ISSN 1015-0870

April 2014
Vol. 32, No. 2

Journal of Bangladesh College of Physicians and Surgeons

Official Journal of
The Bangladesh College of Physicians and Surgeons

www.bcpsbd.org & www.banglajol.info/index.php/JBCPS
Enlisted in DOAJ/HINARI/AsiaJOL/BanglaJOL
Indexed in: IndexCopernicus,Ulrichsweb,Google Scholar,ProQuest,CrossRef,EBSCO

Journal of Bangladesh College of Physicians and Surgeons

Vol. 32, No. 2, April 2014

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

PUBLISHED BY

Dr. HAM Nazmul Ahasan
on behalf of the Bangladesh College of Physicians and Surgeons

PRINTED AT

Asian Colour Printing
130 DIT Extension Road
Fakirerpool, Dhaka-1000

ANNUAL SUBSCRIPTION

Tk. 400/- for local and US$ 40 for overseas subscribers

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, 9884189, 9884194, 9891865 Ext. 124, Fax : 880-2-8828928, E-mail : <bcps@bcps-bd.org> Editor's e-mail: journal.bcps@gmail.com

Journal of Bangladesh College of Physicians and Surgeons (JBCPS)

INFORMATION FOR AUTHORS

MANUSCRIPT PREPARATION AND SUBMISSION

Guide to Authors

The Journal of Bangladesh College of Physician and Surgeons, provides rapid publication (quarterly publication) of articles in all areas of the subject. The Journal welcomes the submission of manuscripts that meet the general criteria of significance and scientific excellence.

Papers must be submitted with the understanding that they have not been published elsewhere (except in the form of an abstract or as part of a published lecture, review, or thesis) and are not currently under consideration by another journal published by **INTERNATIONAL RESEARCH JOURNALS** or any other publisher.

The submitting (Corresponding) author is responsible for ensuring that the article's publication has been signed approved by all the other coauthors. It is also the authors' responsibility to ensure that the articles emanating from a particular institution are submitted with the approval of the necessary institutional requirement. Only an acknowledgment from the editorial office officially establishes the date of receipt. Further correspondence and proofs will be sent to the corresponding author(s) before publication unless otherwise indicated. It is a condition for submission of a paper that the authors permit editing of the paper for readability. All enquiries concerning the publication of accepted papers should be addressed to -

Editor-in-Chief
(Presently **Prof. HAM Nazmul Ahasan**)
BCPS Bhaban
67 Shaheed Tajuddin Sarani
Mohakhali, Dhaka 1212,
BANGLADESH
Phone: +8802-8825005 +8802-8825005
Fax: +88002-8828928

Electronic submission of manuscripts is strongly encouraged, provided that the text, tables, and figures are included in a single Microsoft Word file (preferably in Arial font).

Submit manuscripts as e-mail attachment to the editorial office at: journal.bcps@gmail.com

A manuscript number will be mailed to the corresponding author within two working days.

The **cover letter** should include the corresponding author's full address and telephone/fax numbers and should be in an e-mail message sent to the editor, with the file, whose name should begin with the first author's surname, as an attachment.

The Journal of Bangladesh College of Physicians and Surgeons will only accept manuscripts submitted as e-mail attachments or triplicate Hard copy with a soft copy

Article Types

FIve types of manuscripts may be submitted:

Editorials: It will be preferably written invited only and usually covers a single topic of contemporary interest.

Original Articles: These should describe new and carefully confirmed findings, and experimental procedures should be given in sufficient detail for others to verify the work. The length of a full paper should be the minimum required to describe and interpret the work clearly.

Short Communications: A Short Communication is suitable for recording the results of complete small investigations or giving details of new models or hypotheses, innovative methods, techniques , images in clinical practice, letter to editors, short reports or apparatus. The style of main sections need not conform to that of original article . Short communications are 2 to 4 printed pages (about 6 to 12 manuscript pages) in length.

Reviews: Submissions of reviews and perspectives covering topics of current interest are welcome and encouraged. Reviews should be concise and no longer than 4 to 6 printed pages (about 12 to 18 manuscript pages). It should be focused and must be up to date. Reviews are also peer-reviewed.

Case Reports: This should cover uncommon and/or interesting cases with appropriate confirmation process.

