ISSN 1015-0870

May 2005
Vol. 23, No. 2

Journal of Bangladesh College of Physicians and Surgeons

Official Journal of
The Bangladesh College of Physicians and Surgeons

Journal of Bangladesh College of Physicians and Surgeons

Vol. 23, No. 2, May 2005

Official Journal of the Bangladesh College of Physicians and Surgeons
BCPS Bhaban, 67 Shaheed Tajuddin Ahmed Sarani
Mohakhali, Dhaka-1212, Bangladesh

PUBLISHED BY

Professor Md. Abul Faiz
on behalf of the Bangladesh College of Physicians and Surgeons

PRINTED AT

Asian Colour Printing
130 DIT Extension Road, Fakirerpool
Dhaka-1000, Phone : 9357726, 8362258

ANNUAL SUBSCRIPTION

Tk. 300/- for local and US$ 30 for overseas subscribers

The Journal of Bangladesh College of Physicians and Surgeons is a peer reviewed Journal. It is published three times a year (January, May and September). It accepts original articles, review articles, and case reports. Complimentary copies of the journal are sent to libraries of all medical and other relevant academic institutions in the country and selected institutions abroad.

ADDRESS OF CORRESPONDENCE
Editor-in-Chief, Journal of Bangladesh College of Physicians and Surgeons, BCPS Bhaban, 67, Shaheed Tajuddin Ahmed Sarani, Mohakhali, Dhaka-1212, Tel : 8825005-6, 8856616-7, Fax : 880-2-8828928, E-mail : bcps@bdonline.com

INFORMATION FOR AUTHORS

The Journal of Bangladesh College of Physicians and Surgeons agrees to accept manuscript prepared in accordance with the 'Uniform Requirements Submitted to the Biomedical Journals' published in the New England Journal of Medicine 1991; 324 : 424-8.

Aims and scope:

The Journal of Bangladesh College of Physicians and Surgeons is one of the premier clinical and laboratory based research journals in Bangladesh. Its international readership is increasing rapidly. It features the best clinical and laboratory based research on various disciplines of medical science to provide a place for medical scientists to relate experiences which will help others to render better patient care.

Conditions for submission of manuscript:

- All manuscripts are subject to peer-review.
- Manuscripts are received with the explicit understanding that they are not under simultaneous consideration by any other publication.
- Submission of a manuscript for publication implies the transfer of the copyright from the author to the publisher upon acceptance. Accepted manuscripts become the permanent property of the Journal of Bangladesh College of Physicians and Surgeons and may not be reproduced by any means in whole or in part without the written consent of the publisher.
- It is the author's responsibility to obtain permission to reproduce illustrations, tables etc. from other publications.

Ethical aspects:

- Ethical aspect of the study will be very carefully considered at the time of assessment of the manuscript.
- Any manuscript that includes table, illustration or photograph that have been published earlier should accompany a letter of permission for re-publication from the author(s) of the publication and editor/publisher of the Journal where it was published earlier.
- Permission of the patients and/or their families to reproduce photographs of the patients where identity is not disguised should be sent with the manuscript. Otherwise the identity will be blackened out.

Preparation of manuscript:

Criteria:

Information provided in the manuscript are important and likely to be of interest to an international readership.

Preparation:

a) Manuscript should be written in English and typed on one side of A4 (290 x 210 cm) size white paper.

b) Double spacing should be used throughout.

c) Margin should be 5 cm for the header and 2.5 cm for the remainder.

d) Style should be that of modified Vancouver.

e) Each of the following section should begin on separate page :
   - Title page
   - Summary/abstract
   - Text
   - Acknowledgement
   - References
   - Tables and legends.

f) Pages should be numbered consecutively at the upper right hand corner of each page beginning with the title page.

Title Page :

The title page should contain:

- Title of the article (should be concise, informative and self-explanatory)
- Name of each author with highest academic degree
- Name of the department and institute where the work was carried out
- Name and address of the author to whom correspondence regarding manuscript to be made
- Name and address of the author to whom request for reprint should be addressed

Summary/Abstract :

The summary/abstract of the manuscript :

- Should be informative
- Should be limited to less than 200 words
- Should be suitable for use by abstracting journals and include data on the problem, materials and methods, results and conclusion.
- Should emphasize mainly on new and important aspects of the study
- Should contain only approved abbreviations

Introduction:

The introduction will acquaint the readers with the problem and it should include:

- Nature and purpose of the study
- Rationale of the study/observation
- Strictly pertinent references
- Brief review of the subject excepting data and conclusion

Materials and method:

This section of the study should be very clear and describe:

- The selection criteria of the study population including controls (if any).
- The methods and the apparatus used in the research.
- The procedure of the study in such a detail so that other workers can reproduce the results.
- Previously published methods (if applicable) with appropriate citations.

Results:

The findings of the research should be described here and it should be:

- Presented in logical sequence in the text, tables and illustrations.
- Described without comment.
- Supplemented by concise textual description of the data presented in tables and figures where it is necessaery.

Tables:

During preparation of tables following principles should be followed:

- Tables should be simple, self-explanatory and supplement, not duplicate the text.
- Each table should have a tittle and typed in double space in separate sheet.
- They should be numbered consecutively with roman numerical in order of text. Page number should be in the upper right corner.
- If abbreviations are to be used, they should be explained in footnotes.

Illustrations:

Only those illustrations that clarify and increase the understanding of the text should be used and:

- All illustrations must be numbered and cited in the text.
- Print photograph of each illustration should be submitted.
- Figure number, tittle of manuscript, name of corresponding author and arrow indicating the top should be typed on a sticky label and affixed on the back of each illustration.
- Original drawings, graphs, charts and lettering should be prepared on an illustration board or high-grade white drawing paper by an experienced medical illustrator.

Figures and photographs:

The figures and photographs :

- Should be used only where data can not be expressed in any other form
- Should be unmounted glossy print in sharp focus, 12.7 x 17.3 cms in size.
- Should bear number, tittle of manuscript, name of corresponding author and arrow indicating the top on a sticky label and affixed on the back of each illustration.

Legend:

The legend:

- Must be typed in a separate sheet of paper.
- Photomicrographs should indicate the magnification, internal scale and the method of staining.

Units:

- All scientific units should be expressed in System International (SI) units.
- All drugs should be mentioned in their generic form. The commercial name may however be used within brackets.

Discussion:

The discussion section should reflect:

- The authors' comment on the results and to relate them to those of other authors.
- The relevance to experimental research or clinical practice.
- Well founded arguments.

References:

This section of the manuscript :

- Should be numbered consecutively in the order in which they are mentioned in the text.
- Should be identified in the text by superscript in Arabic numerical.
- Should use the form of references adopted by US National Library of Medicine and used in Index Medicus.

Acknowledgements :

Individuals, organizations or bodies may be acknowledged in the article and may include:

- Name (or a list) of funding bodies.
- Name of the organization(s) and individual(s) with their consent.

Manuscript submission:

Manuscript should be submitted to the Editor-in-Chief and must be accompanied by a covering letter and following inclusions:

a) A statement regarding the type of article being submitted.

b) A statement that the work has not been published or submitted for publication elsewhere.

c) A statement of financial or other relationships that might lead to a conflict of interests.

d) A statement that the manuscript has been read, approved and signed by all authors.

e) A letter from the head of the institution where the work has been carried out stating that the work has been carried out in that institute and there is no objection to its publication in this journal.

f) If the article is a whole or part of the dissertation or thesis submitted for diploma/degree, it should be mentioned in detail and in this case the name of the investigator and guide must be specifically mentioned.

Submissions must be in triplicates with three sets of illustrations. Text must be additionally submitted in a floppy diskette.

Editing and peer review:

All submitted manuscripts are subject to scrutiny by the Editor in-chief or any member of the Editorial Board. Manuscripts containing materials without sufficient scientific value and of a priority issue, or not fulfilling the requirement for publication may be rejected or it may be sent back to the author(s) for resubmission with necessary modifications to suit one of the submission categories. Manuscripts fulfilling the requirements and found suitable for consideration are sent for peer review. Submissions, found suitable for publication by the reviewer, may need revision/modifications before being finally accepted. Editorial Board finally decides upon the publishability of the reviewed and revised/modified submission. Proof of accepted manuscript may be sent to the authors, and should be corrected and returned to the editorial office within one week. No addition to the manuscript at this stage will be accepted. All accepted manuscript are edited according to the Journal's style.

Reprints for the author(s):

Ten copies of each published article will be provided to the corresponding author free of cost. Additional reprints may be obtained by prior request and only on necessary payment.

Subscription information:

Journal of Bangladesh College of Physicians and Surgeons
ISSN 1015-0870

Published by the Editor-in-Chief three times a year in January, May and September

Annual Subscription

Local	BDT	=	300.00
Overseas	$	=	30.00

Subscription request should be sent to:
Editor-in-Chief
Journal of Bangladesh College of Physicians and Surgeons
67, Shaheed Tajuddin Ahmed Sarani
Mohakhali, Dhaka-1212, Bangladesh.

Any change in address of the subscriber should be notified at least 6-8 weeks before the subsequent issue is published mentioning both old and new addresses.

Communication for manuscript submission:

Communication information for all correspondence is always printed in the title page of the journal. Any additional information or any other inquiry relating to submission of the article, the Editor-in-Chief or the Journal office may be contacted.

Copyright :

Reprints of any article in the Journal will be available from the publisher.

JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

Vol. 23, No. 2, Page 50 - 98 **May 2005**

CONTENTS

EDITORIAL

Helicobacter Pylori Infection and Duodenal Ulcer Disease in Bangladesh

Helicobacter pylori is the most prevalent bacterial infection affecting mankind with approximately half the world's population being infected. The majority of the adverse clinical outcomes related to this infection occur in developing countries, where the infection is most prevalent[1]. H. pylori infection is the most important aetiological factor in the development of chronic gastritis and peptic ulcer disease. There is very strong evidence that this infection increases the risk of gastric cancer and gastric mucosa associated lymphoid tissue lymphoma (MALT lymphoma)[2]. H. Pylori eradication in patients with peptic ulcer disease is associated with ulcer healing and a very low rate of recurrence in developed countries[3,4]. Consequently, this is the treatment of choice for peptic ulcer disease associated with H. pylori infection and several expert groups in developed countries have recommended guidelines in this respect[1].

Studies of H. pylori eradication therapy in developing countries has shown equivocal results. This prompted investigations on H. pylori infection and peptic ulcer disease in Bangladesh where the prevalence of these diseases has been found to be high. A study conducted in four villages five miles away from Dhaka city has shown the point prevalence of duodenal ulcer (DU) to be 11.98% and that of gastric ulcer to be 3.5%[5]. The prevalence of H. pylori infection is also very high. Studies conducted in Bangladeshi children by scientists of the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDRB) has shown that 60% of the children are infected by the age of three months and 80% are infected by the age of three years[6] . A seroprevalence study of 268 apparently healthy Bangladeshi subjects between the ages of 15 and 40 years showed 91% to be positive for H. pylori antibody[7]. H. pylori study group of Bangladesh was formed in 1994 to investigate H. pylori infection in Bangladeshi population, its association with peptic ulcer disease and to find out most effective eradication therapy and the recurrence rates after eradication[8]. The first therapeutic trial was to examine the outcome of treatment with two dual therapy (omeprazole, amoxicillin and rantidine, amoxicillin) and two triple therapy regimens (omeprazole, tinidazole, amoxicillin and colloidal bismuth citrate, amoxicillin, metronidazole)[9]. Healing rates with the dual therapy regimens were found to be 67-69% and that with the triple therapy regimens were 85-89%. The second trial was undertaken in two phases to compare the efficacy of furazolidone and metronidazole based triple therapies and to examine the recurrence rates of H, pylori infection[10]. Different groups of DU patients were treated with five different triple drug regimens. Taken as a whole, the eradication rate was found to be 86% and there was no significant difference in the eradication rates of the different regimens. Subsequent studies by investigators in Bangabandhu Sheikh Mujib Medical University and the Combined Military Hospital have shown the eradication rates to be much lower.

In most of the studies, the eradication rate was between 30 and 64%[11,12,13].

Studies in other developing countries have shown conflicting results regarding the eradication rate after therapy. Many studies from India, Pakistan, and Iran have reported eradication rates which are lower than the rates obtained in developed countries and comparable to rates achieved in Bangladesh[14, 15,16.] The reason for the lower eradication rates is not clear. A number of factors may contribute to this. These include: bacterial resistance to drugs, bacterial virulence and host factors. Poor patient compliance and bacterial resistance to drugs have been taken into account in later trials and appear to be unlikely to explain the lower eradication rate. High prevalence of H. pylori cag A and vac A has been reported from

Bangladeshi people[17] but the relationship of these virulence factors to poor response and recurrence has not been investigated. Host factors have not been studied.

Recurrence rates after eradication are much higher in Bangladesh than in developed countries. The re-infection rate was found to be 18% after one year[10]. Follow up of the patients who achieved eradication showed recurrence of 39% after six years (personal communication). High recurrence rates after eradication have also been reported from India, Brazil, Chile and Vietnam[18,19,20]. Lower recurrence rates have also been reported in some studies from developing countries[21,22,23]. The variability of the recurrence rates may be due to lower sensitivity of detection of recurrence in studies which have relied on urea based tests only or on a single test. These may also represent true inter-country differences. Although recurrence of H.pylori infection appears to be high in many developing countries, ulcer recurrence have been reported to be low in patients who have had recurrent infection[18]. If the long term ulcer recurrence is lower in patients who have recurrence of infection after eradication therapy than the natural recurrence rate, it may still be justifiable to give eradication treatment. A comparison of cost effectiveness of eradication therapy versus conventional therapy is in progress in Bangladesh to examine this question.

H. pylori eradication as the mode of treatment for duodenal ulcer disease in Bangladesh therefore poses a problem unlike that in developed countries. The eradication rates are much lower and below the level which is usually considered to be acceptable. The recurrence rates are also very high. Similar findings have been reported from other developing countries. Further works need to be done in order to identify factors responsible for this phenomenon so that more effective therapy for eradication can be found. Till such time, recommendation for H. pylori eradication for routine treatment of duodenal or gastric ulcer, especially in the setting of general practice, may only be given selectively to patients who are likely to be compliant. Success or failure of eradication should be monitored. Work is continuing with newer eradication regimens in Bangladesh to find out better therapies. Studies on cost-effectiveness of eradication compared to conventional therapy is also ongoing. It is hoped that these will yield results that will further the quest for optimal therapy of peptic ulcer diseases.

Dr. Mizanur Rahman Khan MD
Professor Mahmud Hasan PhD
Department of Gastroenterology
Bangabandhu Sheikh Mujib Medical University
Dhaka, Bangladesh

(J Bangladesh Coll Phys Surg 2005; 23 : 50-52)

References :

1. Go MF. Treatment and management of Helicobacter pylori infection. Current Gastroenterology Reports 2002; 4: 471-477.
2. Dixon MF. Pathology of gastritis and peptic ulceration. In: Mobley HLT, Mendz GL, Hazell SL (editors). Helicobacter pylori: physiology and genetics. Washington, D.C.: ASM Press, 2001. pp- 459-469.
3. Borody TJ, Andrews P, Mancuso N, McCauiey D, Jarikiewicz, Ferch N, et al. Helicobacter pylori re-infection rate in patients with cured duodenal ulcer. Amer J Gastroenterol 1994; 89: 529-532.
4. Van der Hulst RW, Rauws EA, Koycu B, Keiler JJ, Ten Kate FJ, Danitert J, et al. Helicobacter pylori reinfection is virtually absent after successful eradication. J Infect Dis 1997; 176: 196-200.
5. Azad Khan AK, Hasan M, Roy PK, Aziz S, Khan MH, Ali SMK, Haq SH. Peptic ulcer in Bangladesh. BMRC Bulletin 1987; 13: 29-42.
6. Mahalanabis D, Rahman MM, Sarker SA, Bardhan PK, Hildebrand P, Beglinger C, et al. Helicobacter pylori infection in the young in Bangladesh, prevalence, socioeconomic and nutritional aspects. Int J Epidemiol 1996; 25: 894-898.
7. Ahmad MM, Rahman M, Rumi AK, Islam S, Huq F, Chowdhury MF, et al. Prevalence of Helicobacter pylori in asymptomatic population- a pilot serological study in Bangladesh. Journal of Epidemiology 1997; 7: 251-154.
8. Morshed MG, Islam MS, Jinnah F, Rumi MAK, Ahmed MM, Huq F. Growth of Helicobacter pylori in candle jar. Lancet, 1995; 340: 511.
9. Ahmed MM, Rahman M, Rahman M, Raihan ASMA, Roy PK, Rahman MT, et al. Two weeks combination therapy with antimicrobials and anti-ulcer agents in duodenal ulcer patients- a comparative study of different regimens in the

healing of DU and eradication of Helicobacter pylori. Bangladesh J Medicine 1999; 10: 4-9.

10. Hildebrand P, Bardhan P, Rossi L, Parvin S, Rahman A, Arfin MS, et al. Recrudescence and reinfection with Helicobacter pylori after eradication therapy in Bangladeshi adults. Gastroenterology 2001; 121: 792798.

11. Rahamn MM, Hossain SMB, Ahmed Z, Siddiqui MAM, Hossain R, Uddin MN, et al. A prospective study of three different Helicobacter pylori eradication regimens in the treatment of peptic ulcer disease. Bangladesh Armed Forces Medical Journal 2001/2002; 29: 1-4.

12. Khan MR, Rasul K, Hasan M. H.pylori eradication therapy for duodenal ulcer disease in Bangladeshi patients by two triple drug regimens: results of a clinical trial. Bangladesh J Med 2003; 14: 39-41.

13. Rahman MT, Mia MAR, Roy PK, Rasul K. Study of efficacy of 14 days triple therapy with furazolidone based regimen (furazolidone plus amoxicillin and omeprazole) in the eradication of Helicobacter pylori in patients with peptic ulcer diseases in Bangladesh. Bangladesh J Med 2003; 14: 1-5.

14. Fokheri H, Malekzadeh R, Bari Z, Yaghoobi M, Sotoudehmanesh R, Ansari R, et al. Suboptimal eradication of Helicobacter pylori with triple therapy among Iranian patients with duodenal ulcer. J Gastroenterol and Hepatol 2002; 17(Suppl): A258.

15. Amarapurkar D, Makesar M, Amarapurkar A, Das HS. Helicobacter pylori eradication: efficacy of conventional therapy in India. Tropical Doctor 2004; 34: 101-102.

16. Qureshi H, Ahmed W, Zuberi SJ. Triple therapy in duodenal ulcer healing-a follow up study. J Pak Med Assoc 1996; 46(Suppl): 194-195.

17. Rahman M, Mukhopadhyay A, Nahar S, Datta S, Ahmad MM, Sarker SA, et al. DNA-level characterization of Helicobacter pylori strains from patients with overt disease and with benign infection in Bangladesh. J Clin Microbiol 2003; 41: 2008-2014.

18. Wheeldon T-U, Hoang TH, Phung DC, Bjorkman A, Granstrom M, Sorberg M. Long-term follow-up of Helicobacter pylori eradication therapy in Vietnam: reinfection and clinical outcome. Aliment Pharmacol Ther 2005; 21: 1047-1053.

19. Rollan A, Giancaspero R, Fuster F, et al. The long-term reinfection rate and course of duodenal ulcer disease after eradication of Helicobacter pylori in a developing country. Am J Gastroenterol 2000; 95: 50-56.

20. Nanivadekar SA, Sawant PD, Patel HD, Shroff CP, Popat UR, Bhatt PP. Association of peptic ulcer with Helicobacter pylori and the recurrence rate. A three year follow up study. J Assoc Physicians India 1990: 38(suppl 1): 703-706.

21. Goh KL, Navaratnam P, Peh SC. Reinfection and duodenal ulcer relapse in south-east Asian patients following successful Helicobacter pylori eradication: results of a 2-year follow-up. Eur J Gastroenterol Hepatol 11996; 8: 1157-1160.

22. Mitchell HM, Hu P, Chi Y, Chen MH, Li YY, Hazell SL. A low rate of reinfection following effective therapy against Helicobacter pylori in a developing nation (China). Gastroenterology 1998; 114: 256-261.

23. Figueroa G, Acuna R, Troncoso M, et al. Low H.pylori reinfection rate after triple threapy in Chilean duodenal ulcer patients. Am J Gastroenterol 1996; 91: 1395-1399.

Situation Analysis of Safe Motherhood Services of Dhaka City Urban Area

S TASNIM[a], AKM SHAHABUDDIN[b], S CHOWDHURY[c], A RAHMAN[d], PC BARUA[e]
F RAHMAN[d], N BEGUM[a], F ISLAM[a], M SARKER[f]

Summary:

Maternal mortality is still quite high in Bangladesh despite decades of programmes and activities. Maternal health care coverage depends on availability and utilization of services . With trend of increase in urbanization there is increasing demands for maternity care services .

The objective of the study was to assess present status of utilization of the antenatal, childbirth, postnatal and neonatal care services at urban setting.

This was a cross sectional descriptive study done during August to December 2001 in the catchment area of randomly selected ten health care centres run by partner NGOs of Urban Primary Health Care Project and Dhaka City Corporation in Dhaka Metropolitan city. A total 3,000 mothers with under one year child was selected randomly from about 49,526 households and information were collected by interviewer-administered structured questionnaire.

Mean age of respondents was 24.6 ± 5.12 years. Among them, 31.14% were illiterate and 67.87% were multipara. About 87.74% received antenatal care during last pregnancy, of which 23.74% from public sector, 32.63% from NGO and 39.63% from private physicians. Antenatal care was provided by graduate doctors in 51.42% and by paramedics in 29.33%. Institutional delivery was 35% and conducted by a trained person in 39% cases. Reasons for not seeking medical care during pregnancy and delivery were financial difficulties (24.53%), no perceived problem (25.16%), transportation problems (11.70%) and fear of Caesarian operation (4.43%).

Pregnancy and childbirth were perceived to be a natural event and delivery should be done at home. Health centres were thought to be a place for dealing with emergencies and complications only.

(J Bangladesh Coll Phys Surg 2005; 23 : 54-58)

Introduction :

Bangladesh is one of the highest populous country of the world with about 2.6 million childbirth per year. Annual growth rate of urban population is over six percent per annum[1] while national figure is 1.5 percent[2]. Maternal mortality is reported to be 3.2 per thousand live births[3]. The infant mortality rate is 53 per thousand live birth but in urban slums the rate is 142 per thousand live birth, which is 58 percent higher than the national average[4]. Despite decades of programmes and activities, maternal health care coverage is still quite inadequate.

Characteristics of service delivery system i.e. availability of health care provider and facility for services, reflects the probable and potential level of access to medical care. Utilization and satisfaction are the indicators of actual or realized access to services.

About 20% of population in Bangladesh reside in urban areas and there is ever increasing thrust on urbanization[4]. Percentage of urbanization in Dhaka increased from 14.79% in 1961 to 53.90% in 1991[5]. It has been found that 61% of urban household are below absolute poverty line, and countless slums and squatter settlements have evolved in most urban areas[6]. It is natural that healthcare facilities are not expanding in the same pace to cope up with increasing demand and it is even harder for urban poor to have an access to the health services.

