabeya Medical College & Hospital on the 1st January 2012 and was highly satisfied seeing the huge development of the college and hospital.
- The inaugural class of the students of 18th batch of JRRMC was held on 15th January 2012. Presided over by the principal, the programme was enlightened by Professor Kismatul Ahsan, Vice Chancellor, Leading University as chief guest. Danobir Mr Ragib Ali, Mr Abdul Hye, teachers, students of 18th batch and their guardians were present on the occasion.
- 35th raising day of Shandhani was observed on 7th February 2012 organized by Shandhani JRRMC unit.
- A discussion meeting and Dua Mahfil was observed on 8th February 2012 to observe the Holy Eid-E-Miladunnabi organized by the social welfare committee of JRRMC.
- On 15th February 2012 "Paediatric and Neonatal ICU" was opened in the hospital by Danobir Mr Ragib Ali. This is the first ICU in this area for critical care management of neonates and paediatric patients.
- International Mother Language Day was observed on 21st February 2012.
- Mr Richard Fular Honorable Member of the British Parliament visited the institution and praised Danobir Mr Ragib Ali that he established such an institute that is giving huge service to the poor patients and also creating new doctors who will serve the needy people of this area.
- A new well furnished and well decorated college cafeteria was inaugurated on 7th April 2012 in the new "Twin Tower" building.
- Bengali New Year "The Pohela Baishakh" was observed on 14th April 2012 in the college. A colourful rally, games, traditional baishakhi mela, cultural programme were arranged to celebrate the day.
- Prizes were distributed to the winners of the indoor games competition on 12th June 2012 by Danobir Mr Ragib Ali.

Seminar

The following seminar held in Jalalabad Ragib-Rabeya Medical College (JRRMC) during January 2012 to June 2012:

A seminar on "Breast Feeding: Best Start for Baby" held on 28th June 2012 organized by the department of Paediatrics.



Instructions for Author(s)

Manuscripts on clinical, review, experimental and historical topics pertinent to medical sciences are accepted for the publication in this journal. The papers are accepted for the publication with an understanding that they are solely submitted for this journal. The statements, comments or opinions expressed in the papers are exclusively of author(s), not of editor(s) or publisher. The manuscripts are to be prepared as described in following instructions. 3 (three) hard copies are to be submitted. Letters about potentially acceptable manuscripts will be sent after review process is complete. No manuscripts will be returned if not accepted for publication. In addition an electronic/digital version of the manuscript composed in MS word 98/2000 should be submitted in a diskette.

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Manuscripts should be typewritten, double-spaced throughout (including references and tables) on one side of good quality A4 sized paper, with margins of at least 25 mm. Each component of the manuscript should begin on a new page in the sequence of title or cover page, abstract with key words, text, acknowledgement, references, tables and legends for illustrations.

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An informative abstract not more than 250 words should briefly describe the objectives, materials and methods, results and conclusion. Number of key words should not more than ten and none that are in the title.

Text should contain Introduction, Materials and Methods, Results and Discussion in sequence.

Introduction

It should briefly disclose the purpose of study. It will help the readers with the problem finding. It should be clear in nature and purpose.

Materials and Methods

Clearly it should include materials, experimental procedures, methods etc. Mention the nomenclature, source of material, equipment with manufacturer's

details in parentheses. Describe new methods in sufficient detail indicating their limitation. Established methods should be cited with authentic references. Ethical standards should be followed in reporting experiments done in human subjects. Precisely identify the dosage and route of administration, when drugs or chemicals are used. Measurements and data should be stated in SI unit, or if SI unit does not exist, use an internationally accepted unit. Abbreviations and acronyms should be used for widely used terms and names, which occurs consistently and frequently in the manuscript.

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Discussion

Emphasize the new and important aspects of the study and conclusion derived from them. Detail data written in introduction and other portions of text should not be repeated. The implication of results and their limitations including suggestion for future research should be included in the discussion.

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Article in journal

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Vega KJ, Pina I, Krevsky B. Heart transplantation in associated with an increased risk for pancreatobiliary

disease. *Ann Intern Med* 1996; 124 (11): 980-3.

As an option, if a journal carries continuous pagination throughout a volume (as many journals do) the month and issue number may be omitted.

b) More than six authors

Parkin DM, Clayton D, Black RJ, Masuyer E, Friedl HP, Ivanov E, et al. Childhood leukaemia in Europe after chernobyl: 5 year follow-up. *Br J Cancer* 1996; 73:1006-12.

c) No author given

Cancer in South Africa (editorial). *S Afr Med J* 1948; 84:15

d) Organization as author

The cardiac society of Australia and New Zealand. Clinical exercise stress testing. Safety and performance guidelines. *Med J Aust* 1990; 146: 267-9.

Books and monographs

a) Personal author(s)

Laurence DR, Bennett PN, Brown MJ. *Clinical Pharmacology*. 8th ed. New York: Churchill Livingstone; 1997.

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

Norman IJ, Redfern SJ, editors. *Mental health care for elderly people*. 5th ed. New York: Churchill Livingstone; 1999.

c) Organization as author and publisher

World Health Organization. *Ethical criteria for medical drug promotion*. Geneva: World Health Organization; 1988.

d) Chapter in a book

Phillips SJ, Whisnant JP. Hypertension and stroke. In: Laragh JH, Brenner BM, editors. *Hypertension: pathophysiology, diagnosis and management*. 2nd ed. New York: Raven Press; 1995. p 465-9.

e) Dissertation or thesis

Kaplan SJ. *Post hospital home health care: the elderly access and utilization (dissertation)*. St. Louis (MO): Washington Uni; 1995.

Other published material

a) Newspaper article

Lee G. Hospitalization tied to ozone pollution: study estimates 50,000 admissions annually. *The Washington Post* 1996; June 21; sect. A: 3 (col. 5).

b) Dictionary and similar references

Student's medical dictionary. 26th ed. Baltimore: Williams and Wilkins; 1995. p.119-20.

Unpublished material

a. In press

Leshner AI. Molecular mechanisms of cocaine addiction. *N Eng J Med* (in press) 1997.

Electronic material

a) Journal articles in electronic format

Morse SS. Factors in the emergence of infectious diseases. *Emerg Infect Dis* [serial online] 1995 Jan-Mar [cited 1996 June 5]; 1(1): [24 screens]. Available from: URL: <http://www.cdc.gov/ncidod/EID/eid.htm>

b) Monograph in electronic format

CDI, clinical dermatology illustrated [monograph on CD-ROM]. Reeves JRT, Maibach H. CMEA Multimedia group, producers. 2nd ed. Version 2.0. San Diego: CAEA; 1995.

C) Computer files

Haemodynamics III: The ups and downs of haemodynamics [computer program]. Version 2.2. Orlando (FL): Computerized Educational Systems; 1993.

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