Review Process:

All manuscripts are initially screened by editor and sent to selective reviewer. Decisions will be made as

rapidly as possible, and the journal strives to return reviewers' comments to authors within 3 weeks. The editorial board will re-review manuscripts that are accepted pending revision. The JBCPS editorial board will try to publish the manuscript as early as possible fulfilling all the rigorous standard journal needs.

I. A. Preparing a Manuscript for Submission to JBCPS
Editors and reviewers spend many hours reading manuscripts, and therefore appreciate receiving manuscripts that are easy to read and edit. Much of the information in this journal's Instructions to Authors is designed to accomplish that goal in ways that meet each journal's particular editorial needs. The following information provides guidance in preparing manuscripts for this journal.

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:

1. Manuscript should be written in English and typed on one side of A4 (290 x 210cm) size white paper.
2. Margin should be 5 cm for the header and 2.5 cm for the remainder.
3. Style should be that of modified Vancouver.
4. Each of the following section should begin on separate page :
   - o Title page
   - o Summary/abstract
   - o Text
   - o Acknowledgement
   - o References
   - o Tables and legends.

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

I. A. 1. a. General Principles

- The text of observational and experimental articles is usually (but not necessarily) divided into the following sections: Introduction, Methods, Results, and Discussion. This so-called "IMRAD" structure is a direct reflection of the process of scientific discovery.
- Long articles may need subheadings within some sections (especially Results and Discussion) to clarify their content. Other types of articles, such as case reports, reviews, and editorials, probably need to be formatted differently.
- Electronic formats have created opportunities for adding details or whole sections, layering information, crosslinking or extracting portions of articles, and the like only in the electronic version.
- Authors need to work closely with editors in developing or using such new publication formats and should submit supplementary electronic material for peer review.
- Double-spacing all portions of the manuscript—including the title page, abstract, text, acknowledgments, references, individual tables, and

legends—and generous margins make it possible for editors and reviewers to edit the text line by line and add comments and queries directly on the paper copy.

- If manuscripts are submitted electronically, the files should be double-spaced to facilitate printing for reviewing and editing.
- Authors should number on right upper all of the pages of the manuscript consecutively, beginning with the title page, to facilitate the editorial process.

I. A. 1. b. Reporting Guidelines for Specific Study Designs

Research reports frequently omit important information. Reporting guidelines have been developed for a number of study designs that JBCPS journals ask authors to follow. Authors should consult the Information for Authors of this journal. The general requirements listed in the next section relate to reporting essential elements for all study designs. Authors are encouraged also to consult reporting guidelines relevant to their specific research design. A good source of reporting guidelines is the EQUATOR Network (http: //www.equator-network.org/home/) or CONSORT network (http:// www.consort-statement.org).

I. A .2. Title Page

The title page should have the following information:

1. Article title. Concise titles are easier to read than long, convoluted ones. Titles that are too short may, however, lack important information, such as study design (which is particularly important in identifying type of trials). Authors should include all information in the title that will make electronic retrieval of the article both sensitive and specific.
2. Authors' names and institutional affiliations.
3. The name of the department(s) and institution(s) to which the work should be attributed.
4. Disclaimers, if any.
5. Contact information for corresponding authors. The name, mailing address, telephone and fax numbers, and e-mail address of the author responsible for correspondence about the manuscript .
6. The name and address of the author to whom requests for reprints should be addressed or a Statement that reprints are not available from the authors.
7. Source(s) of support in the form of grants, equipment, drugs, or all of these.
8. A short running head or footline, of no more than 40 characters(including letters and spaces). Running heads are published and also used within the editorial office for filing and locating manuscripts.
9. The number of figures and tables. It is difficult for editorial staff and reviewers to determine whether he figures and tables that should have accompanied a manuscript were actually included unless the numbers of figures and tables are noted on the title page.

I. A. 3. Conflict-of-Interest Notification Page

To prevent potential conflicts of interest from being overlooked or misplaced, this information needs to be part of the manuscript. The ICMJE has developed a uniform disclosure form for use by ICMJE member journals (http://www.icmje.org/coi_disclosure.pdf) and JBCPS has accepted that.