The objectives of this study was to explore the prevailing maternity care status in terms of utilization of antenatal, childbirth, postnatal and neonatal care

a. Dr. Saria Tasnim, FCPS, MMED, Dr. N. Begum, Dr. F. Islam, Associate Professor of Obstetrics & Gynaecology, Institute of Child & Mother Health
b. Prof. AKM. Shahabuddin, Professor & Ex- Head of the Department Obstetrics & Gynaecology, Institute of Child & Mother Health
c. Prof. S Chowdhury, Professor, Department Obstetrics & Gynaecology, Institute of Child & Mother Health
d. Dr. A Rahman, Dr. F Rahman, Asstistant Professor of Epidemiology, Institute of Child & Mother Health
e. Prof. PC Barua, Professor of Epidemiology, Institute of Child & Mother Health
f. M Sarker, Nursing Superintendent, Institute of Child & Mother Health

Address of correspondence : Dr. Saria Tasnim, Associate Professor, Department of Obstetrics & Gynaecology, Institute of Child & Mother Health, Matuail, Dhaka-1362.

services at urban settings, and to determine the determinants of non -utilization of such services.

Materials and method:
This was a cross sectional descriptive type of study done during August to December 2001 in the catchment area of randomly selected ten health care centers run by partner NGOs of Urban Primary Health Care Project and Dhaka City Corporation in Dhaka Metropolitan city. The criteria of such geographical clustre was the population size of 20 to 25 thousand, at least 25% were poor and population were stable in a clearly delineated area. Information were collected with a interviewer-administered structured questionnaire. A total of 49,526 households were visited to find out mothers with under one year child, and among them 3,000 were selected randomly as respondents.

Results:
The mean age of respondents was 24.65± 5.12 years and 14.18% belonged to age group 15 – 19 years. About 31.14% were illiterate (Table–I). Obstetric profile showed 32.13% had one child, about 25% were married at or below age 14 years and mean age at marriage was 16.7±3.31 years. Average age at first pregnancy was 18.44 ± 3.26 years and the prevalence of teenage pregnancy was 68.12% (Table–II).

About 87.74% respondents received antenatal care during last pregnancy and mean number of visits was 5.53±3.61. Service was availed from public sector by 23.74%, NGO clinic by 32.63% and private facilities 39.63% (Figure: 1). Majority of this care was from graduate doctors (51.42%) and doctor specialist in 17.08% (Table-III). About two third (63.67%) of respondents suffered from some form of complication during the last pregnancy and 46.63% sought treatment (Table-IV). Care was received from graduate doctors and specialists (52.12% and 31.25% respectively) for complications.

The place of last delivery was at service facilities in 46.83%, of these 13.17% at public hospitals (Figure: 2). The delivery was conducted by a trained person in 38.66% cases (doctors 32.42%, paramedics 6.24%) and traditional birth attendants (TBAs) in 50.34% (Figure: 3).

About a qurter (24.53%) respondents expressed that they did not avail the service of any centre or doctor during delivery due to financial difficulties, perceived absence of problem (25.16%), transportation problem (11.70%) and fear of Caesarian section (4.43%) (Table-V).

Table-I

Socio- demographic characteristics of respondents (N=2,954)

Characteristics	Frequency	Percentage
Age of respondent (years):		
15-19	419	14.25
20-24	1,114	37.62
>25	1,421	48.13
Educational level of respondent (years):		
Illiterate	925	31.14
<5 years schooling	669	22.62
> 5 years schooling	1,356	46.24

Table-II

Reproductive health characteristics of the study populations (N=2,954)

Characteristics	Frequency	Percentage
Para:		
Primi -	949	32.13
Multi -	2,005	67.87
Age at marriage (years):		
≤14	738	24.98
15-19	1,689	57.17
>19Years	527	17.85
Marriage before age 18 years	2,221	75.18
Age at First pregnancy (years):		
10-14	260	8.81
15-19	1,754	59.37
>19	940	31.82

Table-III

Antenatal care service utilization by the respondents (N=2,954)

	Number	Percentage
Received antenatal care during pregnancy	2,592	87.74
Provider for antenatal care:		
Graduate doctor	1,334	51.42
Doctor specialist	490	17.08
Paramedics	761	29.33
Others	56	2.17

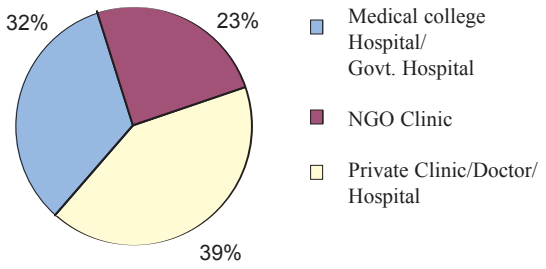

Figure-1 : *Source of antenatal care*

Table-IV

Care received for pregnancy complication by the respondents

Characteristics	Number	Percentage
Suffered from complication	1,881	63.67
Received treatment for complication	1,289	46.63
Source of treatment for complication:		
Medical College Hospital/ Government Hospital	256	20.01
Private clinic/physician/hospital	614	47.63
NGO clinic/UFHP	267	20.74
Others	227	17.62

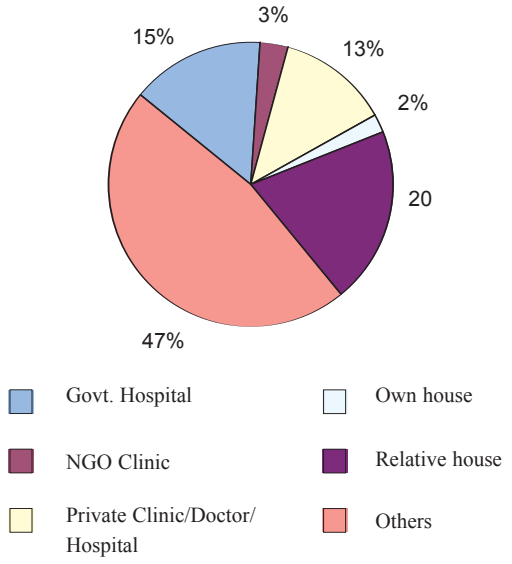

Figure-2: *Distribution of place of delivery*

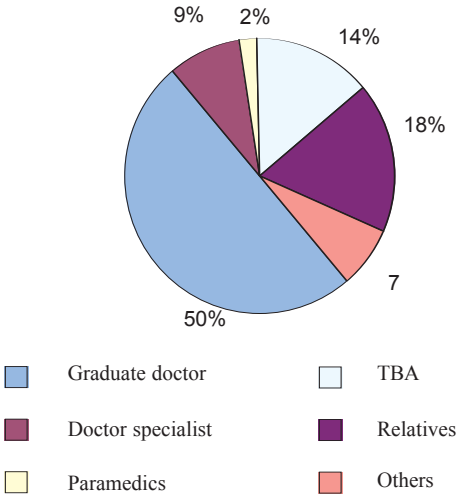

Figure-3 : *Distribution of people conducting delivery*

Table-V

Causes mentioned by the respondents for non-utilization of health centre/physician services during delivery

Causes	Number	Percentage
Long distance	50	2.84
Poor management at the health centres	37	2.12
Financial problem	444	24.53
Transportation problems	216	11.70
Perceived absence of problem	448	25.16
Fear of Cesarean section	129	4.43
Others	184	6.22

Discussion :

A woman's health is intricately entwined with her social status that in turn involves a complex set of interrelated factors. Those factors include her income, employment, education, health and fertility, and society's perception of her role in the family and the community[7].

Teen-age marriage is a reflection of low status of woman, favours high fertility and is a crucial factor in maternal mortality[7]. It is advocated that pregnancy below age 18 years and above 35 years are more susceptible to adverse pregnancy outcome. Bangladesh Demographic and Health Survey, 1997

showed that overall about 60% of Bangladeshi women are married by the time they are 15 years of age and median age at marriage among women of 20-49 years is 14 years. It is reported that 72% of 15- 19 years age group are married and they contribute approximately 20% of total pregnancy[8] . Another study shows currently married teen age women (13-19 years) are 37%[3].

Although antenatal care coverage is reported quite high (87.74%) in the present study but the care did not meet the required routine four visits recommended by WHO[9]. Some of the visits were for doing investigations, collection of report or for associated medical problems and collection of medicine. However, antenatal care coverage was found increased from 30% of births (1996-97)[2] to 35% during 1999-2000[5] and 41% in 2001[3]. Antenatal care was provided by medically trained persons in one third of births and 59% of urban birth and 28% of birth in rural area had antenatal care coverage[10]. Care is most effective if the visits were started early during pregnancy and continued at regular intervals throughout the pregnancy[11]. Women who had attained secondary education or who were from wealthy households were more likely to be assisted at delivery by a medical professional than women with no education or who were from poorer households[4].

It was striking that about 60% of women reported one or more complications during last pregnancy but only 46% sought treatment. The utilization of private clinic/doctor services was more than government facility (47.63% vs 20.01%) and NGO clinic utilization (47.63% vs 20.74%). However, the national survey has revealed that women sought medical care for pregnancy with complication in 56% cases; among them, about 40% attended government facilities, 20% private doctors, 19% NGOs/private clinic and 16% traditional healers[3]. It has been found that utilization of NGO/Private clinic is preferred much more among urban than national (30% versus 15%) and use of antenatal care is strongly associated with level of education and household economic status[3].

It is well appreciated that most important intervention for reduction of maternal mortality is ensuring skilled attendants during delivery. A study has revealed that nationally only 12% births are done by medical trained people (doctors 7%, and nurse, midwives or FWV 5%)[3]. In the urban area, doctors and nurses attend 21% and 16.2% deliveries respectively and 65% are attended by TBAs[12]. This study also showed that 60% of births were conducted by TBAs and relatives. Survey has revealed that in urban areas, 43.3%, 47.7% and 9% births are conducted by nurse/doctor, midwife and relatives respectevly[2].

According to Associates for Community and Population Research the number of deliveries at government facilities constituted 5.33% of the total estimated annual deliveries and 3.97% of complicated obstetric cases attended emergency obstetric care facilities for treatment. Of the institutional deliveries, 64.62% took place in government facilities[13]. Another study found 32% of metropolitan/ town deliveries took place in a service facility[3].

There are a number of socio-cultural determinants for availing maternity care services. Cost is often cited as a barrier (24.53%) even though free services are available in the government facilities. A great proportion (25.16%) did not feel the need to go to any centre, 11.70% mentioned transport problems and 4.43% were afraid of Caesarian section. Another study revealed reasons for not going to a facility during delivery was the perceived absence of need (68%), cost of visit (18%), transport problem (6%), 10% mentioned poor quality service and 4% had fear of service[3]. Many a times delivery occurred at parental home in rural areas, so even for those availing antenatal care from a centre the delivery service of that center was not utilized.

It is often stated that one of the most pressing barrier to attain safe motherhood service is rooted in the powerlessness of women and their unequal access to resources in families, society and economic sectors. Women's limited exposure to information causes them to accept pain and suffering as "women's tale" and they do not perceive pregnancy as an event requiring any additional care. In the current study, 28.16% expressed that they did not perceive any problem so did not avail any antenatal care in their last pregnancy. Studies have shown that use of antenatal care is strongly associated with level of education and economic status[14].

World Health Organization defines the accessibility of the service facilities as the proportion of the given

population that can be expected to use a specified facility, service etc. The barriers of accessibility may be physical e.g. distance, economic e.g. travel cost or fee charged, and socio- cultural e.g. cast or language[15].

Access is not to be equated with the use of services. Utilization proves that access has been achieved, but utilization rates do not permit determination of the degree to which services were not used for any reason. Before deciding whether a pregnant women can or will use a particular health service, following factors should be considered. She must perceive a need for them, she must be aware of her condition, and feel that it warrants medical intervention. The appropriate services must be available to her and the service must be acceptable to her (i.e. she should have confidence in the technical competence and 'humanness' of the facility and its provider). She should also have the ability to obtain the service (the necessary income and time).

Utilization of antenatal care service was quite impressive in Dhaka city but the care provided was, to some extent, incomplete and inadequate. Health seeking for complication during pregnancy and childbirth, although limited, was more from private sector. There is an universal preference for home delivery and majority of deliveries are conducted by traditional birth attendants. Pregnancy and childbirth is traditionally regarded as a natural event and perceived to be dealt with at home, and hospital or health facilities are place for dealing with emergency and abnormal situations.

Acknowledgement:
Financial support for this research was provided by Asian Development Bank (ADB) through Urban Primary Health Care Project (UPHCP) under Local Government division, Ministry of Rural Development & Co-Operatives, Government of the People's Republic of Bangladesh.

References :

1. Asian Development Bank. Urban poverty in Bangladesh: Final Report. Dhaka: Asian Development Bank, 1996.
2. Bangladesh Data Sheet. Bangladesh Bureau of Statistics, Government of Peoples Republic of Bangladesh, 2000.
3. Bangladesh Maternal Health Services and Maternal Mortality Survey 2001. NIPORT, MEASURE, DHSTORC MACRO, March 2002. pp-37-53.
4. Urban Primary Health Care Project. Baseline Household Survey 1999. Mitra and Associate, Bangladesh in collaboration with Johns Hopkins University, September 2000. pp-2-4.
5. Statistical Pocketbook. Bangladesh Bureau of Statistics, Government of Peoples Republic of Bangladesh, 2002.
6. Nazrul Islam. The urban poor in Bangladesh: Comprehensive summary report. Center for Urban Studies, University of Dhaka, 1990.
7. Royston E, Armstrong S (editors). Preventing maternal deaths. Geneva: World Health Organization , 1989. pp-45
8. Bangladesh Demographic and Health Survey, 1993-94. National Institute of Population Research and Training Institute, 1997. pp-109-116.
9. World Health Organization. Mother Baby Package : Implementing Safe Motherhood in Countries. Geneva: World Health Organization, 1994. pp-22.
10. Bangladesh Demographic and Health Survey 1996-97. National Institute of Population Research and Training Institute, 1999.
11. Carrali G, killer J, Piaggio G, Neelofur D K, Gulmezoglu M, Mugford M, et al. WHO systematic review of randomized controlled trials of routine antenatal care. Lancet 2001; 357: 1565-1570.
12. Government of Bangladesh. Country reports and action plans, Saving lives: Skilled attendance at childbirth. Government of the People's Republic of Bangladesh, 2000.
13. Associates for community and population research. Review of availability and use of emergency obstetric care (Emoc) services in Bangladesh, October, 2001.
14. Kwast B E. Building a community based safe maternity programme. Internati J Gynaecol Obstet 1995; 48 (Suppl): S89- S 92.
15. World Bank Human Development Network. Safe Motherhood and the World Bank. The World Bank, Washington D.C., 1999.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 22, No. 2, May 2005

Biliopancreatic Ascariasis: Presentation and Management

M RAHMAN[a], TM BHUIYAN[b], M KABIR[c], N HAQUE[c],
AQM MOHSEN[d], MA RAHMAN[d], MS ARFIN[e], M ALAM[b]
SM HUSSAIN[f], MA MASUD[g], AKA KHAN[h]

Summary:

Biliopancreatic ascariasis is one of the most common and well-described entities caused by Ascaris lumbricoides. Diagnosis can be made by ultrasonography and/or ERCP. ERCP, in addition, has therapeutic potential. A prospective study was done on 'Biliopancreatic ascariasis' to find out different presentations of biliopancreatic ascariasis and to assess the roles of ultrasonography and ERCP for the diagnosis and management of the hepatobiliary and pancreatic complications of ascariasis. All consecutive patients presenting with the evidence of round worm in the biliary tree and/or in the pancreatic duct were included in this study. Total 51 patients were included. Male to female ratio was 1: 2.4 with mean age 38.9 (±17.7) years. Most of the cases were from rural areas (86.30%). Majority of the cases were from the southern part of Bangladesh (88.20%) and recorded during the month of October to March (76.40%). Ultrasonography could correctly diagnose the biliopancreatic ascariasis in 85.70% of cases. All the patients complained of abdominal pain at presentation. Presentations of biliopancreatic ascariasis cases were: biliary colic (45.10%), cholangitis (39.20%), ascaris induced acute pancreatitis (9.80%), acute acalculous cholecystitis (3.90%) and liver abscess (2.0%). Associated findings were choledocholithiasis (19.60%), recurrent pyogenic cholangitis (7.80%) and papillary stenosis (11.80%). Removal of worms was done mostly endoscopically (90.20%). Endoscopic success rate was 93.90%. Papillotomy was done in 86.70% of cases where ERCP had been done. Mean duration of hospital stay after endoscopic treatment was 2.6+1.6 days. Endoscopic removal with papillotomy in most of the cases was safe. During the average follow-up period of 12.4 months, recurrence of biliopancreatic ascariasis occurred in 7.80% of cases.

This study concludes that ultrasonography is a highly sensitive and specific noninvasive method for diagnosis and follow-up of the patients with biliopancreatic ascariasis. Emergency endoscopic removal of round worm is the treatment of choice for biliopancreatic ascariasis.

(J Bangladesh Coll Phys Surg 2005; 23 : 59-67)

a. Dr. Mohammad Mohibur Rahman FCPS, MD, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital,
b. Dr. Tareq M. Bhuiyan FCPS, Dr. Md Mahbub Alam PhD, MD, Assistant Professors, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital.
c. Dr. Mohsin Kabir MD, Dr. Nazmul Haque MD, Medical officers, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital.
d. Prof. A Q M Mohsen FCPS, Prof. Md Anisur Rahman FCPS, Professors, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital.
e. Dr. MS Arfin FCPS, Associate Professor, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital.
f. Dr. Shafiuddin Mahmood Hussain DTCD, Gastro-Liver Hospital and Research Institute, Dhaka
g. Dr. M A Masud FCPS, MD, Associate Professor, Department of Gastroenterology, Dhaka Medical College.
h. Prof. A K Azad Khan, FCPS (Med), FRCP, D.Phil (Oxon), Honoris Causa Medicinae Doctorem (Basel), Honorary Sr. Consultant, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital.

Address of correspondence : Dr. Mohammad Mohibur Rahman FCPS, MD, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM Hospital, Dhaka.

Introduction:

Ascaris lumbricoides is the most common helminth in the human gastrointestinal tract. The infection largely occurs in tropical and subtropical countries, related in part to the warm and humid soil that is conducive to the development of the Ascaris larva, and in part to the poor sanitary and hygienic conditions that maintain the infection.[1] Bangladesh is an area where ascariasis is endemic. Ascariasis is an important cause of hepatobiliary and pancreatic diseases in this area.

Biliopancreatic ascariasis (BPA) is one of the most common and well-described entities caused by Ascaris.[2] BPA can cause different hepatobiliary and pancreatic complications in addition to biliary colic.[3]

Diagnosis of BPA can be made by ultrasonography and ERCP. Ultrasonography is a highly specific and sensitive method for detection of worms in the biliary tree.[4] ERCP, in addition, has therapeutic potential, allowing removal of worms from the ducts or the duodenum.[2] Early diagnosis and treatment is important to prevent the ascariasis related complications.[5]

Immediate endoscopic treatment results in rapid resolution of the problem.[1] But if treatment is delayed, serious complications such as ascending cholangitis, haemorrhagic pancreatitis, and death may occur.[1] Since no significant complications are found, endoscopic treatment has become the treatment of choice in many centres with surgery reserved for the failed cases. Urgent surgery is mandatory when there is biliary perforation due to worms[6, 7].

This study on 'Biliopancreatic ascariasis- presentation and management' was done to find out different presentations of BPA and to assess the roles of ultrasonography and ERCP for the diagnosis and management of the hepatobiliary and pancreatic complications of ascariasis.

Materials and method:

This study was a prospective observational study and was conducted during September 2001 to April 2003 in the Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, Bangladesh Institute of Research and Rehabilitation on Diabetic, Endocrine and Metabolic Disorders (BIRDEM) Hospital and Gastro-Liver Hospital and Research institute, Dhaka.

All consecutive patients presented with upper abdominal pain for at least one day were screened for the study. The cases were selected on the basis of following criteria:

Group I: Patients presented with upper abdominal pain were evaluated initially by ultrasonography. Patients with sonographic evidence of round worm in the biliary tree and/or in the pancreatic duct were included in this study. They were evaluated further by ERCP and findings were compared.

Group II: In cases where ultrasonography was negative for BPA, ERCP was done in some of these cases suspecting other biliary and pancreatic pathology. If round worms were found in the biliary tree and/or in the pancreatic duct on ERCP, they were also included in the study.

Group III: In cases where there was ultrasonographic evidence of BPA but ERCP could not be done, round worm was found in the biliary tree and/or pancreatic duct at laparotomy were also included in the study.

Group IV: In some patients with upper abdominal pain, endoscopy of upper GIT was done to exclude peptic ulcer disease. Ultrasonography was normal in these cases. If round worm was seen protruding through the ampullary orifice at endoscopy, they were also included in this study.

Exclusion criteria: 1. Round worms in the duodenum away from the papilla and not seen invading the ampullary orifice;

2. Round worms in the biliary tree or pancreatic duct with any morbid illness that may mimic the presentation of BPA (especially chronic calculous pancreatitis).

Detailed history including signs/ symptoms, residence, season of occurrence, previous history of biliopancreatic ascariasis, previous surgery of the biliary tree, previous ERCP and papillotomy and clinical examination were recorded in the patients entry form. Blood for Hb%, total count, differential count of WBC, liver function tests like SGOT, SGPT, alkaline phosphatase, and bilirubin were done. Serum amylase was done in patients with clinical suspicion of ascaris induced acute pancreatitis. Ultrasonography of hepatobiliary system and pancreas was done by a real time scanner (Siemens Sonoline SL-1). The sonographic diagnosis of biliary and pancreatic ascariasis was made when non-shadowing long echogenic structures with a central sonolucent tube were visualized in the biliary tree or pancreatic duct (Fig.-1). Acute pancreatitis considered to be caused by *A. lumbricoides* infestation when adult worms were detected in the biliary tree and/or in the pancreatic duct. Diagnosis of recurrent pyogenic cholangitis (RPC) was done on the basis of ERCP evidence and clinical history.

All patients were kept nil per oral till the improvement of the condition (Fig.-2).