I. A. 4. Abstract

- Structured abstracts are essential for original research and systematic reviews. structured abstract means introduction, methods, results and conclusion in abstract
- Should be limited to 250 words
- The abstract should provide the introduction of the study and blinded state and should state the study's purpose, basic procedures (selection of study subjects or laboratory animals, observational and analytical methods), main findings (giving specific effect sizes and their statistical significance, if possible), principal conclusions. It should emphasize new and important aspects of the study or observations. Articles on clinical trials should contain abstracts that include the items that the CONSORT group has identified as essential (http:// www.consort-statement.org).
- Because abstracts are the only substantive portion of the article indexed in many electronic databases, and the only portion many readers read, authors need to be careful that they accurately reflect the content of the article

I. A. 5. Introduction

- Provide a context or background for the study (that is, the nature of the problem and its significance). It should be very specific, identify the specify knowledge in the aspect, reasoning and what the study aim to answer.
- State the specific purpose or research objective of, or hypothesis tested by, the study or observation; the research objective is often more sharply focused when stated as a question.
- Both the main and secondary objectives should be clear.
- Provide only directly pertinent primary references, and do not include data or conclusions from the work being reported.

I. A. 6. Methods

The Methods section should be written in such way that another researcher can replicate the study.

I. A. 6. a. Selection and Description of Participants

- Describe your selection of the observational or experimental participants (patients or laboratory animals, including controls) clearly, including eligibility and exclusion criteria and a description of the source population. Because the relevance of such variables as age and sex to the object of research is not always clear, authors should explain their use when they are included in a study report—for example, authors should explain why only participants of certain ages were included or why women were excluded. The guiding principle should be clarity about how and why a study was done in a particular way. When authors use such variables as race or ethnicity, they should define how they measured these variables and justify their relevance.

I. A. 6. b. Technical Information

- Identify the methods, apparatus (give the manufacturer's name and address in parentheses), and procedures insufficient detail to allow others to reproduce the results. Give references to established methods, including statistical methods (see below); provide references and brief descriptions for methods that have been published but are not well-known; describe new or substantially modified methods, give the reasons for using them, and evaluate their limitations. Identify precisely all drugs and chemicals used, including generic name(s), dose(s), and route(s) of administration.
- Authors submitting review article should include a section describing the methods used for locating, selecting, extracting, and synthesizing data. These methods should also be summarized in the abstract.

I. A. 6. c. Statistics

- Describe statistical methods with enough detail to enable a knowledgeable reader with access to the original data to verify the reported results. When possible, quantify findings and present them with appropriate indicators of measurement error or uncertainty (such as confidence intervals).
- Avoid relying solely on statistical hypothesis testing, such as *P* values, which fail to convey important information about effect size. References for the design of the study and statistical methods should be to standard works when possible (with pages stated).
- Define statistical terms, abbreviations, and most symbols.
- Specify the computer software used.

I. A. 7. Results

- Present results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Please keep the result the sequence of specific objective selected earlier.
- Do not repeat all the data in the tables or illustrations in the text; emphasize or summarize only the most important observations. Extra or supplementary materials and technical detail can be placed in an appendix where they will be accessible but will not interrupt the flow of the text, or they can be published solely in the electronic version of the journal.
- When data are summarized in the Results section, give numeric results not only as derivatives (for example, percentages) but also as the absolute numbers from which the derivatives were calculated, and specify the statistical methods used to analyze them.
- Restrict tables and figures to those needed to explain the argument of the paper and to assess supporting data. Use graphs as an alternative to tables with many entries; do not duplicate data in graphs and tables.

- Avoid nontechnical uses of technical terms in statistics, such as “random” (which implies a randomizing device), “normal,” “significant,” “correlations,” and “sample.” Where scientifically appropriate, analyses of the data by such variables as age and sex should be included.

I. A. 8. Discussion

- Emphasize the new and important aspects of the study and the conclusions that follow from them in the context of the totality of the best available evidence.
- Do not repeat in detail data or other information given in the Introduction or the Results section.
- For experimental studies, it is useful to begin the discussion by briefly summarizing the main findings, then explore possible mechanisms or explanations for these findings, compare and contrast the results with other relevant studies, state the limitations of the study, and explore the implications of the findings for future research and for clinical practice.
- Link the conclusions with the goals of the study but avoid unqualified statements and conclusions not adequately supported by the data. In particular, avoid making statements on economic benefits and costs unless the manuscript includes the appropriate economic data and analyses. Avoid claiming priority or alluding to work that has not been completed. State new hypotheses when warranted, but label them clearly as such.