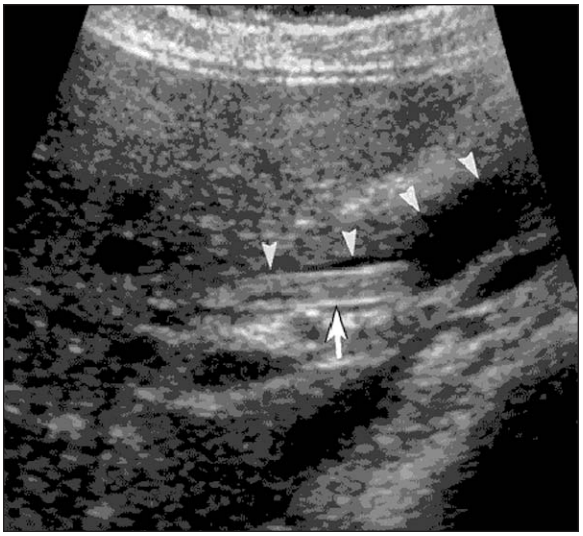

Figure-1 : *Sagittal ultrasonographic image of the porta hepatis shows a tubular echogenic region (arrow) within dilated CBD (arrow head).*

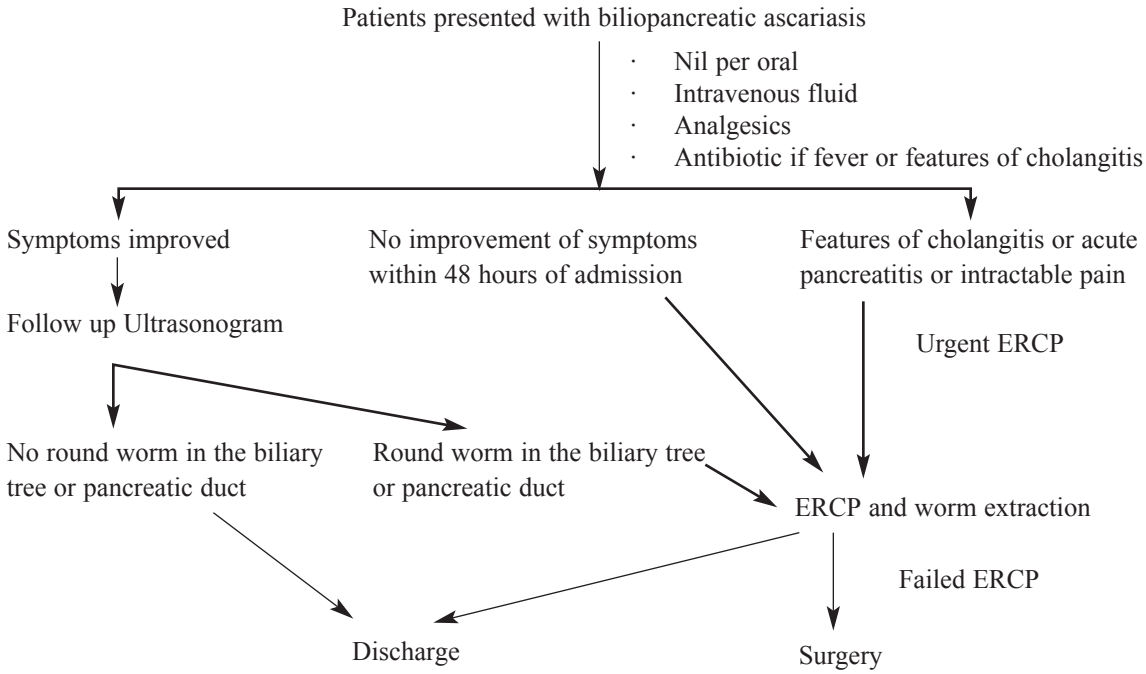

Figure-2 : *Management of biliopancreatic ascariasis cases.*

ERCP was done using a Fujinon video side viewing duodenoscope (Superimage EPX 310 scope). Worms were removed with an Olympus dormia basket with sphincterotomy or without sphincterotomy if papilla was wide opened (Fig.-3). If a part of the worm is visible out side the papilla of Vater, it was caught in a dormia basket and extracted (Fig.-4).

Figure-3 : *Cholangiogram showing round worm (tubular filling defect) within common bile duct (Courtesy: Prof. A Q M Mohsen).*

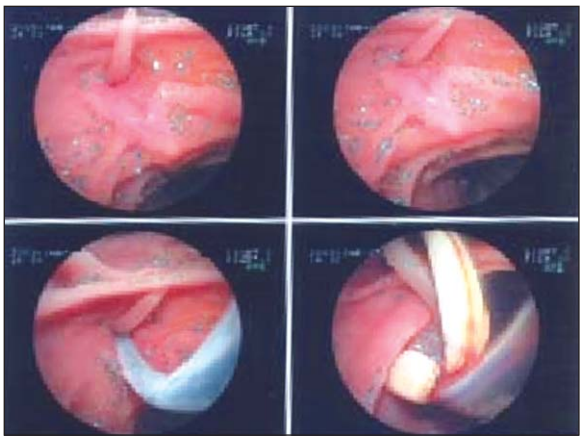

Figure-4 : *ERCP (Video picture) showing the extraction of round worm with the dormia basket in situ (Courtesy: Prof. A Q M Mohsen).*

Endoscopic accessories used for extraction of worms were an Olympus dormia basket catheter type FG-22 Q, an Olympus polypectomy snare type SD-7P, an Olympus papillotome type KD-7Q, Olympus biopsy forceps type FB-25K and electro coagulation equipments type bipolar coagulation.

All adult patients were given three tablets levamisole (in children 3-5 mg/kg) or syrup pyrental palmoate

500 mg (in children 10 mg/kg) stat after the extraction of worm or when symptoms subsided and after one week of the first dose. Thereafter each patient was advised to take regular anthelminthics each two months life long for prevention of re-infestation.

Patients were advised to report if symptoms recur. During follow-up collection of history, physical examination and ultrasonogram were done. ERCP was done if worms were seen within the biliary tree or pancreatic duct.

Statistical analyses were done using the computer based SPSS programme (Statistical Package for Social Science - SPSS Inc, Chicago. Version 10.0). Comparisons between two groups were done by student's t test, chi-square test as applicable; p value of less than 0.05 was considered as significant.

Results:

A total of 51 cases were included in this study. Out of them 36 cases (70.60%) were female and 15 cases (29.40%) were male with age range of 2.5 to 72 years with [mean 38.9 ± 17.7)]. Seventy two percent of the patients were in the age group of 20- 60 years. Most of the cases were from rural areas (86.30%) and they came from the southern part of Bangladesh (88.20%).

Thirty-nine (76.40%) cases were recorded during the month of October to March. In this study the presenting features of BPA were: pain (100%), nausea (92.20%), vomiting (76.50%), worms with vomitus (25.50%), fever (52.90%), jaundice (39.20 % cases) (Table-I). Duration of pain at presentation

Table-I

Presentation of biliopancreatic ascariasis: clinical features

Symptoms and signs	Number of patients	Percentage
Pain	51	100
Site of pain:		
Epigastric	09	17.60
Right hypochondriac region	31	60.80
Both	09	17.60
Diffuse	02	3.90
Radiation of the pain:	19	37.30
Site		
Scapula	11	21.60
Right shoulder	02	3.90
Back	05	9.80
All over the abdomen	01	2.00
Nature of pain:		
Colicky	50	98.00
Dull aching	01	02
Severity of pain:		
Severe	50	98.00
Moderate	01	2.00
Mean duration of symptoms	9.94 ± 6.34 (mean±SD)	
Nausea	47	92.20
Vomiting	39	76.50
Vomiting of worms	13	25.50
Fever	27	52.90
Grading of fever:		
High grade (>100°F)	24	47.10
Jaundice	20	39.20
Per abdomen tenderness on palpation:	40	78.40
Right hypochondriac region	24	47.10
Epigastric region	06	11.80
Both	09	17.60
Diffuse	01	2.00

was ranged from 1- 25 days [(9.94 ± 8 mean±SD) days]. Clinically 39.20% of the patients were icteric and 480% of the patients were febrile (100^{O} -103^{O}F). Abdominal tenderness was present in 78.40% of the patients. Similar history of BPA in the past was present in 17.60% of cases. In eleven patients there was associated disease or condition (Table-II).

Table-II

Associated conditions or diseases in patients with biliopancreatic ascariasis

Diseases or conditions	Number	Percentage
Diabetes mellitus	02	3.90
Diabetes mellitus with hypertension	01	2.00
Pregnancy	02	3.90
History of choledocholithiasis with ERCP and papillotomy and stone extraction	01	2.00
Past history of repeated ERCP with papillotomy	01	2.00
History of laparoscopic cholecystectomy	03	5.90
History of open cholecystectomy with choledocholithotomy	01	2.00

Laboratory data of this study revealed –leucocytosis in 71.30% of cases; eosinophilia (> 6%) in 21.40% of cases. Bilirubin, SGPT, SGOT, Alkaline phosphatase level was raised in 50%, 71.40%, 83.30% and 66.70% cases respectively (Table-III). Serum amylase was done in 21 cases and it was raised in seven cases (33.30%); in four cases it was raised more than three times of the upper level of normal.

Table-III

Laboratory data of the biliopancreatic ascariasis cases

Parameter	Mean value
Total count of white blood cells	11992 + 5381
Serum bilirubin	2.78 + 2.51 mg/dl
SGPT	74.69 + 66.02 iu/l
SGOT	77.2 + 73.6 iu/l
Serum alkaline phosphatase	1.8 + 1.24 times the upper level of normal value

In this study, ultrasonography could correctly diagnose the BPA in 85.70% of cases. USG was false positive in 9.30% of cases. Most common sites of worm in the biliary tree were in the common bile duct (Table-IV).

Table-IV

Sites of worm in biliopancreatic ascariasis cases

Site	Number	Percentage
Common bile duct (CBD)	30	62.50
CBD and common hepatic duct (CHD)	07	14.60
CBD, CHD and intrahepatic biliary tree	08	16.70
Intrahepatic biliary tree	01	2.10
Gall bladder	01	2.10
Pancreatic duct	01	2.10

Most of the patients of BPA presented with biliary colic (45.10%). Hepatobiliary and pancreatic complications were present in 54.90% of the patients (Table-V). Associated findings were choledo-cholithiasis in 10 cases (19.60%), Recurrent pyogenic cholangitis in four cases (7.80%) and papillary stenosis (organic) in six cases (11.80%).

Table-V

Presentations of biliopancreatic ascariasis cases

Presentation	Number	Percentage
Biliary colic	23	45.10
Cholangitis	20	39.20
Ascaris induced acute pancreatitis	05	9.80
Acute acalculous cholecystitis	02	3.90
Liver abscess	01	2.00

Management (Fig.-5): In forty-six cases (90.20%) removal of worms was done endoscopically. Spontaneous expulsion of worms occurred in two cases (3.90%). Surgery was needed in two cases (in one case there was associated intrahepatic stone in left hepatic duct, the other case was ascaris induced acute cholecystitis with empyema gall bladder). In one case complete extraction of worm could not be done (a case of RPC with demised worms within

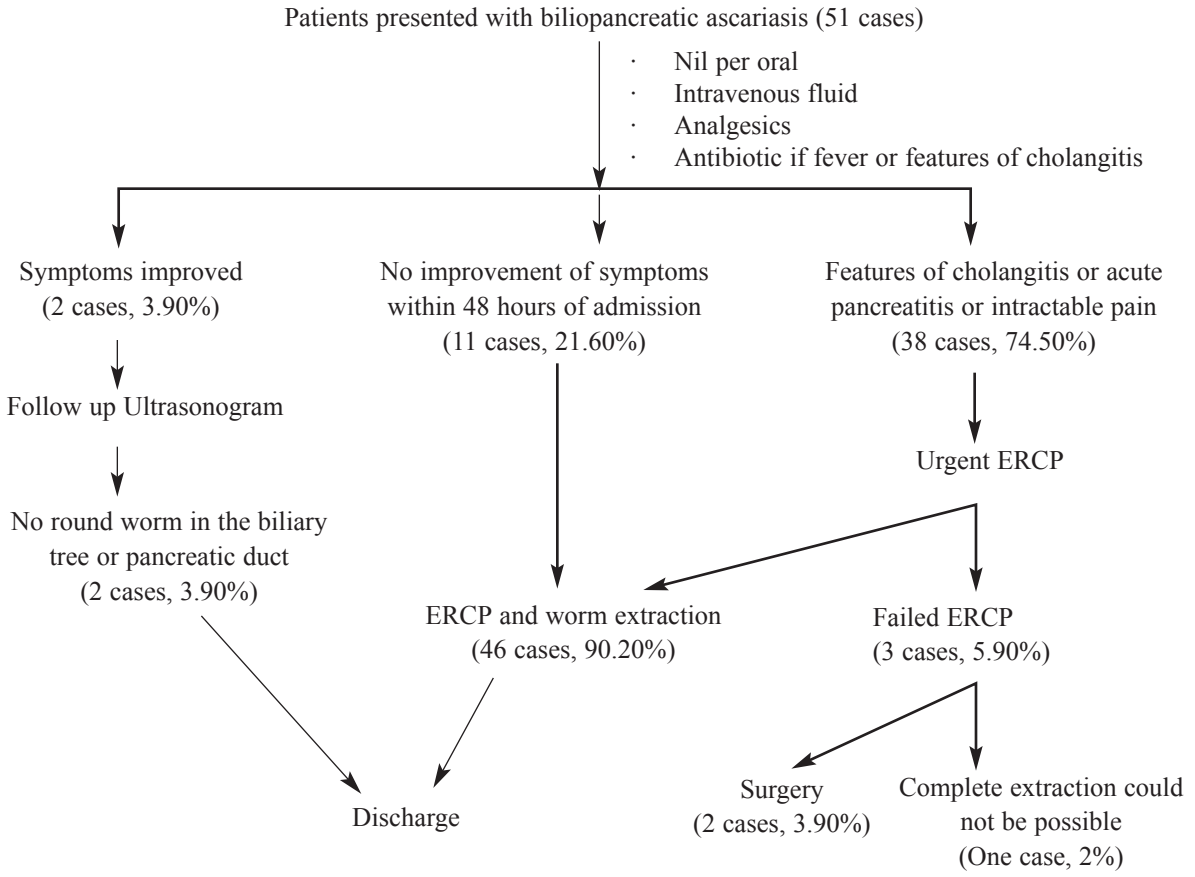

Figure-5 : *Results of management of biliopancreatic ascariasis cases*

intra- and extrahepatic ducts with extensive intra- and extrahepatic stone formation). Overall endoscopic success rate was 93.90%.

Papillotomy was done in 39 cases out of 45 where ERCP had been done. In three cases papillotomy was done previously. In three cases papillotomy was not done (in one case there was technical difficulty due to juxtra papillary diverticula, in other two cases the papilla was wide open).

Mean duration of hospital stay after endoscopic treatment was 2.6+1.6 days (maximum eight days). Majority (71.60%) of the patients could be discharged on the day after ERCP done. Improvement of symptoms of BPA occurred within a mean of 1.6+1.3 days.

Complications occurred in three cases (6.50%), in two cases there was biliary colic with normal amylase level and in one case there was mild cholangitis.

During the average follow-up period of 12.4 months recurrence of BPA occurred in four patients (7.80%).

Discussion:

The prevalence of ascaris varies in different parts of the world. In China and South-East Asia it is highly prevalent[8].

Ascaris-related clinical disease is estimated to be 1.2 to 2 million of cases, with 20,000 deaths in a year.[9] The dangers of migration of the round worm into the biliary tree was first emphasized by Cromwell[10].

Endoscopic treatment of BPA started about twenty years back since 1984 in other parts of the world[7, 10]. In Bangladesh, ERCP was done for the first time in May 1984 in the Gastroenterology Department of Institute of Post-graduate Medicine and Research (IPGMR)[11].

In this study BPA is more common in women (70.6%) than men that is similar to other studies[1,4]. The exact reason for female preponderance is still not clear. It is possible that in young females, hormone progesterone leads to relaxation of smooth muscles of the sphincter of Oddi, allowing the round worms to gain easy entrance to bile duct[8]. The other reason may be greater contact of woman with children[7]. The mean age of patients of this series was 38.9 years which is very similar to an other study[4].

In this study, only three patients (5.80%) of BPA at or below the age 10 years were found. Children do suffer from BPA but less often than adults[12] though the incidence of ascaris infection is more common and heavier in children[13]. Low rate of BPA among children despite high worm load is possibly due to the smaller size of the bile ductal system making it difficult for the worms to enter[2].

In this study, only two cases of pregnant women with BPA were found, though at pregnancy women are more prone to BPA because of smooth muscle relaxation effect of the progesterone on the bile ducts and hypocontractility of the gall bladder during pregnancy[2].

In this study, most of the cases were from the rural areas as recorded in other studies.[4, 14] Poverty and over crowding, unhygienic living conditions and poor sanitation contribute to the spread of infection from person to person[13]. Most of the cases were from the southern part of Bangladesh. The soil of these parts the country is more humid than other parts of the country. Humid soil and temperate climate (the embryo develops in only three weeks in optimal temperature of 25^0C, average temperature is 36^0 to 40^0C)[8] are excellent conditions for the development of the larval stage of the organism[10, 13]. Most of the cases were recorded during the month of October to March (76.40%). Seasonal variations of BPA was observed in a study conducted in Syria[1].

In this study the presenting features of BPA were pain, nausea, vomiting, fever and jaundice. Thirty nine percent of the patients were icteric at presentation and 48% of the patients were febrile (100^0 -103^OF). Abdominal tenderness was present in 78.40% of the patients. Similar result was also found in other studies[4,12]. In this study 25.50% of the patients gave history of worm emesis at the time of acute abdominal pain. Similar results have been reported in another study, they stated that such a history should always be sought in patients with suspected BPA[1].

In this study, history of previous biliary tract disease or surgery was found only in 11.70% cases though in other studies majority of the patients with BPA had history of surgery on the biliary tree, including cholecystectomy or sphincteroplasty[1,4,12]. Cholecystectomy changes the dynamics of the bile duct, favoring the migration into the biliary tree[1].

The diagnosis of BPA requires a high degree of suspicion. The worm can be missed because they move freely in and out of the biliary tree. In this study, ultrasonography could correctly diagnose BPA in 85.70% of cases. Similar result was also found in other studies[1,9]. Ultrasonography was false positive in 9.30% cases. However, no false-positive or false-negative cases were encountered in two studies[15, 16]. But in another study false-positive diagnoses were made at ultrasonography in four out of 12 cases (33.3%)[17].

In this study, most common site of round worm was common bile duct as recorded on other study, and it is seen that worms are rarely found in gall bladder or intrahepatic ducts or pancreatic ducts.[6]

Most common presentation was biliary colic, but good number of patients presented with cholangitis, ascaris induced acute pancreatitis, acute acalculous cholecystitis and liver abscess. In this study, 55% of the patient presented with different hepatobiliary and pancreatic complications of BPA. Similar rates of complications were also seen in other studies[6, 7]. After invading the bile ducts round worms sometimes may cause biliary obstruction, biliary colic, pyogenic cholangitis and septicaemia[13,19]. Worms may induce acute pancreatitis by blocking either bile or pancreatic ducts. Worms in the common bile duct usually block the cystic duct opening and cause distension of cystic duct and gall bladder, which lead to episodes of acute cholecystitis. However, worms in the bile and hepatic ducts may cause hepatic abscess[1, 10, 12].

A few cases of choledo-cholithiasis, RPC and papillary stenosis (organic) were found which were

associated with BPA. The association between ascariasis and intrahepatic stones has also been noted in other part of South-East Asia and South Africa, and ascaris debris and ova are found in stones from patients from these areas. Ascaris related biliary stones are usually of the pigment type and aided in their formation by factors such as bile stasis and ascending bacterial infection. It is estimated that in endemic countries ascaris ova or an immature worm is the cause of stone formation in 10 to 60% of patients[1]. RPC is found almost exclusively in South-East Asia. Parasitic infestation and malnutrition play a role. Parasitic infestation of the biliary tree, mostly by liver flukes and round worms, may initiate epithelial damage and biliary obstruction that ultimately lead to RPC. Analysis of the pigment stones in the RPC has also shown the presence of parasite debris and ova, which may act as a nidus for stone formation[19]. It has been reported that more than 5% of the patients with BPA develop the syndrome of RPC over two years of follow up[6]. Recurrent passage of worms through the papilla can cause papillitis that may lead to papillary stenosis.

Removal of worms was done mostly endoscopically. Spontaneous expulsions of worms occurred in 3.90% of cases. Overall endoscopic success rate was 93.90% which is similar to other studies[6, 20, 21]. Failure was due to presence of worms in the gall bladder or presence of intrahepatic calculi with stricture. Endoscopic extraction of the worms from the papilla leads to rapid relief of symptoms. Rapid relief of symptoms also occurs in patients with ascaris induced acute pancreatitis after extraction of worm from the papillary orifice[13]. As the complications of BPA may be quite high (94.70%), early intervention is necessary[6].

The patients who had endoscopic sphincterotomy had frequent and recurrent invasion of the biliary tree by worms; the wide opening facilitated the entry of worms into the biliary tree[13]. In this study, endoscopic sphincterotomy was done in 87.50% of cases among the patients who were treated with endoscopic removal. In case of BPA whether the worms are dead or alive, whole or fragmented, endoscopic sphincterotomy is necessary not only to remove stones, worms or parts of the worms but also to clean the bile duct with saline solution in case of purulent cholangitis[6].

Endoscopic removal after papillotomy in most of the cases was safe. Complications occurred in three cases (6.50%) which is similar to other studies and this technique is recommended as the preferred treatment of this pathology[6, 22].

Mean duration of hospital stay after endoscopic treatment was 2.6 days. Similar result was also seen in another study where mean duration of hospital stay was 3.1 days in endoscopically treated patients with BPA.[20] The mean duration of hospital stay in patients treated with conservative treatment was 5.1 days[4]. Endoscopic management greatly shortened hospital stay of BPA cases[20].

During the average follow-up period of 12.4 months recurrence of BPA occurred in four patients (7.80%). Recurrence rate in a study conducted in India was 18% over two year's followup.[13] But in another study conducted in a non-endemic area of India similar recurrent rate was found[23]. Less recurrence rate in this study may be due to routine prescription of anthelminthics after every two months and shorter follow-up period.

BPA may cause different hepatobiliary and pancreatic complications. In this part of the world, recurrent attacks may lead to RPC, choledocholithiasis and organic papillary stenosis. Ultrasonography is a simple, rapid, and noninvasive method for diagnosis and follow-up of the patients with BPA. As complications from the BPA are quite high, an urgent ERCP should be performed if BPA is suspected. ERCP and endoscopic extraction of worms are safe with a very low morbidity, shorter duration of hospital stay and a high rate of success. In addition, all patients should receive anthelminthic treatment regularly at least every two months to ensure a worm free intestinal tract specially those from rural areas of the country.

Acknowledgement:

We are grateful to Major General Prof. Sarder Amir Ali (Rtd), Director General of BIRDEM Hospital and Dr. Shafiuddin Mahmood Hussain, Managing Director of Gastro-Liver Hospital and Research Institute for allowing us to carry out this study in their institutes.