I. A. 9. References

I. A. 9. a. General Considerations Related to References

- Although references to review articles can be an efficient way to guide readers to a body of literature, review articles do not always reflect original work accurately. Readers should therefore be provided with direct references to original research sources whenever possible.
- On the other hand, extensive lists of references to original work of a topic can use excessive space on the printed page. Small numbers of references to key original papers often serve as well as more exhaustive lists, particularly since references can now be added to the electronic version of published papers, and since electronic literature searching allows readers to retrieve published literature efficiently.
- Avoid using abstracts as references. References to papers accepted but not yet published should be designated as “in press” or “forthcoming”; authors should obtain written permission to cite such papers as well as verification that they have been accepted for publication.
- Information from manuscripts submitted but not accepted should becited in the text as “unpublished observations” with written permission from the source.
- Avoid citing a “personal communication” unless it provides essential information not available from a public source, in which case the name of the person and date of communication should be cited in parentheses in the text. For scientific articles, obtain written permission and confirmation of accuracy from the source of a personal communication. Some but not all journals check the accuracy of all reference citations; thus, citation errors sometimes appear in the published version of articles. To minimize such errors, references should be verified using either an electronic bibliographic source, such as PubMed or print copies from original sources.
- Authors are responsible for checking that none of the references cite retracted articles except in the context of referring to the retraction. For articles published in journals indexed in MEDLINE, the ICMJE considers PubMed the authoritative source for information about retractions.

I. A. 9. b. Reference Style and Format

- References should be numbered consecutively in the order in which they are first mentioned in the text.
- Identify references in text, tables, and legends by Arabic numerals in superscript.
- References cited only in tables or figure legends should be numbered in accordance with the sequence established by the first identification in the text of the particular table or figure.

I. A. 10. Tables

- Tables capture information concisely and display it efficiently.

- Use tables /fig that are relevant to study
- Try to limit the number of tables/figure
- Type or print each table with double-spacing on a separate sheet of paper. Number tables consecutively in the order of their first citation in the text and supply a brief title for each.
- Do not use internal horizontal or vertical lines. Give each column a short or an abbreviated heading. Authors should place explanatory matter in footnotes, not in the heading. Explain all nonstandard abbreviations in footnotes, and use the following symbols, in sequence:

  *, †, ‡, §, _, ¶, **, ††, ‡‡, §§, _ _, ¶¶, etc.
- Identify statistical measures of variations, such as standard deviation and standard error of the mean.
- Be sure that each table is cited in the text. If you use data from another published or unpublished source, obtain permission and acknowledge that source fully.

I. A. 11. Illustrations (Figures)

- Figures should be either professionally drawn and photographed, or submitted as photographic-quality digital prints. In addition to requiring a version of the figures suitable for printing,

  (for example, JPEG / GIF)
- Authors should review the images of such files on a computer screen before submitting them to be sure they meet their own quality standards. For x-ray films, scans, and other diagnostic images, as well as pictures of pathology specimens or photomicrographs, send sharp, glossy, black-and-white or color photographic prints, usually 127 _ 173 mm (5 _ 7 inches)
- Letters, numbers, and symbols on figures should therefore be clear and consistent throughout, and large enough to remain legible when the figure is reduced for publication.
- Photographs of potentially identifiable people must be accompanied by written permission to use the photograph. Figures should be numbered consecutively according to the order in which they have been cited in the text.
- If a figure has been published previously, acknowledge the original source and submit written permission from the copyright holder to reproduce the figure. Permission is required irrespective of authorship or publisher except for documents in the public domain.
- For illustrations in color, JBCPS accept coloured illustration but when it seems essential. This Journal publish illustrations in color only if the author pays the additional cost. Authors should consult the journal about requirements for figures submitted in electronic formats.

I. A. 12. Legends for Illustrations (Figures)

- Type or print out legends for illustrations using double spacing, starting on a separate page, with Arabic numerals corresponding to the illustrations.
- When symbols, arrows, numbers, or letters are used to identify parts of the illustrations, identify and explain each one clearly in the legend. Explain the internal scale and identify the method of staining in photomicrographs.

I. A. 13. Units of Measurement

- Measurements of length, height, weight, and volume should be reported in metric units (meter, kilogram, or liter) or their decimal multiples.
- Authors should report laboratory information in both local and International System of Units (SI). .
- Drug concentrations may be reported in either SI or mass units, but the alternative should be provided in parentheses where appropriate.