References:

1. Sandouk F, Haffar S, Zada M M, Graham D Y, Anand B S. Pancreatic-biliary ascariasis: experience of 300 cases, Am J Gastroenterol 1997; 92: 2264-7.
2. Khuroo MS, Ascariasis. Gastroenterol Clin North Am 1996; 25: 553-77.
3. Khuroo M S, Zargar S A, Yattoo G N, Koul P, Khan B A, Dar M Y, et al. Ascaris-induced acute pancreatitis. Br J Surg 1992; 79: 1335-8.
4. Gonzalez A H, Regalado V C, Ende J V D. Non-invasive management of Ascaris lumbricoides biliary tact migration: a prospective study in 69 patients from Ecuador. Trop Med Int Health 2001; 6: 146-50.
5. Uysal G, Kosebalaban O, Guven A. Biliary ascariasis. Indian J Pediatr 2001; 68: 1165-6.
6. Toshaiki K, Valdez E, Ustiniano M, Duran A, Gutierrez R, Biliopancreatic ascariasis: the role of endoscopy, In: Fujita R, Nakajawa S (editors). Recent Advances in Gastroenterology Tokyo: Springar, 1999. pp-73-80.
7. Beckingham I J, Cullis S N R, Krige J E J, Bornman P C, Terblanche J, Management of hepatobiliary and pancreatic Ascaris infestation in adults after failed medical treatment. Br J Surg 1998; 85: 907-10.
8. Gilles H M. Soil transmitted helminthes (Geohelminths). In Gordon Cook (editor). Manson's Tropical Diseases. Twelvth edition, London: W. B Saunders; 1996. pp-1374-81.
9. Khuroo M S. Hepatobiliary and pancreatic ascariasis. Indian J Gastroenterol 2001; 20 Suppl 1: C28-32.
10. Kamath P S. Hepatobiliary and pancreatic ascariasis. Indian J Gastroenterol 1991; 10: 137-9.
11. Hasan M, Rahman MT, Khorshed Alam AKM , Azad Khan AK. Endoscopic retrograde cholangiopancreatography: Experience of 100 cases at IPGMR. J Bangladesh Coll Phys and Surg 1990; 8: 8-13.
12. Bahu M da G S, Baldisseroto M, Custodio C M, Gralha C Z, Mangili A R. Hepatobiliary and pancreatic complications of ascariasis in children: a study of seven cases. J Pediatr Gastroenterol Nutr 2001; 33: 271-5.
13. Khuroo MS, Zargar SA, Mahajan R. Hepatobiliary and pancreatic ascariasis in India. Lancet 1990; 335: 1503-6.
14. Solaiman I. Ultrasonographic presentations of ascariasis: a study of 24 cases. Bangladesh Journal of Ultrasonography 2001; 8: 13-15.
15. Mani S, Merchant H. Sonographic evaluation of biliary ascariasis. Australas Radiol 1997; 41: 204-6.
16. Ali M, Khan A N. Sonography of hepatobiliary ascariasis. J Clin Ultrasound 1996; 24: 235-41.
17. Schulman A, Loxton A J. Sonographic diagnosis of biliary ascariasis. Am J Roentgenol 1982; 139: 485-9.
18. Khuroo M S, Zargar S A, Mahajan R, Bhat R L, Javid G. Sonographic appearances in biliary ascariasis. Gastroenterology 1987; 93: 267-72.
19. Mahadevan U, Bass N M. Sclerosing cholangitis and recurrent pyogenic cholangitis. In: Sleisenger MF, Fordtran BFS (editors). Gastrointestinal Liver Disease, Seventh edition, Vol.1. Philadelphia: W. B. Saunders, 2002. pp-1145-48.
20. Hoi M T, Desjeux A, Bach T T, Barthet M, Grimaud JC. Endoscopic management of biliary and pancreatic ascariasis in Vietnam. Report of a series of 91 cases. Gastroenterol Clin Biol 2002; 26: 968-72.
21. Kamiya T, Justiniano M, Duran A, Uechi C. Biliopancreatic ascariasis: endoscopic approach. J Gastroenterol 2002; 37 (Suppl 13): 97-9.
22. Eugene P, Dimagno, Chari S. Acute pancreatitis. In: Sleisenger MF, Fordtran BFS (editors). Gastrointestinal and Liver Disease, Seventh edition, vol.1. Philadelphia: W. B. Saunders, 2002. pp-921.
23. Misra S P, Dwivedi M. Clinical features and management of biliary ascariasis in a non-endemic area. Postgrad Med J 2000; 76: 29-32.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 23, No. 2, May 2005

Iatrogenic Genitourinary Fistulae: A Survey in Khulna Medical College

S NAHAR[a], MK SARKAR[b], KP DAS[c], N AKTAR[d]

Summary:

It was a retrospective study done in Khulna Medical College, Khulna, Bangladesh from January 2001 to July 2004. Within this time 44 cases of genito-urinary fistulae (GUF) were treated, 15 vesico-vaginal fistulae were obstetric in origin and rest 29 were iatrogenic fistulae. Highest incidence was vesico-vault and vesico-vaginal fistulae, next was uretero-vault fistula which was the sequlae of surgery mostly done by non-gynaecologist and non-specialist (about 90%) and by obstetrician and gynaecologist (about 10%). Ninety three percent fistulae developed in rural and urban clinics and 6.80% in hospitals. The results of the treatment were excellent. Technical improvement of the surgery and referral to specialized centre for fistula management certainly improves the success and diminishes the suffering of the patients.

(J Bangladesh Coll Phys Surg 2005; 23 : 68-71)

Introduction:

Genitourinary fistula (GUF) is a major problem in many developing countries, specially the vesico-vaginal fistula (VVF) commonly caused by prolonged obstructed labor is one of the worst complications of child birth. Urogenital fistulae are not uncommon consequences of gynecological surgery. Iatrogenic fistulae due to gynecological surgery generally appear from three days to six weeks after surgery and the communication tracts are uretero-vault, vesico-vault and vesico-uterine. Most authors quote an incidence rate for VVF after total abdominal hysterectomy (TAH) to be 0.5-2%, others suggest only a 0.05% incidence rate of injury to either the bladder or ureter.[1] Lee, in a series of 35,000 hysterectomies, found that more than 80% of genitourinary fistula were due to gynecological surgery for benign diseases[2]. Uncomplicated TAH accounted for more than 70% of these surgeries. The indications of these are pelvic inflammatory disease (PID), endometriosis, dysfunctional uterine bleeding (DUB), fibroid and prolapse. The purpose of this study was to know the aetiological background of iatrogenic fistula, its prevention and management in Khulna Medical College, Khulna, Bangladesh.

a. Dr. Shamsun Nahar, Associate Professor, Obstetrics & Gynaecology, Khulna Medical College, Khulna, Bangladesh.
b. Dr. Mrinal Kumar Sarkar, Assistant Professor, Obstetrics & Gynaecology, Khulna Medical College, Khulna, Bangladesh.
c. Dr. Krishna Pada Das, Assistant Register, Khulna Medical College, Khulna, Bangladesh.
d. Dr. Nasrin Aktar, Consultant, Obstetrics & Gynaecology, Khulna Medical College, Khulna, Bangladesh.

Address of correspondence : Dr. Shamsun Nahar, Associate Professor (Obstetrics & Gynaecology), Khulna Medical College, Khulna, Bangladesh.

Materials and method:

This was a retrospective study done in Khulna Medical College (KMC) during January 2001 to July 2004. The patients presented with symptoms of continuous dribbling of urine per vagina or continuous dribbling of urine with normal urge of micturation following abdominal or vaginal surgery. The nature of the previous surgery was explored to know whether it was caesarean section or caesarean subtotal or total hysterectomy, total time required to the development of fistula, the place of surgery i.e clinic or hospital and the qualification of surgeons. Clinical examination was done under general anaesthesia to know the position, size, and number of fistulae, associated fibrosis and vaginal stenosis. Dye test was done for confirmation in few cases. Special investigations like intravenous urography (IVU) and cystoscopy were done for confirmation of GUF when needed. Local repair, reimplantation of ureter into bladder or repair by transvesical route under general anaesthesia was done. All cases were followed upto three months.

Results:

Retrospective analyses of 44 cases of genito-urinay fistula were done in KMC during the period of January 2001 to July 2004. The age range of the patients was 15 - 50 years. Fifteen cases were having obstetric fistulae due to pressure necrosis from obstructed labor and iatrogenic fistulae were found in 29 cases (Fig-1). Table-I presented the characteristics of study patients. Among patients having iatrogenic

fistulae 16 were having vesico-vault fistula and vesico vaginal fistula 12 uretero-vault fistulae and one vesico-uterine fistula. Three fistulae resulted from surgery by obstetric specialist, seven cases were done by general surgeon and 19 cases were by unskilled non-specialist hand. More than 93% surgery was performed in clinic and 6.8% in hospitals. Local repair was done in 15 cases (51.70%), ureter reimplantation in 10 (34.40%) and transvesical repair in one (3.40%) case. In recently developed three cases each has uretero-vault fistula, VVF and vesico-uterine fistula. In uretero-vault fistula and VVF fistula the track was closed within 90 days and 45 days respectively but in vesicouterine fistula, whose complaint was menouria ,the patient refused further surgical intervention.

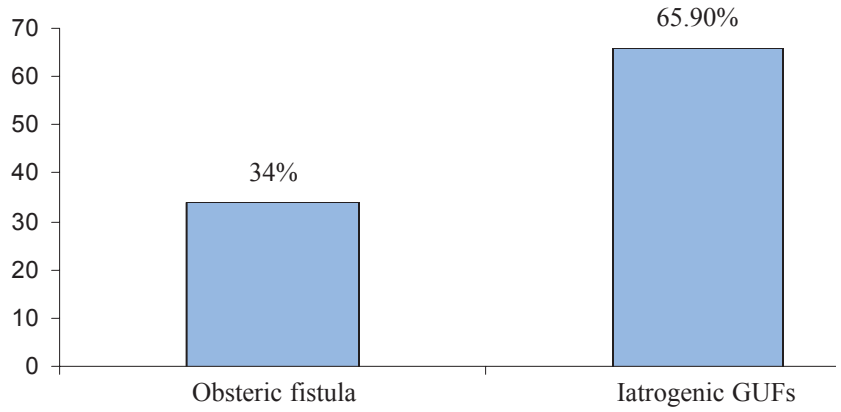

Figure -1: *Presented the obstetric and iatrogenic GUFs.*

Table-I

The types, nature of surgery, qualification of surgeons and place of surgery

Types of fistula (n – 29)	Causes of IGUF	Qualification of the surgeon	Place of operation
Vesico-vault and vesico-vaginal fistula (n-16)	Gynaecological surgery: TAH – 8 Vaginal hysterectomy - 1. Obstetric surgery, Caesarean subtotal / total hysterectomy – 7.	Unkilled or non specialist – 11 Specialist in General Surgery – 4 Specialist in Obs. & Gynae – 1	Clinic (n – 14) Hospital (n – 2)
Utetero-vault fistula (n-12)	Gynaecological surgery total abdominal hysterectomy (n-12)	Non specialist – 8 Specialist in general surgery – 3 Specialist in Obs. and Gynae – 1	Clinic (n – 12)
Vesico-uterine fistula (n – 1)	Third Caesarean section	Specialist in Obs. and Gynae – 1	Clinic (n – 1)

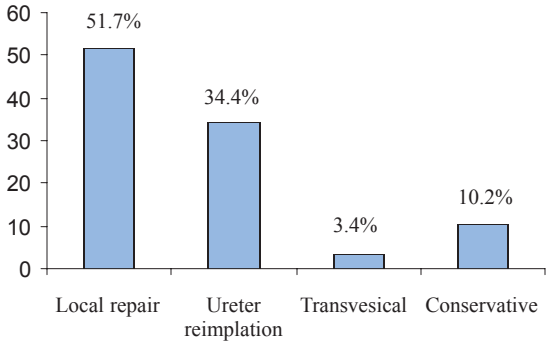

Figure-2: Presents the management of IGUF

Discussion:

Urogenital fistula is uncommon consequence of gynaecological surgery. Vesico-vaginal fistula due to gynaecological surgery generally appears 1 - 6 weeks after surgery and recurrent fistula within three months of repair[3].

This retrospective analysis of GUFs cases in Khulna Medical College it was found that in recent few years the iatrogenic fistula is more common than the obstetric fistula. A UNFPA survey estimates that over two million women in Bangladesh suffer from obstetrical fistula. Over 88% of deliveries in the country still take place in inexpert hands. Actually there is no report of development of iatrogenic fistula in our country which was the sequlae of gynaecological surgery by untrained and inexperience hands. In this series 65.50% of fistulae were developed by unskilled practitioners, general surgeon in 24% and by obstetrician and gynecologist in 10.30%; 93% of operations were performed in clinics and 6.80% were in hospitals. Recently, the obstetric fistulae are gradually declining in number due to development of trained personnels by the activities of comprehensive emergency obstetric care (EOC), improvement of spinal anaesthesia and blood transfusion facilities. On the other hand, iatrogenic fistulae are gradually increasing in its incidence. Most patients live in rural areas and illiterate, and they have little basic knowledge of the disease. They have no idea who is the right person for consultation. In obstetric fistula, more than 50% of the women were deserted by their husbands after the fistula developed and about iatrogenic fistula, the family were worried and seek for medical advice in 100% cases. Treatment started within six months of fistula development. Controversy surrounds the length of delay between diagnosis and surgical repair of GUFs. Analysis of the data showed that no definition has been established for early and late intervals. Traditionally, operation time was in the range of 8 to 12 weeks interval between index surgery and repair. O'conor agrees that the exact timing for repair depends on the tissue health. Most of his patients were brought to surgery approximately three months after index surgery.[4] All cases were repaired after three months from the index surgery. GUF in developing countries are attributed to inadvertent bladder injury during pelvic surgery (90%)[5,6]. It involves relatively limited focal bladder injury leading to smaller VVFs than those are observed in obstetrics fistulae. Numerous authors highlighted the risk of various types of bladder trauma during pelvic surgery. Such injuries include unrecognized intraoperative laceration of the bladder, bladder wall injury from electrocautery or mechanical crushing and the dissection of the bladder into an incorrect plane, causing avascular necrosis.[7,8,9] Suture placement through the bladder wall itself may not play a significant role in VVF development. However, the risk of formation of a haematoma or avascular necrosis after a suture is placed through the bladder wall can lead to infection and abscess of bladder wall. This wall defect permits the escape of urine into vagina and may be followed by an eventual epithelization of the track. Symmonds evaluated 800 GUFs over a 30 year period at the Mayo clinic, 85% of the VVFs were related to pelvic operation and 75% were related to hysterectomy, and 50% being secondary to simple uncomplicated total hysterectomy or vaginal hysterectomy.[10] The patients in this study are operated by local repair, ureter reimplantation into bladder and transvaginal repair in 92% cases and the success rate was 100%, and among conservatively managed three cases one developed VVF after vaginal hysterectomy and one uretero-vault fistula; the fistula tract spontaneously closed within three months. One case of menouria had previous three caesarean section and she refused to further surgery. She was advised to use continuous oral contraceptive pill. Oral oestrogen tablet was used to improve the tissue vascularization and healing in two postmenopausal patients.

GUFs are hidden tragedy for the patient and her family and for the treating surgeons. To reduce the incidence of GUFs medical ethics should be followed by all physicians. Government should take initiatives for improving the training facilities both for government and non-government doctors. Periodic follow up of the service quality of private clinics will reduce the incidence of iatrogenic genitourinary fistulae.

Reference:

1. American College of Obstetricians and Gynecologists. Genitourinary fistulas. ACOG Technical Bulletin 1985; 83: 1-6.
2. Lee RA, Symmonds RE, Willams TJ. Current status of genitourinany fistula. Obstet Gynecol 1988; 72: 313-9.
3. Flynn MK, Amundsen CL. Delayed presentation and successful repair of a recurrent vesico-vaginal fistula after hysterectomy and primary abdominal repair. Int Urogynecol J 2004; 15: 53-5.
4. O' conor VJ Jr. Review of experience with vesicovaginal fistula repair. J Urol 1980; 123: 367 - 9.
5. Rizvi JH. Genital fistulae, A continuing tragedy. J Obstet Gynaecol Res 1999; 25: 1-7.
6. Tancer ML. Observations on prevention and management of vesico-vaginal fistula after total hysterectomy. Surg Gynecol Obstet 1992; 175: 501 - 6.
7. Phillips J. Laparoscopic repair of post-hysterectomy vesico-vaginal fistula: Two cases reports. Gynaecol Endos 1996; 5: 123 - 4.
8. Smith GL, Williams G. Vesico-vaginal fistula. BJU Int 1999; 83: 564 - 9.
9. Stovsky MD, Ignatoff JM, Blum MD. Use of electro-coagulation in the treatment of vesico-vaginal fistulas. J Urol 1994; 152: 1443 - 4.
10. Symmonds RE. Incontinence, vesical and urethral fistulas. Clin Obstrt Gynecol 1984; 27: 499 - 512.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 22, No. 2, May 2005

Traumatic Pancreatic Pseudocysts : Personal Experience and Review of Current Management

RH JOARDER

Summary:

Posttraumatic pseudocysts of pancreas are rare and presentation at times masquarads acute abdominal emergencies leading to untimely laparotomy. Critical analysis of the patients and review of literatures will be helpful in grading and managing traumatic pancreatic conditions. Retrospective review of case reports is done. Six patients with pancreatic pseudocysts were treated. Elective surgery in two patients gave excellent result; one patient improved with conservative treatment. Three patients underwent emergency surgery. One of these patients had uneventful recovery, another developed pancreatic fistula and the other expired in the third week due to DIC.

(J Bangladesh Coll Phys Surg 2005; 23 : 72-75)

Introduction:

Traumatic pseudocysts of pancreas are uncommon clinical entity and constitute only 1 to 2% of all abdominal injuries[1]. High index of suspicion can help in making early diagnosis and to avoid untimely laparotomy. Deep location of the organ in the retroperitonium may make the diagnosis difficult and therefore delayed. Considerable force is necessary to traumatize pancreas such force often damage other organs. Release and activation of enzymes can cause auto digestion of pancreas and surrounding structures. Associated major vascular and visceral injury result in high mortality. Experience of any one surgeon in managing traumatic pancreatic pseudocysts will be small. Successful management of pancreatic trauma will therefore be based on accrued published experiences of others. Experience with the management of six such patients of pancreatic pseudocyst developed following blunt abdominal trauma is reported.

Materials and method:

The case records of six patients diagnosed and managed as traumatic pancreatic pseudocysts were reviewed. Patients with pseudocysts presented to hospital at seven days, three weeks and seven months after blunt abdominal trauma. Elective treatment in these three patients was based on clinical diagnosis supported by ultrasonography (USG) in all and ERCP in one patient (Fig.-1). In other three patients, pancreatic trauma was noticed at emergency laparotomy. Fall from height, kick, bicycle handle bar, fall on projecting object and road traffic accidents were different injury agents. Details of patient are given in Table-I.

Table-I

Patient characteristics of traumatic pancreatic pseudocysts (n=6)

Pt. no.	Sex	Age (year)	Trauma agent	Delay at presentation	Size	Site	Name of operation	Outcome
1	M	16	Fall from height	3 weeks	20 x10 cm	Head	Cysto-gastrostomy	No recurrence. Asymptomatic at 6 months follow up.
2	F	29	Kick	7 months	20x 10 cm	Body	Cysto-gastrostomy	Asymptomatic at 1 year follow up.
3	M	13	Bicycle handle bar	8 hours	10 x 20 cm	Body	Exploratory laparotomy.	Cyst disappeared spontaneously
4	M	16	Fall on projecting ends of fencing	7 days	10 x 8 cm	Body	Expectant	Improved
5	M	32	Road traffic accident	3 days	12 x 8 cm	Body	Distal pancreatectomy and splenectomy	Pancreato-cutenous fistula that resolved spontaneously
6	M	30	Road traffic accident	4 hours	12 x 8 cm	Body	External drainage only	Expired on 14th postoperative day due to uncontrolled bleeding from wound site, haematemesis and melaena

Address of correspondence : Dr. Ruhul Hassan Joarder. Assistant Professor of Surgery, Mymensingh Medical College, Mymensingh.

Case one:

A 16 year old boy was admitted with a painful swelling in the left upper abdomen which developed following fall from a mango tree, from about 20 feet height. The swelling grew very rapidly over a period of four days. Nausea and vomiting accompanied the swelling. Scratch mark of trauma was evident on the skin overlying a bulge occupying the left hypochondrium measuring about 20cm/10cm in dimension. It was tender, outline not sharply demarcated, firm and did not move with respiration. Plain abdominal radiography showed a large soft tissue mass with displacement of colonic loops towards the right side. Ultrasonogram confirmed the diagnosis of pancreatic pseudocyst. His pseudocyst required cysto-gastrostomy, done four weeks after diagnosis. He was well one year postoperatively.

Case two:

A 29 year old housewife presented with a painful swelling in the epigastrium of four months' duration. Seven months back her husband kicked her in the upper abdomen. The swelling gradually enlarged to occupy the epigastrium, umbilical and left hypochondriac region. She also had complaints of loss of appetite, weight loss and vomiting after taking meals.

Ultrasonography of upper abdomen showed an echolucent mass in the pancreatic region suggestive of pancreatic pseudocyst. ERCP showed obstruction of main pancreatic duct with leakage of contrast into the cyst. (Fig.-1). Elective cysto-gastrostomy gave excellent result with relief of pain, swelling and vomiting. Her appetite improved and she was able to take adequate meals.

Case three:

A 13 year old boy presented with abdominal pain, fever and respiratory distress developed following a blunt abdominal trauma sustained 36 hours back by bicycle handlebar, which he was riding.

He had tachycardia, tachypnoea and raised temperature (102ºF). His abdomen was distended, tense and tender along with positive rebound tenderness and absence of bowel sound.

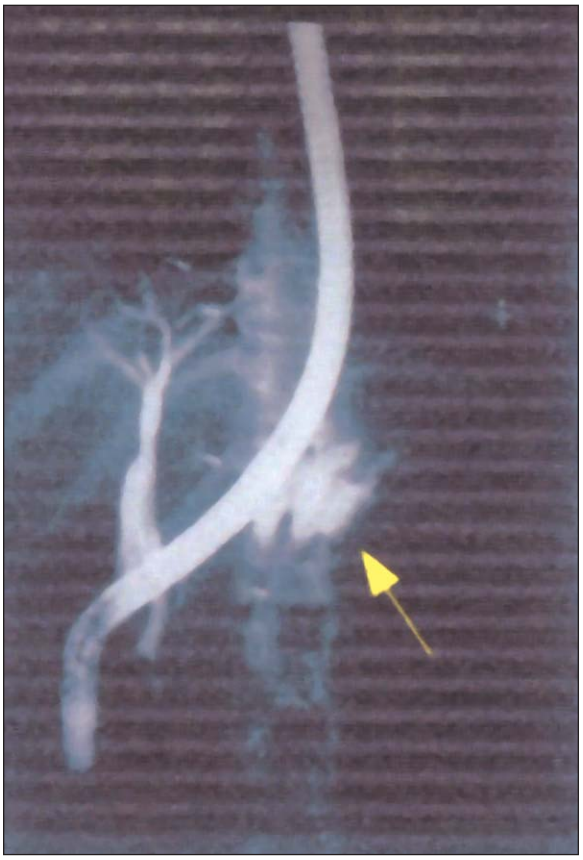

Figure-1: *ERCP showing obstruction of main pancreatic duct with leakage of contrast into the pseudocyst.*

Plain abdominal x-ray was unremarkable. Exploratory laparotomy revealed about 600 ml of haemorrhagic non-smelling intraperitoneal fluid and a large (10 cm x 8 cm) pseudocyst in the region of the swollen oedematous pancreas. Other viscous were intact. His recovery was good and the cyst disappeared by six weeks post-operatively.

Case four:

A 16 year old boy fell down by the abdomen on the projecting ends of a roadside bamboo fencing while his rickshaw was knocked from behind by a babytaxi. About a week after the incidence, he presented with a diffuse tender swelling involving epigastrium and umbilical region. USG showed swollen head of the pancreas along with a pseudocyst (10cm x 8cm) in that region. He improved with conservative treatment and was well at his follow up at six months.

Case five:
A 32-year-old man sustained blunt abdominal trauma 3 days back from road traffic accident. On admission tachycardia, fever and distended tender abdomen demanded urgent laparotomy. His peritoneal cavity contained plenty of haemorrhagic fluid and a large retroperitoneal haematoma. Pancreas was exposed in all surfaces. It was found severely lacerated at the level of body on the left of the vertebral column (type-II injury). Distal pancreatectomy with splenectomy was done. On the first postoperative day intraperitoneal drainage tube drained about 600 ml of foul smelling dirty white fluid. In the subsequent days, efflux reduced to about 100 ml per day. The drain tube was removed on the second week. The fistula persisted for another week there after and stopped draining spontaneously without additional measure.

Case six:
A 30-year-old public bus driver sustained steering wheel injury to the abdomen and compound fracture of right femur. He was resuscitated with blood transfusion and intravenous fluids. Laparotomy, done four hours after admission, revealed a large retroperitoneal haematoma in the region of the pancreas and haemorhagic intraperitoneal fluid. Pancreas appeared swollen and contused, particularly in the head region (type-III injury). Only external drainage was done using urine drainage bag. In the early postoperative days the drainage bag contained nonsmelling dirty white fluid in an amount close to one liter per day. Later his high output pancreatic fistula used to drain about 300 ml/day. His initial postoperative improvement was soon masked by malnutrition resulting in gross weight loss, abdominal wound dehiscence and sepsis. On the twelvth postoperative day he had haematemesis, melaena and abdominal wound-site bleeding. He developed DIC with altered prothrombin time, raised FDP and thrombocytopaenia. He died on the fourteenth day after operation from uncontrolled gastrointestinal and wound-site bleeding.

Discussion:
Isolated pancreatic injury is rare, seen only in 0.4% of trauma victims, 20% of them may develop pseudocyst[2]. Posttraumatic pseudocysts are usually associated with pancreatic duct injury and may present late[3]. Duct injury was noted in four of six patients. Two of the patients presented late at three weeks and seven months after injury. Successful surgical management of pancreatic trauma depends on precise delineation of duct injury. Either transduodenal cannulation of papilla of Vater or cannulation of the duct at pancreatic tail can obtain per-operative pancreatogram. Duodenal fistula from transpapillary pancreatography is potentially not a real problem. Bernie et al has reported reduction in mortality rate after the introduction of operative pancreatography[4]. Others have judged this procedure to be complicated and unnecessary in acute situation[5,6]. Preoperative ERCP is recommended in stable patients to avoid potential complication of duodenotomy, but wide spread use is limited by logistic and technical factors, particularly in trauma situation[7,8]. Preoperative ERCP in the second patient showed the precise site of major pancreatic duct injury with leakage of contrast into the pseudocysts and non-visualization of the duct in the body and tail i.e. distal to head region (Fig.-1) indicating obstruction at this level.

The basic plan of management of pancreatic trauma have remained unchanged. In the presence of signs of peritoneal irritation in a trauma victim there is no reason to delay laparotomy to perform pancreatic imaging. At operation, all surfaces of the pancreas should be fully exposed which is sufficient to demonstrate any significant injury. Lucas has divided isolated pancreatic injury into three groups to plan appropriate management[9]: superficial contusion with minimum damage (grade-I), deep laceration or trans-section of left pancreas (grade-II) and severe injury of the head of pancreas (grade-III). Combined pancreatic and duodenal injury is classified separately and variety of procedures have been described based on diverting gastric contents away from the injured pancreas and draining or debriding pancreatic tissue[9,10]. This type of injury is more severe because of inevitable contamination and activation of leaking enzymes. External tube drainage should be the usual treatment in grade-I and grade-II injury, although distal resection may occasionally be required. Grade-III and more complex injury require skilled surgical management. The mortality rate associated with resection of head of pancreas should be prohibitive and patient with grade-III injury should undergo

external or Roux en Y drainage or duodenal exclusion as deemed necessary on an individual basis. Nevertheless unless resection appears inevitable and safe the primary management probably is external drainage. A fistula may result but this will often close spontaneously or with addition of octreotide (long acting somatostatin) therapy and any of which persists may be diverted into Roux en Y loop.[11] Few patients now die as a direct result of pancreatic injury.Among those who die, three-quarter die of haemorrhage from liver or major vessels and of the remainder half die from organ failure or other miscellaneous causes of trauma[12]. The sixth patient had type-III injury involving the head of the pancreas. He expired on the fourteenth post-operative day due to DIC and multiple organ failure.

Post-trauma pseudocyst often presents late. USG and CT scan fail to diagnose duct Injury[3,1]. ERCP demonstrates site and severity of duct injury and can help in planning management. Posttraumatic pseudocyst in adult and in children due to peripheral duct injury may resolve spontaneously[13]. Percautenous aspiration or continuous catheter drainage can be used to treat those associated with distal duct injury. Early reports indicate endoscopic drainage with or without placement of nasocystic drainage tube or stent in selected patient is feasible and safe.[3] Proximal duct injury however requires surgical intervention either in the form of resection or internal drainage depending on maturity of the cyst wall.

References:

1. Lewis G, Krige SEG, Boreman PC, Terbalanche J. Traumatic pancreatic pseudocysts. Br J Surg 1993; 80: 89-93.
2. Akhrass R, Yaffe MB, Brandt CP, Reigle M, Fullon WF jr, Malangoni MA. Pancreatic trauma- A 10 year multi institutional experience. Am J Surg 1997; 63: 598-604.
3. Funnel IC, Borreman PC, Krigr JEJ, Benigfield SJ, Terbalanche J. Endoscopic drainage of pancreatic pseudocyst. Br J Surg 1994; 81: 879-881.
4. Berni GA, Bandyk DF, Oreeskovich MR, Carrico CJ. Role of intra operative pancreatography in patients with injury to pancreas. Am J Surg 1982; 143: 602-5.
5. Faliciano DV, Martin TD, Cruse PA, et al. Management of combined pancreaticoduodenal injuries. Ann Surg 1987; 205: 673-9.
6. Jones RC. Management of pancreatic trauma. Am J Surg 1985; 150: 698- 704.
7. Laraja RD, Lobbato VJ, Cassaro S, Reddy SS. Intraoperative endoscopic retrograde cholanngiopancreatography in penetrating trauma of the pancreas. J Trauma 1986; 26: 1146-7.
8. Haward SR, Lucas CE, Sugawa C, Ledgerwood AM. Emergent endoscopic retrograde cholangiopancreatography. Arch Surg 1989; 124: 745-6.
9. Lucas CE, Diagnosis and treatment of pancreatic and duodenal injury. Surg Clin North Am 1977; 57: 49-65.
10. Wilson RH, Moorehead RJ. Current management of trauma to pancreas. Br J Surg 1991; 18: 1196-1199.
11. Warshaw AL, Castillo CFD, Ratlner DW. Pancreatic cysts, pseudocysts and fistula. In: Zinner MJ (editor), Maingot's Abdominal Operations, vol. II Tenth edition. USA: Prentece Hall Interntional, 1997. pp- 1937.
12. CD Johnson. Pancreatic trauma. Br J Surg USA: 1995; 82: 1153-54.
13. Burnweit E, Weson D, Stringer D, Fritler R. Percautenous drainage of traumatic pancreatic pseudocyst in children. Trauma 1990; 30: 1273-7.

REVIEW ARTICLE

Carotid Artery Stenosis and Intervention: A Review

MA HAYEE[a], QD MOHAMMAD[b]

(J Bangladesh Coll Phys Surg 2005; 23 : 76-80)

Introduction :

There is no epidemiological data on the incidence or prevalence of cerebrovascular disease (CVD) in Bangladesh. Clinical experience indicates that this is likely to be similar to that of the Western countries. Even a few years back it was believed that stroke could not be treated. But recent advances in neuroscience and interventional neurology have changed the picture. Interventional neurology is a branch of neuroscience which has rapidly grown with the advances in interventional techniques in neurological diseases in the last decade. Stroke ranks third leading cause of death, after heart diseases and cancers. It is an important cause of disability in the elderly people[1,2]. Each year, more than 6,00,000 Americans suffer from acute ischaemic stroke resulting in death in more than 90,000 women and 60,000 men[3]. The average annual incidence of stroke in Japan is 3.94 for males and 2.52 for females per 1000 population with cases of cerebral infarction outnumbering cerebral haemorrhage[4]. Stenosis due to atherosclerosis commonly occurs to the carotid bifurcation but can also occur in the carotid siphon[5]. Atherosclerotic disease at the bifurcation of thee common carotid artery (CCA) is associated with 20 to 30% of ischaemic strokes[6]. Wepfer was the first in 1658, to recognize the significance of carotid artery obstruction and its relationship to underlying "fibrous masses" and thrombus[7]. Later, Hunt in the early twentieth century emphasized the importance of carotid artery atherosclerosis in cerebrovascular disease[8,9]. Fisher in 1951 recognized that the basic mechanisms causing focal cerebral ischaemia from carotid artery disease were embolization, decreased flow thought the carotid artery or both[10]. Atherosclerosis cannot be cured as yet, but its genesis can be modified by dietary changes, regular exercise and control of diabetes and hypertension. The effects of atherosclerosis can be changed by either endarterectomy or balloon angioplasty and stenting.

Evolution of carotid artery angiography and stenting:

Carotid revascularization has previously been done by carotid endarterectomy and was performed by neurosurgeons[11-13]. Other areas of the vascular territory even then had well established endovascular procedures[14,15]. The risk of stroke caused by embolization during the procedure prevented the application of this technology in the territory of the brain[16,17,18]. The distal vascular bed of coronary, aorto-iliac or subclavian are not as sensitive as that of central nervous system[19,20]. The benefit of carotid endarerectomy for the prevention of stroke in patients with extracranial carotid stenosis has been well established[21-24]. Endarterectomy is the accepted standard treatment of carotid stenosis[25,26]. This procedure was first started in 1950 by Eascott, De Bakey and Cooley[27]. After the landmark study of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and Asymptomatic Carotid Atherosclorotic. Study (ACAS), carotid endarterectomy has proved beneficial in reducing the stroke[21,23,28-29]. The patient inclusion criteria for the NASCET were very strict[2l]. Patients with one or more of the following conditions were excluded: (a) age more than 79 years, (b) restenosis following carotid endarterectoy, (c) tandem lesion, (d) high grade carotid stenosis with contralateral carotid occlusion, (e) radiation induced carotid stenosis, (f) no angiographic visualization of both carotids and their intracranial branches and (g) heart, lung, liver or renal failure. There were certain groups of patients which were temporarily excluded: (a) uncontrolled diabetes, (b) uncontrolled hypertension, (c) unstable

a. Dr. MA Hayee, Associate Professor and Head of the Department of Neurology, Sir Salimullah Medical College, Dhaka, Bangladesh.

b. Prof. QD Mohammad, Professor and Head of the Department of Neurology, Dhaka Medical College, Dhaka, Bangladesh

Address of correspondence : Dr. MA Hayee, Associate Professor and Head of the Department of Neurology, Sir Salimullah Medical College, Dhaka, Bangladesh.

angina, (d) myocardial infarction within previous six months, (e) contralateral carotid endarterectomy within four months, (f) signs of progressive neurological dysfunction and (g) major surgical procedure within previous 30 days. These patients could be included if the conditions resolved within 120 days.

Therefore many patients who suffered from symptomatic carotid artery stenotic disease could not be included in NASCET. By that time many neuroscientists have already acquired enough experience in cerebral artery manipulation by balloon angioplasty as a treatment of cerebral aneurysm. With this experience, many centres started angioplasty of carotid arteries only in NASCET excluded patients[30]. But follow-up angioplasty revealed a high percentage of restenosis within two years[29]. From 1995, different centers started angioplasty followed by stenting in NASCET excluded patients. Wholey et al in 2000 showed that by that time a total number of endovascular carotid procedures that have been performed worldwide included 5,210 procedures involving 4,757 patients [31]. There was a technical success rate of 98.4%. The peri-operative (from procedure to 30 days of procedure) stroke and death rate was 2.35% (stroke 1.49% and death 0.86%). But in NASCET trial, the peri-operative death and stroke rate was 5.8%. In addition, anaesthetic hazards and risks of a major operation are there. From the above result, the endovascular treatment of carotid stenosis is safe. But it is difficult to compare carotid artery stenting with endarterectomy without a randomized trial. A randomized trial has been designed and is going on. The superiority of the two procedures may be decided after the result of the study. But the people and neuroscientists are gradually incling towards endovascular treatment. "The Carotid Revascularization Endarterectomy versus Stent Trial" and " The Carotid and Vertebral Artery Transluminal Angioplasty Study" have explained the efficacy of percutaneous angioplasty and stenting and its role in stroke preventing[32'33]. A number of retrospective studies of percutaneous angioplasty and stenting, however, have reported superior results to those of carotid endarterectomy terms of stroke and mortality rates[34-37]. These favourable results seem to support at least a limited role for percutaneous angioplasty and stenting in the management of carotid stenosis.

Percutaneous transluminal angioplasty and stenting (PTAS) of carotid stenosis:

Presently majority of patients with carotid artery stenosis are treated by endarterectomy. because the result of randomized trials of angioplasty are pending. But the PTAS have dramatically altered traditional approaches to the management of both coronary and peripheral vascular diseases. The major advantage is that it is less invasive than conventional surgical procedures, offering benefits without the risks of general anaesthesia, and with lesser procedural morbidity and mortality, shorter hospital stay and lower cost. Although the traditional standard of care in treating cervical carotid stenosis is carotid endarterectomy, in certain subsets of patients (NASCET excluded), PTAS is the alternative measure. In those patients, carotid artery angioplasty and stenting has been shown to be effective and safe[38-41]. PTAS are commonly performed in Europe and America despite the lack of prospective randomized study comparing this modality with carotid endarterectomy.

The endovascular approach for the treatment of carotid stenosis began with the use of percutaneous balloon angioplasty in 1980[42]. In 1987, Theron et al published the first large series of internal carotid angioplasty in 48 patients with de novo atherosclerotic or post-surgical restenosis[43]. Since then many centres have reported their experiences with PTAS. With time there has been an increase in the knowledge base, catheter, balloon and stent technology, which has resulted in improved results of carotid angioplasty and stenting with low mortality and morbidity even in high-risk patients. The growth rate of the procedure is good. In June 1997, 2,047 PTAS procedures were done by the original 24 centres[44]. The number of PTAS procedures was increased to 2,591 in January 1998, a 27% annual growth rate. The current rate of annual growth in the original 24 centres is 47%.

The ideal patents for the endovascular stent placement are:

- Contralateral ICA occlusion;
- Restenosis following carotid endarterectomy[45,46];
- Radiation-induced carotid stenosis[47,48];
- Isolated high ICA lesion inaccessible for surgery[49];
- Numerous confounding medical comorbid conditions[50];
- Tandem lesion [30];
- Fibromuscular dysplasia[30];
- Unstable angina awaiting for coronary revascularization[51];
- Carotid stenosis associated with neck malignancy[52]; and
- Post traumatic carotid stenosis[52];

Patients who are not suitable for PTAS[51] are:

- Patients with femoral or iliac access problem;
- Patients with contraindications to anticoagulation therapy with heparins; and
- Patients with contraindication for antiplatelet therapy.

Recently, various devices have been used for thromboembolic protection during stenting. Use of these devices further reduces the risk of perioperative embolic stroke[53]. After the publication of Hobson's study result, PTAS for carotid stenosis is evolving from its initial[32] controversial situation to a reasonable alternative for treating carotid occlusive disease.

Atherosclerotic involvement of the intracranial internal carotid artery is the carotid siphon which is not also surgically approachable. It should be treated because 60% of those patients present with stroke[54]. PTAS is also performed in these cases to prevent stroke.

Suggested protocol for PTAS for Bangladesh:

All patients with symptomatic carotid stenosis should be included in this protocol. All patients selected for PTAS should undergo a through neurological examination before the procedure. Baseline investigations like CBC, PT, APTT, blood sugar, BUN, serum creatinine, ECG, X-ray chest should be done. A base line MRI/CT scan should be done to document any preexisting infarction. Oral aspirin 325 mg/day or clopidogrel 75 mg/day should be started at least three days prior to the procedure. After the procedure the patient should be maintained on 75 mg aspirin daily life long and clopidogrel 75 mg daily for three months. The procedure should be performed under local anaesthesia and conscious sedation should be used only if required. Right femoral approach should be ideal but left may be chosen when right femoral could not be used. The guiding catheter should be placed in the common carotid artery proximal to the stenosis. A baseline activated clotting time (ACT) should be performed before the procedure. A bolus dose of heparin (70 to 90 mg/Kg) would be given just before the inflation of balloon to achieve an ACT of two to 2.5 times of baseline. Atropine syringe should be attached to the IV line and to be injected only if patient develops bradycardia during balloon dilatation or stent placement. A 4-vessel cerebral direct subtraction angiogram in a minimum of two planes should be done to analyze the intracranial circulation. The stenosis should be crossed with a guide wire under road map image. Pre-dilatation should be performed if the stenosis is of high grade. Suitable stent should be placed. Heparin should not be given after placement of the stent and the effect of heparin already given should be allowed to dissipate naturally. At the end of the procedure a complete angiogram should be done to evaluate the condition of the stenosed area and identification of any embolic stroke during the procedure. The femoral sheath should be removed when ACT returns to baseline level. The patient should be under close observation for the day and discharged on the following day.

Conclusion:

Symptomatic carotid stenotic patients should be treated by PTAS. It is the need of time to develop a full-fledged interventional neurology unit.

References:

1. Easton JD. Current advances in the management of stroke. Neurology 1998; 51: S1 - S2.
2. Kuller LH. Incidence rate of stroke in the eighties: The end of the decline in stroke? Stroke 1989; 20: 841-43.

3. American Heart Association. 1999 Heart and Stroke Statistical Update. Dallus, Texus, 1998. pp 13-15.

4. Komachi Y, Tanaka H, Shimamoto H, Fisher C, Kuller LH, Davis SM, et al. A collaborative study of stroke incidence in Japan: 1975-1979. Stroke 1984; 15: 28-36.

5. Fisher C. Occlusion of the internal carotid arteries. Arch Neurol Psychiat 1951; 65: 346.

6. Donan GA, Davis SM, Chambers BR, Featherstone RL, Brown MM, Kuller LH, et al. Surgery for prevention of stroke. Lancet 1998; 351: 1372-73.

7. Guradjian. History of occlusive cerebrovascular disease: From Wepfer to Moniz. Arch Neurol 1979; 36: 340-43.

8. Chiari H. Uber das verhalten des teilungwinkeles der carotids communis bei der endarteritis chronica deformans. Ver Disch Path Ges Pathol 1905; 9: 326.

9. Hunt J. The role of carotid arteries in the causation of vascular lesion of brain, with remarks on certain special features of the syptomatology. Am J Med Sci 1914; 147: 704.

10. Fisher C. Occlusion of the carotid arteries. Arch Neurol Psychiat 1954; 72: 187.

11. Diethrich EB. Indications of carotid artery stenting: A preview of the potential derived early clinical experience. J Endovas Surg 1996; 3: 132-39.

12. European Carotid Surgery Trialists. Collaborative Group: MRC European Carotid Surgery Trial: Interim results for asymptomatic patients with severe (70-90%) or with mild (0-29%) carotid stenosis. Lancet 1991; 337: 1235-41.

13. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. JAMA 1995; 18: 1421-28.

14. Gibon C, Cannon C, Piana R, Davis SM, Coward LJ, Featherstone RL, et al. Angiographic predictors of reocclusion after thrombolysis: Results from the thrombolysis in myocardial infarction(TIMI) 4 trial. J Am Coll Cardiol 1995; 25: 282-89.

15. Coward LJ, Featherstone RL, Brown MM. Percutaneous transluminal angioplasty and stenting for carotid artery stenosis(Cochrane Review). In: The Cochrane Library, Issue 4. Chichester, UK: Jhon Wiley & Sons, Ltd. 2004. pp-653-57.

16. Grotta J. Elective stenting of extracranial carotid arteries [editorial). Circulation 1997; 95: 303-305.

17. Hopkins LS, Lanzino G, Mericle RA, Coward LJ, Featherstone RL, Coward LJ, Featherstone RL, et al. Carotid Intervention: A neurosurgeons perspective. J Invas Cardio11998; 10: 279-91.

18. Terada T, Higashida RT, Halbach VV, Coward LJ, Featherstone RL, Knobloch W, et al. Transluminal angioplasty for atherosclerotic disease of the distal vertebral and basilar arteries. J Neurol Neurosurg Psychiatry 1996; 60: 377-381.

19. Veen G, Meyer A, Verheugt F, Knobloch W, Lanzino G, Mericle RA, et al. Culprit lesion morphology and stenosis severity in prediction of reocclusion after coronary thrombolysis: Angiographic results of the APRICOT study. J Am Coll Cardiol 1993; 22: 1755-62.

20. Schiele TM, Niehues R, Knobloch W, Metzger G,Jacksch R. Interventions in carotid stenosis: carotid surgery, percutaneous transluminal baloon angioplasty and stent implantation. dications, results of clinical studies. An overview. Surg Clin Notrh Am 2004; 84: 1281-94.

21. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effects of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. N Eng J Med 1991; 325: 445-53.

22. Barnett HJ, Taylor DW, Eliasziw M, Lanzino G, Mericle RA, Schiele TM, et al. Benefits of carotid endarterectomy in patients with symptomatic moderate to severe stenosis. . N Eng J Med 1998; 339: 1415-25.

23. Executive Committee for the Asymptomatic Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. JAMA 1995; 273: 1421-18.

24. Europian Carotid Surgery Trialists' Collaborative Group. Randomized trial of endarterectomy for recently symptomatic carotid stenosis: Final results of the MRC Europian Carotid Surgery Trial (ECST). Lancet 1998; 351; 1379-87.

25. Zarins CK. Carotid endarterectomy, the gold standard. J Endovas Surg 1996; 3: 1015.

26. Ringleb PA, Kunze A, Allenburg JR, Hennerici MG, Jansen O, Maurer PC, et al. The stent supported percutaneous angioplasty of the carotid artery vs endarterectomy trial. J Endovas Ther 2003; 10: 1031-38.