I. A. 14. Abbreviations and Symbols

- Use only standard abbreviations; use of nonstandard abbreviations can be confusing to readers.
- Avoid abbreviations in the title of the manuscript.
- The spelled-out abbreviation followed by the abbreviation in parenthesis should be used on first mention unless the abbreviation is a standard unit of measurement.

I. B. Sending the Manuscript to the Journal

- If a paper version of the manuscript is submitted, send the required number of copies of the manuscript and figures; they are all needed for peer review and editing, and the editorial office staff cannot be expected to make the required copies.
- Manuscripts must be accompanied by a cover letter, conflicts of interest form, authorship and declaration, proforma of which is available in JBCPS web site.

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 manuscripts are edited according to the Journal's style.

Submission Preparation Checklist

As part of the submission process, authors are required to check off their submission's compliance with all of the following items, and submissions may be returned to authors that do not adhere to these guidelines.

Check Lists

Final checklists before you submit your revised article for the possible publication in the Journal of Bangladesh College of Physicians and Surgeons:

1. Forwarding/Cover letter and declaration form
2. Authorship and conflicts of interest form
3. Manuscript
    - o Sample of the above documents is available in the following links: http://www.bcpsbd.org (registration required for download)
    - o If you have submitted mention document (1, 2, 3) above, when you first submitted your article then you don't need to re-submit but if there is change in the authorship or related then you have to re-submit it.

- General outline for article presentation and format
    - Δ Double spacing
    - Δ Font size should be 12 in arial
    - Δ Margins 5 cm from above and 2.5 cm from rest sides.
    - Δ Title page contains all the desired information (vide supra)
    - Δ Running title provided (not more than 40 characters)
    - Δ Headings in title case (not ALL CAPITALS, not underlined)
    - Δ References cited in superscript in the text without brackets after with/without comma (,) or full stop (.)
    - Δ References according to the journal's instructions – abide by the rules of Vancouver system. Use this link to get into the detail of Vancouver system.
- **Language and grammar**
    - Δ Uniformity in the language
    - Δ Abbreviations spelt out in full for the first time
    - Δ Numerals from 1 to 10 spelt out
    - Δ Numerals at the beginning of the sentence spelt out
- **Tables and figures**
    - Δ No repetition of data in tables/graphs and in text
    - Δ Actual numbers from which graphs drawn, provided
    - Δ Figures necessary and of good quality (colour)
    - Δ Table and figure numbers in Arabic letters (not Roman)
    - Δ Labels pasted on back of the photographs (no names written)
    - Δ Figure legends provided (not more than 40 words)
    - Δ Patients' privacy maintained (if not, written permission enclosed)
    - Δ Credit note for borrowed figures/tables provided
    - Δ Each table/figure in separate page

If you have any specific queries please use at www.bcps.com

Manuscript Format for Research Article

- **Title**
    - Δ Complete title of your article
    - Δ Complete author information
    - Δ Mention conflict of interest if any

- **Abstract**
  - Δ Do not use subheadings in the abstract
  - Δ Give full title of the manuscript in the Abstract page
  - Δ Not more than 200 words for case reports and 250 words for original articles
  - Δ Structured abstract (Including introduction, methods, results and discussion, conclusion) provided for an original article and (Introduction, results and discussion , conclusion) for case reports.
  - Δ Key words provided – arrange them in alphabetical order (three – five)
- **Introduction**
  - Δ Word limit 150 -200 words
  - Δ Pertinent information only
- **Material and Methods**
  - Δ Study Design
  - Δ Duration and place of study
  - Δ Ethical approval
  - Δ Patient consent
  - Δ Statistical analysis and software used.
- **Result**
  - Δ Clearly present the data
  - Δ Avoid data redundancy
  - Δ Use table information at the end of the sentence before full stop between the small bracket
- **Discussion**
  - Δ Avoid unnecessary explanation of someone else work unless it is very relevant to the study
  - Δ Provide and discuss with the literatures to support the study
  - Δ Mention about limitation of your study
- **Conclusion**
  - Δ Give your conclusion
  - Δ Any recommendation
- **Acknowledgement**
  - Δ Acknowledge any person or institute who have helped for the study
- **Reference**
  - Δ Abide by the Vancouver style
  - Δ Use reference at the end of the sentence after the full stop with superscript
- **Legends**
  - Δ Table
  - Δ Figures

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

Indexed on HINARI, EMSCO, DOAJ, Index Copernicus, Ulrichs Web, Google Scholar, CrossRef, ProQuest, Scientific Common.