27. De Bakey MH. Carotid endarterectomy revisited. J Endovas Surg 1996; 3: 4.

28. Clinical Advisory. Carotid endarterectomy for patients with asymptomatic internal carotid stenosis. J Neurol Sci 1995; 273: 1421-28.

29. Clinical Advisory. Carotid endarterectomy for patients with asymptomatic internal carotid stenosis. Stroke 1994; 25: 2523-24.

30. Guterman LR, Fessler RD, Hopkins LN. Cervical carotid revascularization. Neurosurg Clin North Am 2000; 10: 39-48.

31. Wholey MH, Wholey M, Mathias K, Schiele TM, Kunze A, Allenburg JR, et al. Global experience in cervical carotid stent placement. Cathet Cardiovas Interven 2000; 50: 160-67.

32. Hobson RW II, Broth T, Ferguson R, Kunze A, Allenburg JR, Mathias K, et al. Regarding "statement regarding carotid angioplasty and stenting". J Vas Surg 1997; 25: 1117.

33. Callow AD. Recurrent stenosis after carotid endarterectomy. Arch Surg 1982; 117: 1082-85.

34. Bettmann MA, Ketzen BT, Whisnat J, Mathias K, Broth T, Ferguson R, et al. Carotid stenting and angioplasty. Stroke 1998; 29: 336-38.

35. Yadav JA, Robin JS, Iyer, et al. Elective stenting of the extracranial carotid arteries. Circulation 1997; 95: 376-81.

36. Featherstone RL, Brown MM, Coward LJ; ICSS Investigators. International carotid stenting study: protocol for randomised clinical trial comparing carotid stenting with endarterectomy in symptomatic carotid artery stenosis. J Endovasc Ther 2003; 100: 1031-38.

37. Henry M, Amor M, Klonaris C, Henry I, Masson I, Chati Z, et al. Angioplasty and stenting of the extracranial carotid artery. Tex Heart Inst J 2000, 27: 150-58.

38. Hunt J. The role of carotid arteries in the causation of vascular lesions of the brain, with remarks on certain special features of the symptomatology. Am J Med Sci 1964; 147: 704.

39. Fisher C. Occlusion of the carotid arteries. Arch Neurol Psychiat 1954; 72: 187.

40. Fisher C. Occlusion of the internal carotid arteries. Arch Neurol Psychiat 1951; 65: 346.

41. Setacci C, Pula G, Baldi 1, de Donato G, Setacci F, Cappelli A, et al. Determinants of in-stent restenosis after carotid angioplasty : a case control study. Stroke 2000; 31: 1439.

42. Kerber CW, Hornwell LD, Loeden OL. Catheter dilatation of the proximal carotid stenosis during distal bifurcation endarterectomy. AJNR 1980; 1: 348-49.

43. Theron J, Raymond J, Casasco A, Broth T, Ferguson R, Pula G, et al. Percutaneous angioplasty of atherosclerotic and postsurgical stenosis of carotid arteries. AJNR 1987; 8: 495-500.

44. Vitek J, Iyer S, Roubin G. Carotid stenting of 350 vessels: Problem faced and solved. J Invas Cardiol 1998; 10: 311-14.

45. Lanzino G, Mericle RA, Lopes DK, Pula G, Casasco. A, Broth T, et al. Percutaneous transluminal angioplasty and stenting for recurrent carotid artery stenosis. J Neurosurg 1999; 90: 688-94.

46. Meyer FB, Piepgras DG, Fode NC, Surgical treatment of recurrent carotid artery stenosis. J Neurosurg 1994; 80: 781-87.

47. Loftus CM, Biller J, Hart MN, Casasco A, Broth T, Roubin G, et al. Management of radiation induced accelerated carotid atherosclerosis. Arch Neurol 1987; 44: 711-14.

48. Murros KE, Toole JF. The effect of radiation on carotid arteries: A review article. Arch Neurol 1989; 46: 449-55.

49. Lopez DK, Mericle RA, Lanzino G, Roubin G, Lopes DK, Pula G, et al. Carotid angioplasty and stenting before coronary artery bypass grafting [abstract]. Neurosurg 1998; 43: 686.

50. Harbaugh RE, Stieg PE, Moayeri N, Iyer S, Roubin G, Casasco A, et al. Case problem in neurological surgery: Clinicopathological review. Carotid coronary artery bypass graft conundrum. Neurosurg 1998; 43: 926-31.

51. Faggioli Gl, Curl R, Ricotta JJ. The role of carotid screening before coronary artery bypass. J Vas Surg 1990; 12: 724-31.

52. Prall JA, Breja KE, Coldwell DM, Casasco A, Lanzino G, Roubin G, et al. Incidence of unsuspected blunt carotid artery injury. Neurosurg 1998; 42: 495-98.

53. Henry M, Amor Max, Henry 1, Lanzino G, Mericle RA, Lopes DK, et al. Carotid stenting with cerebral protection: First clinical experience using the Purcu Surge guide wire system. J Endovas Surg 1999; 6: 321-31.

54. Caplan NR, Babikian V, Helgason C, Fessler RD, Hopkins LN, Zarins CK, et al. Occlusive disease of the intracranial vessel. Neurology 1985; 35: 975-82.

Robinow Syndrome (Foetal Face Syndrome) with Radiological Mesomelic and Rhizomelic Brachymelia of the Lower Limbs: A Case Report

BS NAHER[a], A RAHMAN[b], S AFROZA[c], A HOSSAIN[d], S TAHMINA[e], R JAHAN[f]

Summary:

Robinow syndrome was diagnosed in a newborn baby on the basis of clinical and radiological evidence. The syndrome in a rare entity and reportedly the first cure diagnosed in this country.

(J Bangladesh Coll Phys Surg 2005; 23 : 81-83)

Introduction :

Robinow syndrome is a rare inherited disorder. It was described by Robinow in 1969 as a syndrome of characteristic facies, micropenis, cryptorchidism in the male and mesomelic limb shortening[1]. The characteristic facial appearences (foetal face) are frontal bossing, macrocephaly, hypertelorism, wide palpebral fissures with downward slanting, short upturned broad nose with anteverted nares, hyperplastic alveolar ridges, long philtrum, small chin and triangular mouth with downward angles. In addition, hypoplastic genitalia, nevus flammeus, capillary haemangioma on the glabella, mesomelic brachymelia of forearm and small hands with clinodactyly are also noted[2]. The inheritance pattern in Robinow syndrome is not clear. Both autosomal dominant and recessive inheritance have been reported[3-6]. It has been suggested that more severe mesomelic brachymelia and more triangular mouth are indicative of autosomal recessive inheritance[7]. A newborn is described here with radiological rhizomelic and mesomelic brachymelia of the lower limbs. Probably cases with such diagnosis have not been reported before in this country.

a. Dr. Begum Sharifun Naher, MBBS, FCPS, MD (Paed), Asst. Professor (Paediatrics), SSMC & Mitford Hospital.

b. Dr. Abdur Rahman, DCH, DCN-Rutr., MSc in MCH, Senior Consultant (Paediatrics), MCHTI, Azimpur.

c. Prof. Syeda Afroza, MBBS, FCPS, DMEd (UK), MMEd (UK), Clinical Fellow of Neonatology (UK), Professor of Paediatrics and Joint Director, Institute of Child & Mother Health, Matuail, Dhaka.

d. Dr. Amir Hossain, MBBS, DCH, Medical Officer (Paediatrics), MCHTI, Azimpur.

e. Dr. Shah Tahmina, MBBS, Medical Officer (Paediatrics), MCHTI, Azimpur.

f. Dr. Rownak Jahan, MBBS, Medical Officer (Paediatrics), MCHTI, Azimpur.

Address of correspondence : Dr. Begum Sharifun Naher, MBBS, FCPS, MD (Paed), Asst. Professor (Paediatrics), SSMC & Mitford Hospital.

Case report :

A just born male baby, third issue of a consanguineous parents was delivered by caesarean section at term with breech presentation on third September, 2003 at Maternal and Child Health Training Centre (MCHTI), Azimpur. Antenatal history was uneventful. There was no history of the similar problem in the family. On routine examination, the birth weight of the baby was 3000 gm, length was 49 cm and occipitofrontal circumference (OFC) was 38 cm which fell on + 2SD. His face was dysmorphic characterized by prominent eyes with downward slanting and wide palpebral fissures, open posterior fontanelle, macrocephaly, low set ear, triangular mouth with downturned angles, long philtrum, glabeller haemangioma on the central part of the forehead, nevus flameus on the occiput and loose skin around the neck. (Figures 1 and 2). There was micrognathia. Cryptorchidism was evident on the left side (Fig.-3). Limb shortening was not obvious. On the basis of clinical features the baby was diagnosed as a case of Robinow syndrome.

Figure-1 : *Showing downward slanting of palpebral fissure.*

Figure-2 : *Showing glabellar haemangioma on the central part of the head.*

Figure-3 : *Showing left sided cryptorchidism.*

On investigation, his haemoglobin level was 12 gm/dl and random blood sugar 5.7 mmol/litre. Test for VDRL and HBsAg were negative. X-Ray limbs showed short tibias and femora (Fig.-4). Both rhizomelic and mesomelic shortening were found in the lower limbs. With above clinical and radiological findings the diagnosis was established as Robinow syndrome. His postnatal period was uneventful till the second week of life. In follow up visit at the age of three weeks, he was found to have umbilical granuloma and was treated accordingly.

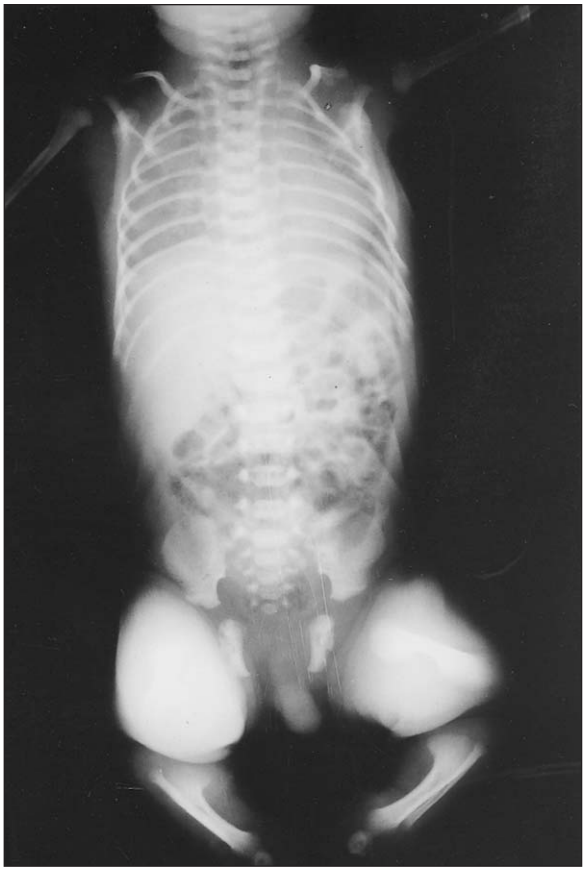

Figure-4 : *X-ray showing rhizomelic and mesomelic braclymelia of the lower limbs (short fibia and femora).*

Discussion:

Langer has divided bony defects of the extremities into four general types: acromelia or distal shortening, mesomelia or shortening of the mid segment, rhizomelia or proximal shortening and proportionate shortening of all segments of the limb [8]. Some of these disorders involve defective growth of both the tubular bones or the axial skeleton or both, and are recognizable at birth. The disorder first described by Robinow et al[1] is evident at birth. So far, over 100 cases have been reported. The gene for the autosomal recessive form was identified as the ROR2 gene on chromosome 9q22. The same gene, ROR2

has been shown to cause autosomal dominant brachydactyly.[9] Along with bony defects the peculiar facies and the genital abnormalities serve to differentiate this condition from other dwarfing syndromes. Affected individuals with Robinow syndrome tend to have short stature but their weight and length may be within normal range at birth[10]. Short stature may appear by three to four years of age[10]. The present case had normal length and weight at birth.

Mesomelic brachymelia was considered almost essential to diagnose Robinow syndrome. Mesomelic shortening of the forearms is one of the cardinal features of the condition but the pattern of limb shortening can be extremely variable. There may be rhizomelic brachymelia[11]. In this case, there was rhizomelic and mesomelic brachymelia. The upper limb deformities were found in the cases described by Wadia et a1[12]. In this case, there was lower limb deformities without any involvement of upper limbs.

The cardinal features of Robinow syndrome are slight to moderate shortness of stature at the postnatal onset, frontal bossing, hypertelorism, macrocephaly, shortening of forearm (mesomelic brachymelia), genital abnormality and nevus flammeus[13,14]. But shortening may be found in lower limbs. Rhizomelic brachymelia (shortening of proximal part of the lower limb) can be a part of this syndrome. These mesomelic and rhizomelic brachymelia may not be evident at birth clinically.

Achondroplasia may mimic this syndrome. Clinically, normal interorbital distance and trident feature in hands is seen in achondroplasia. Radiologically achondroplasia differs from Robinow syndrome by the presence of caudally decreasing lumber interpediculer distances and lordotic lumbosacral angle with shortened sciatic notch[1]. Once the Robinow syndrome is suspected clinically, radiological evaluation is to be carried out to establish the diagnosis.

References :

1. Robinow M. Silverman FN, Smith HD. A newly recognized dwarfing syndrome. Am J Dis Child 1969 ; 117: 645-51.
2. Olivieri C, Maraschio P, Caselli D, Martini C, Beluffi G, Maserati E, et al. Interstitial deletion of chromosome 9, int del (9) (9q22.31-q3 L2), including the genes causing multiple basal cell nevus syndrome and Robinow/ brachydactyly 1 syndrome . Eur J Pediatr 2003; 162: 100-3.
3. Shprintzen R.J, Goldberg RB, Saenger P, E Sidoti E. Male to male transmission of Robinow's syndrome Am J Dis Child 1982; 136: 594-97.
4. Vallee L, Van Nerom PY, Ferraz FG, Delecour M, Maroteaux P, Farriaux JP, et al. Robinow's syndrome with dominant transmission. Arch Fr Pediatr 1982; 39: 447-48.
5. Wadlington W, Tucker V, Schimke R. Mesomelic dwarfism with hemivertebrae and small genitalia (the Robinow syndrome). Am J Dis Child 1973; 126: 202-05.
6. Saal HM, Poole AE, Lodeiro J G, Weinbaum P J, Gremstein RM. Autosomal recessive Robinow syndrome: Evidence for genetic heterogeneity. Am J Hum Genet 1985; 37: 74.
7. Afzal AR, Rajab A, Fenske CD, Oldridge M, Elanko N, Ternes Pereira E, et al. Recessive Robinow syndrome, allelic to dominant brachydactyly type B, is caused by mutation of ROR2. Nat Genet 2000; 25: 419-22.
8. Langer LO Jr. Mesomelic dwarfism of the hypoplastic ulna, fibula, mandible type. Radiology 1967; 89: 654-60.
9. Patton MA, Afzal AR. Robinow syndrome. J Med Genet 2002; 39: 305-10.
10. Butler MG, Wadlington WB. Robinow syndrome: Report of two patients and review of the literature. Clin Genet 1987; 31: 77-85.
11. Bain MD, Winter RM , Burn J. Robinow syndrome without mesomelic brachymelia . A report of five cases. J Med Genet 1986; 23: 350-54.
12. Wadia RS, Shirole DB, Dikshit MS. Recessively inherited costovertebral segmentation defect with mesomelia and peculiar facies (Covesdem syndrome). A new genetic entity. J Med Genet 1978; 15: 123-27.
13. Lyons KJ, Smiths Recognizable Patterns of Human Malformation. Fifth edition. Philadelphia : WB. Saunders Co.1997.
14. Turken A, Balci S, Senocak ME, Hicsonmez A. A large inguinal hernia with undescended testis and micropenis in Robinow syndrome. Clin Dysmorphol 1996; 5: 175-8.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 22, No. 2, May 2005

Camurati- Engelmann Syndrome: A Case Report

MN ABSAR[a], AH SARKAR[b], MS RAHMAN[c]

Summary:

A case of Camurati- Engelmann's syndrome, a form of progressive diaphyseal dysplasia of unknown aetiology in a boy of 14 months of age is reported. The boy presented with delayed motor mile stones, weakness, anterior bowing of tibia, and firm and prominent musculature. Diagnosis was made on the basis of characterstic radiological findings. This is the first case reported so far in Bangladesh.

(J Bangladesh Coll Phys Surg 2005; 23 : 84-86)

Introduction:

Prograssive diaphyseal dysplasia or Camurati-Engelmann syndrome or Engelmann's disease (as designated in earlier literature) is a rare autosomal disease, primarily affecting long bones and musculature. Cockaine in 1920 for the first time reported the disease in a 9.5 year old boy who presented with sclerosis of long bones and base of the skull[1]. The disease historically known as Camurati-Engelmann's disease was re-designated as progressive diaphyseal dysplasia in 1948 to stress the involvement of diaphysis and progressive nature of the disorder[2]. In course of time it was revealed that membranous bones, and in advanced stage even vertebral columns are also affected[2,3]. There is also involvement of muscles, resulting in weakness, waddling gait and pain[3]. Camurati reported this disease as a case of hereditary symmetrical osteitis of the lower limbs in a seven year old boy in 1923. The characterstic roentgenographic description came from Engelmann in 1929. Hence the name Engelmann's disease or Camurati-Engelmann's disease[1]. About 100 cases are so far been reported in different literature[4].

a. Prof. M. Nurul Absar, MBBS, FCPS, MHPEd. Professor of Paediatrics, Rangpur Medical College Hospital, Rangpur, Bangladesh

b. Dr. Altaf Hossain Sarkar. MBBS, DMRD, MD. Asst. Professor of Radiology and Imaging, Rangpur Medical College Hospital, Rangpur. Bangladesh.

c. Dr. (Col).Md. Sayedur Rahman. MBBS, MCPS, FCPS. Classified specialist in Radiology and Imaging. Combined Military Hospital, Rangpur Cantonment, Rangpur, Bangladesh.

Address of correspondence : Prof. M. Nurul Absar. Professor of Paediatrics, Rangpur Medical College Hospital, Rangpur, Bangladesh, E- Mail: nsnafis@yahoo.com

Diversity of presentations was found in different literature according to the site and extent of involvement, and it has been suggested that the disease could be a systemic disease with predominant muscle and bone manifestations[4,5].

This case is reported with characteristic clinical and radiological presentation, only difference with others is that the child had increased muscle bulk.

Case report:

A 14 month old boy was brought to the hospital by his father with the complaints of anterior bowing of both legs with delayed mile stones. On inquiry, it was revealed that the child was lagging behind in motor abilities only. Other mile stones were normal. His birth history was uneventful. His nutrition seemed to be adequate. He was properly immunized. There was no history of other sibs or any relatives of the child being affected. The child had muscle weakness. He could stand up with difficulty.

He walked with waddling gait. He also had difficulty to keep on standing with a tendency to fall when attempted to walk. The father said that his son was clumsy in walking. This, he compared with motor development of other children. This is however the age by which a child can stand independently and walk steadily without fall. He did not have any pain in his limbs. His father's main concern was the anterior bowing of the legs. His muscles looked prominent and firm on feeling. His leg muscles were more prominent and pelvic girdle muscles were weaker than others.

Initial presentation aroused suspicion of myopathy. Other systemic examination revealed no abnormality. There was no neurological abnormality either.

His biochemical markers were normal (Table-I) and excluded the possibility of myopathy. X-ray of the legs (Fig.-1) showed diaphyseal sclerosis with sub-periosteal lamellar bone formation. Muscle biopsy could not be done. Bone biopsy also could not be done.

Table-I

Biochemical markers of the patient

CPK	30 U/L(Range: 5-195 U/L)
LDH	245 U/L(Range: 30-460 U/L)
Serum calcium	09 mg/dl(Range: 8.1- 10.4 mg/dl)
Serum phosphate	04.1 mg/dI(Range: 2.4-4.5 mg/d l)
Serum alkaline phosphatase	285 U/L(Range: 98-279 U/L)*

*Higher level may be considered normal for a growing child

So, it was diagnosed to be a case of Engelmann's disease. The child is in follow-up for one year. He is improving gradually in terms of muscle weakness and gait abnormality.

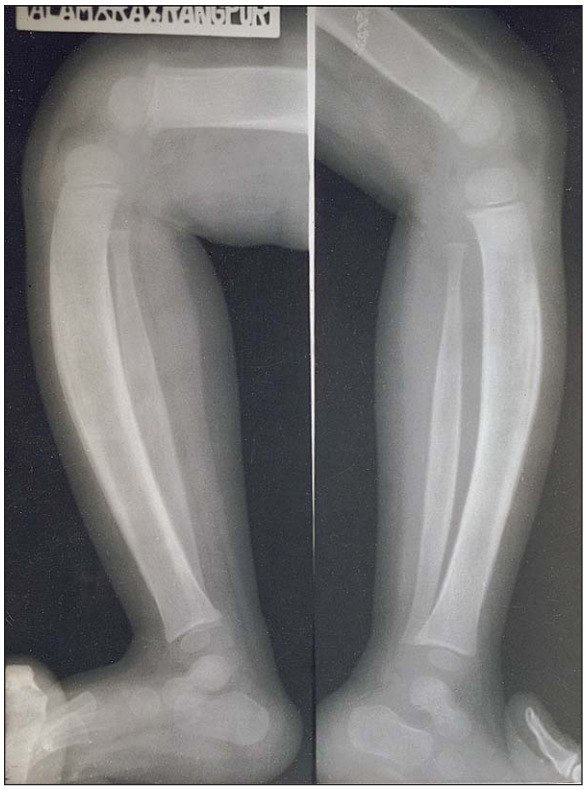

Figure-1 : *Radiological finding of tibia and fibula of the case*

Discussion:

Camurati-Engelmann syndrome is a combination of muscular dystrophy and dysplasia of bone. Often it has been described as progressive diaphyseal dysplasia of bone. But it has been reported by many authors that almost all bones of the body including long bones, axial bones and skull bones may be affected[1-8]. In this patient only the long bones of lower extremity were involved.

Since Camurati and Engelmann reported the syndrome, cases are being reported from different parts of the world with diverse clinical presentations with the key presentations like muscle weakness, gait disturbance (characteristically waddling or woobling gait) and sclerotic change in the bone remaining the constant features[1,4].

These were the features in this case also. In addition, the boy had anterior bowing of legs.

Applegate and colleagues reported cranial neuropathy due to stenosis of foramina of skull bones resulting in impaired hearing, difficulty in talking and chewing [4]. However, no neurological deficit was detected in this boy. About 100 cases have been reported up till now[4].

Engelmann's disease is yet to be studied as regards its cause and pathogenesis. Extensive literature review reveals that this rare disease presents with gait disturbance, muscular weakness, pain in the leg, thin and small muscle mass, anterior bowing of tibia, shiny skin over anterior surface of tibia, expression less face, tight skin of maxilla, enlarged jaw and occasional involvement of cranial nerves[1,4]. This patient presented with many of these features. Patients are biochemically normal, as was the reported case here. Bone biopsy shows thickening of diaphyseal cortices and endosteally and subperiosteally formed new bones. However, bone biopsy could not be done in this case.

Patients sometimes present with the features of muscular dystrophy, particularly of pelvic girdle type. Radiology of long bones confirms the diagnosis. Among the atypical features, delayed mile stone and systemic manifestation like fatigue, poor appetite, lamellated periosteal reaction, joint involvement with contracture and crippling pain are also noticed[5,7-10]. In this case all the features except joint involvement and pain were noticed.

There are reports of sporadic cases but familial propensity is marked. Sparks and Graham have suggested autosomal dominant mode of inheritance with variable expressivity of the gene[8]. No familial tendency was found in this case.

Treatment of Engelmann's disease is uncertain. Corticosteroid has been tried with limited success. Reports of alleviation of symptoms of pain and other manifestation to some degree has been reported but its mode of action could not be explained[9,10]. Surgical intervention in crippling disease like joint involvement and contracture has been suggested but not strongly recommended. Analgesics failed to show any response[8]. No treatment was given to this boy. But on follow up he has shown some noticeable improvement in gait and muscle strength.

The case reported here simulates with the manifestations of Engelmann's disease in many respect. The typical presentations, muscle weakness, gait disturbance and anterior bowing of the legs were clinically very obvious in this case. Absence of biochemical abnormality and radiological evidence of diaphyseal sclerotic change with subperiosteal lamellar bone formation suggests Engelmann's disease. Noticeable deviation here is improvement in gait and weakness of the patient. However, improvement has been rported in earlier literature also.

References:

1. Girdany BR, Sane S, Graham CB. Engelmann's Disease. Pediatr Radiol 1973; 4: 414-437.
2. Kaftory JK, Kleinhaus U, Naveh Y. Progressive diaphyseal dysplasia (Camurati-Engelmann): Radiographic follow-up and CT findings. Radiology 1987; 164: 777-782.
3. Naveh Y, Kaftory JK, Alon U,Ben-Devid, Berant M. Progressive diaphyseal dysplasia: Genetics and clinical and radiologic manifestations. Pediatrics 1984; 74: 399-405.
4. Applegate LJ, Applegate GR, Kemp SS. MR of multiple cranial neuropathies in a patient with Camurati-Engelman disease: Case report. AJNR 1991; 12: 557-559.
5. Mikity VG, Jacobson G. Progressive diaphyseal dysplasia (Engelmann's disease). J Bone Joint Surg 958; 40-A: 206-210.
6. Stronge F, McDowell HB. A case of Engelmann's disease. J Bone Joint Surg 1950; 32-B: 38-39.
7. Wolf BH, Ford HW. An unusual radiological manifestation of Engelmann's disease in a young negro child. Radiology 1971; 99: 401-402.
8. Sparkes RS, Graham B. Camurati- Engelmann disease, genetics and clinical manifestation with a review of literature. J Med Genetics 1972; 9: 73-85.
9. Verbruggen LA, Bossuyt A, Schreuer R, Somers G. Clinical and scientigraphic evaluation of corticosteroid treatment in a case of progressive diaphyseal dysplasia. J Rheumatol 1985; 12: 809-813.
10. Naveh Y, Alon U, Kaftori JK, Berant M. Progressive diaphyseal dysplasia: Evaluation of corticosteroid therapy. Pediatrics 1985; 75: 321-323.

COLLEGE NEWS

(J Bangladesh Coll Phys Surg 2005; 23 : 87-99)

EXAMINATION NEWS :

Result of FCPS Part -I, FCPS Part -II and MCPS Examinations held in January, 2005 are given below :

4817 candidates appeared in FCPS Part - I Examination held in January, 2005, of which 986 candidates came out successful. Subject-wise results are as follows :

FCPS Part-I Examiantion:

	Number of candidates appeared	Passed
Medicine	1530	288
Surgery	844	181
Paediatrics	572	102
Obst. & Gynae	1019	294
Ophthalmology	124	31
Otolaryngology	123	07
Psychiatry	38	02
Anaesthesiology	68	07
Radiology	100	10
Radiotherapy	17	02
Dermatology & Venereology	121	14
Physical Medicine	29	09
Dental Surgery	126	17
Family Medicine	18	02
Haematology	40	12
Biochemistry	06	02
Microbiology	18	02
Histopathology	24	04
	4817	986

456 candidates appeared in FCPS Part-II Examination in different subjects. List of candidates who satisfied the board of examiners is as follows : -

Roll No.	Name of candidate	Graduated from	Speciality
019	Dr. Mohammad Masud Hasan	Dhaka Medical College, Dhaka.	Medicine
021	Dr. Muhammad Hezbullah	Sylhet MAG Osmani M.C.	Medicine
033	Dr. Panchanan Das	Chittagong Medical College, Chittagong.	Medicine
043	Dr. Shiuly Majumdar	Sylhet MAG Osmani Medical C.	Medicine
061	Dr. Mohammad Main Uddin	Khulna Medical College, Khulna.	Medicine
069	Dr. Mahjuba Umme Salam	Dhaka Medical College, Dhaka.	Medicine
116	Dr. Chanchal Kumar Ghosh	Sir Salimullah Medical College, Dhaka.	Medicine
118	Dr. Raihan Rabbani	Dhaka Medical College, Dhaka.	Medicine

Roll No.	Name of candidate	Graduated from	Speciality
124	Dr. Md. Abdur Rakib	Dhaka Medical College, Dhaka.	Surgery
127	Dr. Md. Mehedi Hasan	Sir Salimullah Medical College, Dhaka.	Surgery
133	Dr. Md. Habibullah Sarkar	Rajshahi Medical College, Rajshahi.	Surgery
136	Dr. Md. Mosharraf Hossain	Dhaka Medical College, Dhaka.	Surgery
140	Dr. Forhad Hossain Chowdhury	Sir Salimullah Medical College, Dhaka.	Surgery
145	Dr. Hriday Ranjan Roy	Rajshahi Medical College, Rajshahi.	Surgery
166	Dr. Shakil Farid	Sir Salimullah Medical College, Dhaka.	Surgery
170	Dr. Maruf Alam Chowdhury	USTC, Chittagong.	Surgery
184	Dr. Tapesh Kumar Paul	Dhaka Medical College,Dhaka.	Surgery
191	Dr. Bidhan Chandra Das	Dhaka Medical College, Dhaka.	Surgery
203	Dr. Md. Moniruzzaman Sarker	Rajshahi Medical College, Rajshahi.	Surgery
212	Dr. Muhd. Mustaque Husain	Dhaka Medical College, Dhaka.	Surgery
213	Dr. Mohammad Abul Hasnat	Sir Salimullah Medical College, Dhaka.	Surgery
220	Dr. Md. Surman Ali	Rajshahi Medical College, Rajshahi.	Surgery
222	Dr. Md. Shofiur Rahman	Sir Salimullah Medical College, Dhaka.	Surgery
224	Dr. Mohammad Yasin Chowdhury	MAG Osmani Medical College, Sylhet	Surgery
232	Dr. Md. Al-Amin Mridha	Rajshahi Medical College, Rajshahi.	Paediatrics
247	Dr. Md. Zakirul Islam	Sir Salimullah Medical College, Dhaka.	Paediatrics
255	Dr. Shakil Ahmed	Chittagong Medical College, Chittagong.	Paediatrics
259	Dr. Najnin Umme Zakia	Chittagong Medical College, Chittagong.	Paediatrics
266	Dr. Mohammad Monir Hossain	MAG Osmani Medical College, Sylhet	Paediatrics
269	Dr. Didarul Alam	Chittagong Medical College, Chittagong.	Paediatrics
271	Dr. Md. Abdur Rouf	Chittagong Medical College, Chittagong.	Paediatrics
272	Dr. Md. Anwar Hossain	Mymensingh Medical College, Mymensingh.	Paediatrics
283	Dr. Sheikh Muhammad Shaheedul Islam	Dhaka Medical College, Dhaka.	Paediatrics
288	Dr. Suraiya Begum	Dhaka Medical College, Dhaka.	Paediatrics
292	Dr. Selina Akter	JIMC, Bajitpur.	Obstetrics & Gynaecology
293	Dr. Nazma Haque	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
294	Dr. Sharmila Barua	Chittagong Medical College, Chittagong.	Obstetrics & Gynaecology
295	Dr. Most. Dalia Akhter	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
296	Dr. Ferdousi Chowdhury	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
298	Dr. Shamsoon Nahar	Dhaka Medical College, Dhaka.	Obstetrics & Gynaecology
300	Dr. Monira Ahmed	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
301	Dr. Muna Shalima Jahan	Sir Salimullah Medical College, Dhaka.	Obstetrics & Gynaecology
302	Dr. Tanvin Khanam	Rajshahi Medical College,Rajshahi.	Obstetrics & Gynaecology

Roll No.	Name of candidate	Graduated from	Speciality
303	Dr. Mst. Hosna Ara Khatun	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
306	Dr. Samar Kumar Ghosh	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
307	Dr. Mahmuda Khatun	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
309	Dr. Mahbuba Akhter Banu	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
312	Dr. Jamila Khatun	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
314	Dr. Parul Akhter	MAG Osmani Medical College, Sylhet	Obstetrics & Gynaecology
315	Dr. Nasrin Akhter	Chittagong Medical College, Chittagong.	Obstetrics & Gynaecology
316	Dr. Dil Afroza Akhtar	Rajshahi Medical College, Rangpur.	Obstetrics & Gynaecology
317	Dr. Kamrun Sattar	Chittagong Medical College, Chittagong.	Obstetrics & Gynaecology
320	Dr. Pervin Akther	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
321	Dr. Tayeeba Tanjin Mirza	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
322	Dr. Samina Begum	Dhaka Medical College, Dhaka.	Obstetrics & Gynaecology
323	Dr. Kaoser Jahan	Sir Salimullah Medical College, Dhaka.	Obstetrics & Gynaecology
324	Dr. Joynab Akhter	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
327	Dr. Yisrat Jahan	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
328	Dr. Jahanara	Sir Salimullah Medical College, Dhaka.	Obstetrics & Gynaecology
333	Dr. Syeda Sayeeda	Sir Salimullah Medical College, Dhaka.	Obstetrics & Gynaecology
334	Dr. Mossammat Dilruba Akter	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
338	Dr. Khondker Asaduzzaman	Chittagong Medical College, Chittagong.	Obstetrics & Gynaecology
339	Dr. Nasreen Sultana	Dhaka Medical College, Dhaka.	Obstetrics & Gynaecology
340	Dr. Md. Zafirul Hassan	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
345	Dr. Kamrun Nahar	USSR	
346	Dr. Nasima Begum	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
347	Dr. Shireen Akhter Khanam	Chittagong Medical College, Chittagong.	Obstetrics & Gynaecology
348	Dr. Zahirun Nessa	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
351	Dr. Shahanaz Ahmed	Sir Salimullah Medical College, Dhaka.	Obstetrics & Gynaecology
353	Dr. Sultana Razia	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
354	Dr. Dilruba Akter	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
356	Dr. Mahfuza Rahman	Mymensingh Medical College, Mymensingh.	Obstetrics & Gynaecology
357	Dr. Rowshan Ara	Rajshahi Medical College, Rajshahi.	Obstetrics & Gynaecology
364	Dr. Nadira Begum	MAG Osmani Medical College, Sylhet	Obstetrics & Gynaecology
365	Dr. Shahida Begum	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
366	Dr. Beauty Rani Roy	MAG Osmani Medical College, Sylhet	Obstetrics & Gynaecology
368	Dr. Kazi Shamsun Nahar	Khulna Medical College	Obstetrics & Gynaecology

Roll No.	Name of candidate	Graduated from	Speciality
370	Dr. Suha Jesmin	Dhaka Medical College, Dhaka.	Obstetrics & Gynaecology
371	Dr. Sultana Jebunnaher	Sher-e-Bangla Medical College, Barisal.	Obstetrics & Gynaecology
373	Dr. Niloufar Shameem Afza	Dhaka Medical College, Dhaka.	Obstetrics & Gynaecology
374	Dr. Razia Sultana	IPGMR, Dhaka.	Obstetrics & Gynaecology
375	Dr. Rokeya Khan	Sir Salimullah Medical College, Dhaka.	Obstetrics & Gynaecology
376	Dr. Nazneen Begum	Dhaka Medical College, Dhaka.	Obstetrics & Gynaecology
399	Dr. Md. Abdur Rakib	Sir Salimullah Medical College, Dhaka.	Ophthalmology
400	Dr. Md. Alauddin	Mymensingh Medical College, Mymensingh.	Ophthalmology
401	Dr. Md. Hafizur Rahman	Khulna Medical College	Ophthalmology
404	Dr. S.M. Masud Parvez	MAG Osmani Medical College, Sylhet	Ophthalmology
405	Dr. Nimai Chandra Karmakar	Rajshahi Medical College, Rajshahi.	Ophthalmology
410	Dr. A.H.M. Noor-E-As Sayeed	Sir Salimullah Medical College, Dhaka.	Otolaryngology
418	Dr. A.S.M. Tazul Islam	Sher-e-Bangla Medical College, Barisal.	Otolaryngology
419	Dr. Md. Kamruzzaman	IPGMR, Dhaka.	Otolaryngology
420	Dr. A.F.M. Ekramuddaula	Sir Salimullah Medical College, Dhaka.	Otolaryngology
424	Dr. Kamol Krishna Pramanik	Sir Salimullah Medical College, Dhaka.	Otolaryngology
426	Dr. Ranjit Kumar Mistry	Sher-e-Bangla Medical College, Barisal.	Otolaryngology
407	Dr. Mohammad Abdul Wahab	Sher-e-Bangla Medical College, Barisal.	Psychiatry
377	Dr. Md. Abdul Aleem	Mymensingh Medical College, Mymensingh.	Anaesthesiology
378	Dr. Mir Mahmud Hossain	Chittagong Medical College, Chittagong.	Anaesthesiology
379	Dr. Md. Rezaul Hoque Pk.	Rajshahi Medical College, Rajshahi.	Anaesthesiology
381	Dr. Hasina Akhter	MAG Osmani Medical College, Sylhet	Anaesthesiology
385	Dr. Paresh Chandra Sarker	Rajshahi Medical College, Rajshahi.	Anaesthesiology
433	Dr. Taslima Rahman	Sir Salimullah Medical College, Dhaka.	Radiology
434	Dr. Md. Khairul Islam	Sir Salimullah Medical College, Dhaka.	Radiology
436	Dr. Syed Zoherul Alam	Sylhet MAG Osmani M.College	Radiology
438	Dr. Zeenat Meraj	Dhaka Medical College, Dhaka.	Dermatology & Venereology
442	Dr. Shamima Akhter	MAG Osmani Medical College, Sylhet	Dermatology & Venereology
443	Dr. Mohammod Abu Hena Chowdhury	Dhaka Medical College, Dhaka.	Dermatology & Venereology
445	Dr. Jebun Nessa	Dhaka Medical College, Dhaka.	Physical Medicine & Rehabilitation
446	Dr. Md. Mahmudur Rahman	MAG Osmani Medical College, Sylhet	Physical Medicine & Rehabilitation
447	Dr. Md. Abdul Mazed	MAG Osmani Medical College, Sylhet	Physical Medicine & Rehabilitation
451	Dr. Md. Abdul Aziz	Rajshahi Medical College, Rajshahil.	Haematology
452	Dr. Abu Jafar Mohammed Saleh	Sher-e-Bangla Medical College, Barisal.	Haematology
454	Dr. S.M. Anwar Sadat	Chittagong Medical College, Chittagong.	Oral & Maxillofacial Surgery
456	Dr. Mohammed Jahangir Hossain	Chittagong Medical College, Chittagong.	Urology

213 candidates appeared in MCPS Examinations in different subjects. List of candidates who satisfied the board of examiners is as follows :

Roll No.	Name of the candidate	Speciality
013	Dr. Syed Alamgir Safwath Rana	Medicine
015	Dr. Ahmed Mohammed Ali	Medicine
047	Dr. Shakera Ahmed	Surgery
057	Dr.Sheik Md. Abdullah	Surgery
063	Dr. Md.Merajul Islam	Surgery
065	Dr. Muhammad baqi Billah	Surgery
071	Dr. Lakshman Chandra Kundu	Paediatrics
092	Dr. Md. Arshed Ali	Obstetrics & Gynaecology
099	Dr. Parveen Akhtar	Obstetrics & Gynaecology
107	Dr. Shahela nazneen	Obstetrics & Gynaecology
109	Dr. Rifat Sultana	Obstetrics & Gynaecology
121	Dr. Seema Chowdhury	Obstetrics & Gynaecology
126	Dr. Mahbuba Nargis	Obstetrics & Gynaecology
136	Dr. Momena Khatun	Obstetrics & Gynaecology
138	Dr. Nahida Naznin	Obstetrics & Gynaecology
141	Dr. San Yaat Rabbi	Ophthalmology
142	Dr. Md. Fakhrul Islam	Ophthalmology
143	Dr. Md. Sultanul Haq Aftabi	Ophthalmology
145	Dr. A.K.M. Nizam Uddin	Ophthalmology
148	Dr. A.N.M. Nurul Huda	Otolaryngology
165	Dr. Sharifur Rashid	Psychiatry
168	Dr. Debashis Chowdhury	Anaesthesiology
175	Dr. Mainul Hasan Khan	Radiology
176	Dr. Mohammad Mamun-ur-Rashid	Radiology
188	Dr. Md. Asadozzaman	Dermatology & Venereology
189	Dr. Md. Nazrul Islam	Dermatology & Venereology
197	Dr. Md. Kamrul Hassan Sardar	Forensic Medicine
198	Dr. Md. Abdus Shahid	Forensic Medicine
206	Dr. Arif Ahmed	Clinical Pathology
210	Dr. Md. Nasim Iqbal	Clinical Pathology
211	Dr. Rehena Akhter	Clinical Pathology

Election of the Councillors and Executive Committee:
The Election of 8 Councillors of the College was held on 25-02-2005.

List of Councillors:
Following is the 20 member list of the Councillors:

Existing Councillors:
(for the session 2003-2007)

1.. Prof. Syed Atiqul Haq
2. Prof. Abu Zafar Md. Zahid Hossain
3. Prof. Quazi Deen Mohammad
4. Prof. A.H.M. Towhidul Anowar Chowdhury
5. Prof. S.A.M. Golam Kibria
6. Prof. Mahmud Hasan
7. Prof. Md. Ruhul Amin
8. Prof. Abdul Bayes Bhuiyan

Newly elected Councillors:
(for the session 2005 - 2009)

9. Prof. T.I.M. Abdullah-Al-Faruque
10. Prof. Md.Abul Kashem Khandaker
11. Prof. Md. Abdul Mobin Khan
12. Prof. Nazmun Nahar
13. Prof. Md. Sanawar Hossain
14. Prof. Md. Abdul Hadi
15. Prof. Md. Saiful Islam
16. Prof. Abdul Kader Khan

New Councillors Nominated by Government of People's Republic of Bangladesh:

17. Prof. M. A. Majed
18. Prof. Tofayel Ahmed
19. Prof. A.H.M. Ahsanullah
20. Prof. A.N.M. Atai Rabbi

Executive Committee:
The Election Commission of the Bangladesh College of Physicians and Surgeons has declared the members of new Executive Committee for 2 years from March, 2005 as follows :

President:	-	Prof. Md. Abdul Hadi
Senior Vice-President:	-	Prof. Md. Abdul Mobin Khan
Vice-President:	-	Prof. Quazi Deen Mohammad
Treasurer:	-	Prof. T.I.M. Abdullah-Al-Faruque
Members:	-	Prof. A.H.M. Towhidul Anowar Chowdhury
	-	Prof. Md. Abul Kashem Khandaker

Appointment of the Secretary and the Controller of Examinations:
The Council has appointed Prof. Abu Zafar Md. Zahid Hossain as the Honorary Secretary of the College.
The Council has appointed Prof. Mohammad Saiful Islam as the Controller of Examinations of the College.

Various Committees and Faculties of the Bangladesh College of Physicians and Surgeons:
The Council of Bangladesh College of Physicians and Surgeons has formed the following Committees and Faculties of the College for 2 years with effect from March, 2005.