BanglaJOL is supported by IN

JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

Vol. 32, No. 2, Page 59-121 **April 2014**

CONTENTS

EDITORIAL

Training in Post Graduate Medical Education: Demands Rethinking

In general primary goal of doctor in care and well being of patients. This requires to acquire knowledge, learn clinical skills and develop professional attitudes. This can be achieved through a scheduled training curriculum identifying the central importance of competence-based learning, underpinned by structured, observational assessments. At the heart of the curriculum is patient safety and care within the framework of clinical governance. The emphasis on developing medical practitioners who are patient-focused and accountable to the public for delivering safe medical care is fundamental to creating the medical workforce of the future. It focuses on both clinical skills – with particular emphasis on chronic disease management and the care of acutely ill patients – as well as the development of more generic skills such as communications and team working. Doctors in training must develop the capacity to reflect on the relationship between personal performance and those outcomes and to learn from efforts to change

Bangladesh College of physicians and Surgeons (BCPS) as a pioneer and authorized organization in this field is playing pivotal role in health manpower development and maintenance of quality health services in the country. Despite its all-out effort, the optimization of the quality of its system and thus the outcome is sometimes questioned especially in the face of continuous piling of huge number of unsuccessful candidates in post graduate final examination. To overcome these, a number of relevant issues, related to training of doctors have been raised to address, viz. motivation of pursuit students, place of training, attitude & opportunities of the trainers, monitoring of training and distribution & number of trainees under a supervisor.

Lack of motivation of students is not straightway applicable to them as they entered the course through competitive examination conducted by the BCPS. Though controversies arises on inconsistent results in different sessions and in different specialties. Adequate and appropriate inputs from the senior teachers are earnestly sought; introduction of single best answer (SBA) type questions partially improved the assessment system but the target still remains to be reached. So, to develop a fair system of selection of optimum number of quality candidates for training is a crying need.

Institution and or place at which candidates take their clinical training have impact on their learning attitude and spectrum. It is the fact that all institutes where training is officially recognized are not equally equipped, both in infrastructures and patient management system; so the training outcome may vary.

Teachers, supervising postgraduate trainee are not always and equally oriented and optimized to training contents and modules due continuous updating. So regular refresher course, involving both senior and junior teachers supervising training might be of help.

In the existing system, a trainee, being placed to a training position at one or a number of recognized institutions is to complete the stipulated tenure to get a certificate duly signed both by the supervisor and hospital authority which confer him/her eligibility to seat for exam in clinical part. This mere certification of complete training is not ensuring the optimum learning as are reflected in their performance in exams. So stratification of learning in training period and structuring of each stratum with fixing up of time frame to complete each term through assessment is urgently warned.

We are already inundated with a number of qualified students, but do not have specific plan to train them. As the size of the enrolled students have outnumbered the minimum number of institutes and supervisors, we shall have to find out maximum number of students under a supervisors using all the training institutes of the country to train them within possible shortest period of time.

To address these issues, BCPS needs a coordinated approach involving the ministry of Health, Directorate

of Health Services. NGOs and other health related private organization and institutes. Keeping in mind the existing and continuously increasing demand of specialist doctors, we shall have to have approximate calculation of number of candidates whom we can provide adequate training with possible drop out during and after completion of training. Accordingly, on standardizing the examination system, like the Royal Colleges of UK to select the top one third , expected number of candidates may be recruited from the top scorers not merely those who obtained qualified marks.

Total course of each specialty will have to be continuously updated by the qualified experience teacher of that specialty and learning level will have to be staged. Contents at each level will be structured. As candidates at various levels, need different level of training and all the recognized institutes are not equally equipped, institutions may be ranked according to their existing facilities and students as such may be placed in one of the hospitals of similar categories on rotation basis maintaining the hierarchy of training levels and institutions. Irrespective of experience, all the supervisors should have refresher course highlighting the update and implementation systems of training program from time and again.

Over all, we shall have to keep in mind that, postgraduate doctors we are producing are also teachers of our Medical institutes. So, component of postgraduate training should contain Continued Professional Development programme including Teaching methodology, Research and Assessment of performance.