Examination Committee:

Professor Mobin Khan	Chairperson
Professor T. A. Chowdhury	Member
Professor M. A. Majid	"
Professor A. B. Bhuiyan	"
Professor Md. Tahir	"
Professor M. A. Mannan Miah	"
Professor Syed Maruf Ali	"
Professor Syed Mukarram Ali	"
Professor Md. Abdullah	"
Professor Mohammad Saiful Islam	(ex-officio)

Reference Committee:

Professor Md. Abdul Hadi	Chairperson
Professor A.H.M. Ahsanullah	Member
Professor Tofayel Ahmed	"
Prof. T.I.M. Abdullah-Al-Faruq	"
Prof. Abdul Kader Khan	"
Prof. Mohammad Saiful Islam	"
Prof. Md. Omar Ali	"
Prof. Sayeba Akhter	"

Finance and Tender Committee:

Professor Quazi Deen Mohammad	Chairperson
Professor A. H. M. Ahsanullah	Member
Professor Syed Atiqul Haq	"
Professor T.I.M. Abdullah-Al-Faruq	"
Professor Tofayel Ahmed	"
Professor Nazmun Nahar	"

Prof. M.A. Majid	"
Major Gen. (Retd.) Prof. Ziauddin Ahmed	"
Professor K.M.H.S. Sirajul Haque	"
Prof. Humayun Kabir Chowdhury	"
Professor A.N.M. Zia-ur-Rahman	"
Dr. (Maj. Gen.) Md. Ali Akbar	Member
Professor Mohammad Hanif	"
Dr. Parveen Shahida Akhter	"
Prof. Md. Abdul Quadir	"
Brig. Gen. (Retd.) Razia Khanam	"
Professor Salim Md. Jahangir	Member Secretary

Disciplinary Committee:

Professor M. A. Matin, M.P.	Chairperson
Professor A.H.M. Ahsanullah	Member
Professor T.A. Chowdhury	"
Professor Md. Abul Quasem	"
Professor Md. Tahir	"
Professor A. K. M. Mahbubur Rahman	"

Museum Committee:

Professor Md. Sanawar Hossain	Chairperson
Professor S. A. M. Golam Kibria	Member
Major Gen. (Retd.) A.S.M. Matiur Rahman	"
Professor Syed Mukarram Ali	"
Professor A. K. M. Anowarul Azim	"
Professor Abdush Shakur	"
Professor Abu Ahmed Ashraf Ali	"
Professor Anowara Begum	"
Professor Shafiqul Haque	"
Professor Kohinoor Begum	"
Professor Md. Khademul Islam	"
Professor Md. Mazibar Rahman	"
Dr. Ahmed Sayeed	"
Professor Brig. Gen. Nazrul Islam	"
Professor Kh. Manzare Shamim	"
Dr. A.M.S.M. Sharfuzzaman	"
Professor Maliha Rashid	"
Dr. Kamal Ibrahim	"
Dr. Feroze Quader	"
Dr. Farhat Hossain	"
Dr. Ferdousi Sultana	"
Dr. Farida Yesmin	"
Dr. Md. Shahab Uddin Talukder	"
Dr. Hossain Imam Al Hadi	"
Professor Syed Mahbubul Alam	Member Secretary

Library Committee:

Professor A.H.M. Ahsanullah	Chairperson
Prof. Mohammad Saiful Islam	Member
Professor Md. Ruhul Amin	"
Professor Abdush Shakur	"
Professor A. K. M. Anwarullah	"
Professor Syed Serajul Karim	"
Professor Md. Shahid Hossain	"
(Now out of Country)	
Dr. (Brig. Gen.) Anjan Kumar Deb	"
Dr. (Brig. Gen.) Md. Rabiul Hossain	"
Professor Brig. Gen. Md. Golam Rabbani	"
Dr. Firoz Ahmed Quraishi	"
Dr. A.K. Mustaque	"
Professor Maliha Rashid	"
Dr. Nooruddin Ahmed	"
Dr. Feroze Quader	"
Dr. Muhammad Ali	"
Dr. S. M. Amjad Hossain	"
Dr. A. B. M. Yunus	"
Dr. Md. Shahadot Hossain Sheikh	"
Dr. Md. Roushon Ali	"
Dr. Mahfuza Shirin	"
Dr. Md. Abdul Kader	"
Dr. Shah Habibur Rahman	"
Dr. Md. Rajibul Alam	Member Secretary

Journal Committee:

Professor M. A. Majed	Chairperson
Professor Md. Abul Faiz	Editor-in-Chief
Prof. T.I.M. Abdullah-Al-Faruque	Member
Professor Mahmud Hasan	"
Professor M. A. Majid	"
Prof. Md. Harun-Ur-Rashid	"
Prof. K.M.H.S. Sirajul Haque	"
Professor Md. Salehuddin	"
Professor M. A. Salam	"
Professor Syed Kamaluddin Ahmed	"
Dr. Projesh Kumar Roy	"
Professor A.K.M. Khorshed Alam	"
Professor Shafquat Hussain Khundker	"
Professor Ameena Majid	"
Professor Choudhury Ali Kawser	"
Dr. Emran Bin Yunus	"
Professor U.H. Shahera Khatun	"
Professor Mohammed Abu Azhar	"
Professor A.K.M. Fazlul Haque	"

Dr. Md. Rajibul Alam	"
Dr. Syed Azizul Haque	"
Dr. Nooruddin Ahmed	"
Professor Abid Hossain Mollah	"
Dr. Md. Mazibur Rahman Bhuiyan	"
Dr. Dewan Saifuddin Ahmed	"
Dr. Abdul Wadud Chowdhury	"
Dr. Md. Azharul Islam	"
Dr. Mohammad Monir Hossain	"
Dr. A.K.M. Aminul Hoque	"
Dr. Hasina Afroz	"
Dr. Md. Mujibur Rahman Howlader	"

Students' Advisory Committee:

Professor M. A. Majid	Chairperson
Professor Md. Arshad Ali	Member
Professor Md. Taiabur Rahman	"
Professor Sultana Jahan	"
Professor Md. Salehuddin	"
Dr. Ghulam Mahmood	"
Professor Ameena Majid	"
Professor Md. Mustafizur Rahman	"
Dr. Md. Hazrat Ali	"
Professor A.H.M. Shamsul Alam	"
Dr. (Col.) Harunur Rashid	"
Professor Syeda Afroza	"
Dr. (Lt. Col.) Md. Amzad Hossain Fakir	"
Dr. A.M.S.M. Sharfuzzaman	"
Dr. Md. Jannatul Islam (Surgery)	"
Professor Md. Anisur Rahman	"
Dr. Radheshyam Saha	"
Dr. Md. Sayedul Hoque	"
Dr. Md. Zahid Hassan Bhuiyan	"
Dr. Md. Sazzad Khondokar	"
Dr. Md. Shah Alam	"
Dr. (Lt. Col.) Muhammad Saiful Islam (Anaes.)	"
Dr. Md. Abid Kamal	"
Dr. Md. Rais Uddin Mondal	"
Prof. Fakhruddin Mohammad Siddiqui	Member Secretary

Continuing Professional Development(CPD) Committee:

Professor M. A. Mannan Miah	Chairperson
Professor Md. Harun-ur-Rashid	Member
Professor Ferdous Ara J. Janan	"
Professor Md. Zahangir Kabir	"
Professor Sadiqa Tahera Khanam	"
Professor Sayeba Akhter	"
Professor Hosne Ara Begum	"
Professor Ameena Majid	"
Professor Md. Afzal Hossain	"
Dr. Ahmed Sayeed	"
Dr. Md. Shahidul Alam Khan	"
Dr. Manzurul Alam	"
Professor Md. Golam Rabbani	"
Dr. Azizul Kahhar	"
Professor Syeda Afroza	"
Professor Khabir Uddin Ahmed	"
Dr. Md. Abdul Hayee	"
Dr. Anwarul Haider	"
Dr. Nezamuddin Ahmed (Anaes)	"
Dr. Luthful Aziz	"
Dr. Muhammad. Quamruzzaman	"
Dr. Mohammad Monir Hossain	"
Dr. Badrunnesa Begum	"
Dr. Mollah Md. Abu Sayed	"
Dr. Md. Atiar Rahman	"
Dr. Shahin Akhter Zahan Habib	"
Dr. Mohammad Monir-Uz-Zaman	"
Dr. Md. Saifullah	"
Dr. Feroze Quader	Member Secretary

Fellows' Welfare Committee:

Professor Md. Fazlul Haque	Chairperson
Dr. (Brig. Gen. Retd.) Muhammad Jahangir Hossain	Member
Professor Rowshan Ara Begum	"
Professor Md. Saaidur Rahman	"
Dr. Moudud Hossain Alamgir	"
Professor Israil Biswas	"
Dr. S. M. Zafar Ullah	"
Dr. Md. Omar Faruque Yusuf	"
Dr. Ahsanul Habib	"
Dr. Md. Lutfor Rahman	"
Dr. Md. Rafiqul Islam	"
Professor Syed Mahbubul Alam	"
Dr. A. K. M. Daud	"
Dr. Md. Abdul Quayum	"
Dr. Md. Shahinur Rahman	"
Dr. Md. Ibrahim Siddique	"
Dr. Abdul Wadud Chowdhury	"
Dr. Rafiques Saleheen	"
Dr. Md. Abdul Mannan Khan	"
Dr. Mohammad Mosharaf Hossain	"
Dr. Abul Basher Mohammed Muksudul Alam	"

Dr. Md. Raziul Haque	"
Dr. Masuda Begum	"
Dr. Md. Ashraf Uddin	"
Dr. Md. Sana Ullah	"
Dr. Md Saifullah	"
Dr. Md. Rajibul Alam	Member Secretary

Planning & Development Committee:

Professor M. A. Hadi	Chairperson
Professor Mobin Khan	Member
Professor S. A. M.Golam Kibria	"
Professor T.I.M. Abdullah-Al-Faruque	"
Professor A.H.M. Ahsanullah	"
Professor Abdul Bayes Bhuiyan	"
Professor Tofayel Ahmed	"
Professor Md. Nurul Amin	"
Professor Sultana Jahan	"
Professor Abu Zafar Md. Zahid Hossain	"
Professor Mohammad Saiful Islam	Member Secretary

Faculty of Anaesthesiology:

Professor Md. Khalilur Rahman	Chairperson
Professor S. N. Samad Choudhury	Member
Professor K. M. Iqbal	"
Professor A.K.M. Shafiqur Rahman	"
Professor Salim Md. Jahangir	"
Professor U. H. Shahera Khatun	"
Dr. Manzurul Alam	"
Professor Abu Yousuf Fazle Elahi Chowdhury	"
Dr. Kamal Ibrahim	"
Professor A.K.M. Shamsul Alam	"
Professor Wahiduddin Mahmood	"
Dr. (Brig. Gen. Retd.) Razia Khanam	"
Professor Mohammad Manirul Islam	"
Dr. Muhammad Ali	"
Dr. Md. Azharul Islam	"
Dr. Nezam Uddin Ahmed	"
Dr. Zerzina Rahman	"
Dr. Lutful Aziz	"
Dr. Abdul Khaleque Beg	Member Secretary

Faculty of Medicine including Dermatology & Venereology:

Professor Md. Tahir	Chairperson
Professor Mobin Khan	Member
Professor Quazi Deen Mohammad	"
Professor Md. Abul Kashem Khandaker	"
Professor Syed Atiqul Haq	"
Professor Mahmud Hasan	"
Professor Tofayel Ahmed	"
National Professor Nurul Islam	"
Professor Md. Nurun Nabi	"
Professor M. N. Alam	"
Professor A.Z.M. Maidul Islam	"
Professor A. K. Azad Khan	"
Professor Harun-ur-Rashid	"
Professor K.M.H.S. Sirajul Haque	"
Professor Firdous Ara J. Janan	"
Professor Md. Jalaluddin	"
Professor Md. Fazlul Haque	"
Professor Abduz Zaher	"
Professor Md. Zahangir Kabir	"
Professor Naseem Akhter Chowdhury	"
Professor A.K.M. Rafique Uddin	"
Professor Kaniz Moula	"
Professor Hasina Banoo	"
Professor Md. Nazrul Islam	"
Professor A.Q.M. Mohsen	"
Professor Md. Abul Faiz	"
Professor Faridul Islam	"
Professor Ghulam Mahmood	"
Dr. (Maj.Gen.) Md. Abdul Moyeed Siddiqui	"
Professor Chandanendu Bhushan Sarker	"
Dr. (Brig.Gen.) Md. Golam Rabbani	"
Dr Emran Bin Yunus	"
Professor Mohammed Abu Azhar	"
Dr. Md. Ridwanur Rahman	"
Professor Md. Rajibul Alam	"
Dr. Syed Azizul Haque	"
Dr. Md. Ali Hussain	"
Dr. (Lt. Col.) Md. Abdul Wahab	"
Professor Fakhruddin Mohammad Siddiqui	Member Secretary

Faculty of Radiology & Radiotherapy:

Professor A.S.Q.M. Sadeque	Chairperson
Professor M. N. Huda	Member
Professor (Retd.) A. Rab Bhuiyan	"
Professor Fazle Elahi	"
Professor Syed Mizanur Rahman	"
Dr. (Brig. Gen. Retd.) Chowdhury Abdul Gaffar	"
Professor A.M.M. Shariful Alam	"
Professor Muhammad Mahbubur Rahman	"
Dr. Parveen Shahida Akhter	"
Dr. (Col.) Zuberul Islam Chowdhury	"

Dr. Md. Mokles Uddin	"
Dr. (Brig. Gen.) Jahangir Alam	"
Dr. Md. Salahuddin Al-Azad	"
Dr. Md. Dayem Uddin	"
Dr. Syed Md. Akram Hussain	Member Secretary

Faculty of Haematology:

Professor M. A. Rashid	Chairperson
Professor Jalilur Rahman	Member
Dr. A.B.M. Yunus	Member
Dr. Md. Mahbubur Rahman	"
Dr. (Major) Susane Giti	"
Dr. Anupam Barua	"
Dr. Salma Afrose	"
Dr. (Col.) Abdul Hai	"
Dr. Masuda Begum	"
Dr. (Lt. Col.) Faruk Ahmed	"
Dr. Mainuddin Ahmed	"
Dr. Mohammad Golam Rabbani	"
Dr. Md. Moniruzzaman	"
Dr. (Lt. Col.) A.K. Md. Mustafa Abedin	"
Dr. Alamgir Kabir	"
Professor Mohiuddin Ahmed Khan	"
Dr. Shahed Ahmad Chowdhury	"
Dr. Md. Mohibur Rahman	"
Dr. Muhammad Mehedi Hasan	"
Dr. Rukhsana Khanam	"
Dr. (Major) A.K.M. Abu Yousuf	"
Dr. Jissan Wajed	"
Dr. (Major) Suraiya Akhtar	"
Dr. Md. Mizanur Rahman	"
Dr. Niru Nazmun Nahar	"
Dr. Mohammad Humayun	"
Dr. Mohammed Mosleh Uddin	"
Dr. (Col.) Zahid Mahmud	Member Secretary

Faculty of Surgery including Dentistry:

Professor A.N.M. Atai Rabbi	Chairperson
Professor M. A. Hadi	Member
Professor T.I.M. Abdullah-Al-Faruque	"
Professor S.A.M. Golam Kibria	"
Professor Md. Sanawar Hossain	"
Professor Abdul Kader Khan	"
Professor M. A. Majid	"
Professor Ashraf Hossain	"
Professor Shamsuddin Ahmed	"
Professor Md. Nabi Alam Khan	"
Professor M. A. Awal	"
Professor Abdush Shakur	"
Professor Rashid-E-Mahbub	"
Professor Md. Shelim Bhuiyan	"
Professor M. Alimuzzaman	"
Professor Abu Ahmed Ashraf Ali	"
Professor A.K.M. Shariful Islam	"
Professor Md. Khalilur Rahman	"
Professor Humayun Kabir Chowdhury	"
Professor A.K.M. Mahbubur Rahman	"
Professor Shafiqul Haque	"
Professor Abdus Sobhan Pramanik	"
Professor Syed Serajul Karim	"
Professor Hasan Md. Abdur Rouf	"
Professor Md. Margub Hossain	"
Professor A.N.M. Zia-ur-Rahman	"
Professor Meer Mahbubul Alam	"
Professor Shafquat Hussain Khundkar	"
Professor Md. Shahjahan Ali	"
Professor Md. Khademul Islam	"
Dr. Md. Mahbub-ul-Alam	"
Dr. Md. Wahiduzzaman	"
Professor Abdul Haque	"
Dr. (Brig. Gen.) A.K.M. Jafarullah Siddiq	"
Professor Md. Shahid Hossain	"
Professor Md. Afzal Hossain	"
Maj. Gen. (Dr.) Md. Ali Akbar	"
Professor Md. Mazibar Rahman	"
Professor Israil Biswas	"
Professor Zahidul Haq	"
Professor Shahid Karim	"
Professor Syed Mahbubul Alam	"
Dr. Md. Mujibur Rahman Howlader	"
Professor Md. Omar Ali	Member Secretary

Faculty of Physical Medicine & Rehabilitation:

Professor Md. Quamrul Islam	Chairperson
Professor Shamsuddin Ahmed	Member
Professor Birendra Nath Bhattacharjee	Member
Professor Aminuddin Ahmed Khan	"
Dr. Md. Taslim Uddin	"
Dr. Mohammad Abdur Rashid	"
Dr. Shamsun Nahar	"
Dr. Sohely Rahman	"

Professor Md. Habibur Rahman	"
Dr. Md. Hilalul Islam	"
Dr. Md. Mahfuzur Rahman	"
Dr. Md. Abdus Shakoor	"
Professor Md. Moyeenuzzaman	Member Secretary

Faculty of Otolaryngology:

Professor Md. Alauddin	Chairperson
Professor M. A. Majed	Member
Professor Md. Nurul Amin	"
Professor Md. Abdullah Haroon	"
Dr. (Brig. Retd.) Syed Ahsan Karim	"
Professor Nilakanta Bhattacharjee	"
Professor Md. Abdullah	"
Professor Md. Abul Hasnat Joarder	"
Professor Md. Abdul Quadir	"
Dr. Md. Kamrul Hassan Tarafder	"
Dr. Mohammad Zillur Rahman	"
Professor S.M. Khorshed Alam Mazumder	"
Professor Md. Monwar Hossain	"
Dr. Md. Azharul Islam	"
Brig. Gen. Muhammd Shahid Khurshid Alam	"
Dr. Hossain Imam Al Hadi	"
Professor Khabir Uddin Ahmed	Member Secretary

Faculty of Ophthalmology:

Professor Md. Humayun Kabir	Chairperson
Professor M. A. Matin	Member
Professor Md. Mustafizur Rahman	"
Professor Md. Salehuddin	"
Professor Md. Abdul Halim Khan	"
Professor Md. Abdul Hadi Faquir	"
Professor Md. Saleh Ahmed	"
Professor Md. Israfil	"
Professor Sk. Md. Abdul Mannaf	"
Professor A.S.M. Kamaluddin	"
Professor Ava Hossain	"
Professor Jamal Nizamuddin Ahmed	"
Professor Syed Maruf Ali	"
Dr. Md. Shafiqul Islam	"
Professor Deen Mohd. Noorul Huq	"
Dr. Md. Shahidul Islam (Faruque)	"
Professor Md. Hassan Shahid Suhrawardy	"
Dr. Md. Abid Kamal	Member Secretary

Faculty of Psychiatry:

Professor A.K.M. Nazimuddowla Chowdhury	Chairperson
Professor Anwara Begum	Member
Professor M. A. Sobhan	"
Professor Md. Rezaul Karim	"
Professor Syed Kamaluddin Ahmed	"
Professor Md. Nazmul Ahsan	"
Professor Saroj Kumar Das	"
Dr. (Col. Retd.) Md. Nurul Azim	"
Professor Md. Enayet Karim	"
Professor Md. Golam Rabbani	"
Professor Abul Hasnat Mohammad Firoz	"
Professor Waziul Alam Chowdhury	"
Professor Mohammad Ahsanul Habib	"
Dr. Md. Sayadul Islam Mullick	"
Dr. Jhunu Shamsun Nahar	"
Dr. A.H.M. Mustafizur Rahman	"
Professor Mahmood Hasan	"
Dr. Md. Abdul Hamid	"
Dr. (Col.) Md. Sajjadur Rahman	"
Professor Md. Shah Alam	Member Secretary

Faculty of Obstetric & Gynaecology:

Professor A.H.M. Towhidul Anowar Chowdhury	Chairperson
Professor Shahla Khatun	Member
Professor Abdul Bayes Bhuiyan	"
Professor A.K.M. Anowarul Azim	"
Professor Monowara Amina Begum	"
Professor Sultana Jahan	"
Professor Latifa Shamsuddin	"
Professor Sultana Razia Begum	"
Professor Rehana Begum	"
Professor Mahmuda Khatun	"
Professor Anowara Begum	"
Professor Kohinoor Begum	"
Professor Sayeba Akhter	"
Professor Md. Shah Alam	"
Professor Nasima Begum	"
Professor Shamsun Nahar	"
Professor Merina Khanam	"
Professor Rowshan Ara Begum	"
Professor Md. Azizul Islam	"
Professor Sameena Chowdhury	"
Professor Laila Arjumand Banu	"
Dr. Fatema Begum	"
Professor Atika Begum	"
Professor Maliha Rashid	"
Professor M. Anwar Hussain	"
Professor Ameena Majid	Member Secretary

Faculty of Basic Medical Sciences:

Professor Syed Mukarram Ali	Chairperson
Professor M. H. Mullick	Member
Professor M. A. Hai	"
Professor A.K.M. Nurul Anowar	"
Professor S.A.R.Chowdhury	"
Professor F. A. Azim	"
Professor M. A. Rashid	"
Professor Md. Nazrul Islam (Virology)	"
Professor Jalilur Rahman	"
Professor Saleha Husain	"
Professor Md. Motahar Hossain	"
Professor Md. Ruhul Amin Miah	"
Professor Md. Shahadat Hossain	"
Professor Abdullah Akter Ahmed	"
Professor Badrul Islam	"
Professor Tareak-Al-Nasir	"
Professor Md. Ruhul Amin	Member
Professor Kh. Md. Shefayetullah	"
Professor Naima Muazzam	"
Professor Mohammed Kamal	"
Professor Kh. Manzare Shamim	"
Professor Nadira Islam	"
Professor Nilufar Sultana	"
Professor Shamim Ara	"
Major General Md. Jalal Uddin	"
Dr. (Col.) Zahid Mahmud	"
Dr. A.B.M. Yunus	"
Dr. (Brig. Gen.) Shazadi Nilufar	"
Professor Zinnat Ara Begum	"
Professor Nilufar Begum	"
Professor Humaira Naushaba	"
Dr. Firoza Khatun	"
Dr. Abida Ahmed	"
Professor Mohammad Mozammel Hoque	Member Secretary

Faculty of Paediatrics:

Professor Md. Nurul Islam	Chairperson
Professor Nazmun Nahar	Member
Professor Md. Ruhul Amin	"
National Professor M.R. Khan	"
Professor M.Q.K. Talukder	"
Professor Md. F. H. Nazir	"
Professor Md. Hamidur Rahman	"
Professor Chowdhury Badruddin Mahmood	"
Professor M.A. Mannan Miah	"
Professor Md. Moazzam Hossain	"
Professor Munimul Haque	"
Professor Kishwar Azad	"
Professor Md. Sirajul Islam	"
Professor Hosne Ara Begum	"
Dr. Md. Abdul Halim	"
Professor Choudhury Ali Kawser	"
Dr. Md. Badrul Alam	"
Professor Naila Zaman Khan	"
Professor Md. Nurul Absar	"
Professor Mohammad Hanif	"
Professor Md. Ekhlasur Rahman	"
Professor A.R.M. Luthful Kabir	"
Professor Ainun Afroza	"
Professor Syed Zahid Hossain	"
Professor Soofia Khatoon	"
Professor Syeda Afroza	"
Professor Md. Saeedur Rahman	"
Dr. Md. Nazrul Islam	"
Professor Golam Muin Uddin	"
Professor Md. Abid Hossain Mollah	"
Professor Abdul Hannan	Member Secretary

Faculty of Family Medicine:

Professor T. A. Chowdhury	Chairperson
Professor Mobin Khan	Member
Professor Quazi Deen Mohammad	"
Professor T.I.M.Abdullah-Al-Faruque	"
Professor Syed Atiqul Haq	"
Professor S.A.M. Golam Kibria	"
Professor Mahmud Hasan	"
Professor Md. Ruhul Amin	"
Professor Abdul Bayes Bhuiyan	"
Professor Nazmun Nahar	"
Professor Md. Sanawar Hossain	"
Professor M. A. Majed	"
Professor Tofayel Ahmed	"
Professor A.H.M. Ahsanullah	"
Professor A.N.M. Atai Rabbi	"
Professor M. A. Majid	"
Professor Md. Ashraf Hossain	"
Professor Md. Tahir	"
Professor Shamsuddin Ahmed	"
Professor Md. Abdul Hadi Faquir	"
Professor M. A. Mannan Miah	"
Professor Salim Md. Jahangir	"
Professor Choudhury Ali Kawser	"
Professor Muhammad Mahbubur Rahman	"
Professor Md. Moyeenuzzaman	"
Professor Mirza Mazharul Islam	"
Professor (Dr.) Falahuzzaman Khan	"
Professor A.Z.M. Maidul Islam	"
Professor Md. Abidul Haque	"
Dr. Md. Shams -Ul Alam	"
Dr. Md. Mosaddeque Hossain Biswash (Dumbel)	"
Professor A.H.M. Firoz	"
Dr. Nooruddin Ahmed	Member Secretary

The President and the Honorary Secretary of the College shall be the ex-officio members of all Committees and Faculties.