Another important aspect and barrier of training is the payment, because a substantial proportion of our qualified students are not already in job. So this issue will have to be addressed appropriately.

The most important aspect of training is the monitoring and evaluation. Presently, a six monthly reporting by the supervisor is in practice. Further improvement may be done through regular evaluation and monitoring of training contents, quality of teaching and supervision, assessment and trainee progress. Both supervisors and trainee will have to contribute to the monitoring and evaluation process. Feedback from the supervisors will be systematically sought, analyzed and will be used as part of the monitoring process. Feedback from the trainee will also be sought confidentially regarding training, clinical experience and the quality of supervision. Comments, suggestions and advice may also be incorporated in the improvement of professional development process thus improved and modern treatment with time demanded health care services may be provided to the people of our community.

The training has no meaning or relevance if it is not set in the context of patient care. Its roots are in the service, not in the classroom. Our new doctors must be encouraged to immerse themselves in a clinical environment where this service is delivered.

(J Banagladesh Coll Phys Surg 2014; 32: 59-60)

Prof. ABM Maksudul Alam
Councilor, BCPS
and Principal & Professor of Anaesthesiology
Shaheed Suhrawardy Medical College
Sher-e-Bangla Nagar, Dhaka

References:

1. A guide to CPSP training programs. Kerachi: Coll Physicians Surg Pak;2005
2. John SC Biggs: Postgraduate Medical Training in Pakistan: Observations and Recommendations; Journal of The College of Physicians and Surgeons of Pakistan 2006,Vol. (1):58-63
3. Curricullum for the foundation years in postgraguate education and training. By- The Foundation Programme Committee of Academyof Medical Royal Colleges, in co-operation with Modernising Medical Careers in the Departments

Role of Serial C-reactive Protein (CRP) in Relation to Total Leucocyte Count, Platelet Count & Blood Culture for early Diagnosis of Neonatal Septicemia in Developing Countries

C BARUA[a], MN ANWAR[b], M SHAHIDULLAH[c], S HOSSAIN[d], S BARUA[e], N CHOWDHURY[f]

Abstract:

Neonatal septicemia is a clinical syndrome of systemic illness accompanied by bacteremia occuring in the first 28 days of life. Neonatal septicemia is one of the major causes of neonatal death in developing countries. Early diagnosis and treatment can prevent neonatal mortality and morbidity. The present study includes: 1) usefulness of CRP (C-reactive protein), Total Leucocyte Count, Platelet Count and Blood Culture in early diagnosis of Neonatal Sepsis, 2) significance of serial CRP in diagnosis of neonatal sepsis. 3) the prognostic value of CRP in neonatal sepsis. This is a prospective study done in neonatal ward, Chittagong Medical College Hospital and carried out from January 2008 to January 2011. Sample size was 300. One hundred fifty neonates with suspected sepsis as cases and 150 healthy babies as control were enrolled in this study. Seventy two percent of cases neonates were preterm and low birth weight. Common risk factors for neonatal septicemia which were identified in this study; preterm (72%), low birth weight (72%), premature rupture membrane (60%), chorioamnionitis (26%) and maternal urinary tract infection (16%) . Out of 150 cases of suspected neonatal sepsis total 80.7%% had raised CRP, in initial sample 70.39% were CRP positive and in 2nd sample additional 9.31% case were CRP positive . In control group 91% were CRP negative. CRP was positive in 100% of culture proven sepsis. Sensitivity of CRP was 80.67% and specificity of CRP was 76.44%. Leucocytosis was observed in 7% of cases and leucopenia was found in 11% of cases. In 82 % cases leucocyte count was found normal. In control group, 95% had normal leucocyte count and 5% had leucocytosis but no leucopenia. Sensitivity of leucocyte count was 18% and specificity was 20.68%. Thrombocytopenia was found in 28% of case group. Out of 150 cases only 15.33% yielded growth of organisms in blood culture. Klebsiella was the most common pathogen isolated which was followed by E.coli and Strph. aureus. Sensitivity of blood culture was 15.33% and specificity was 100% Therefore serial CRP can be taken as alternative method for diagnosis of neonatal sepsis specially in developing countries where blood culture is not readily available.

Keyword: CRP in neonatal sepsis

(J Banagladesh Coll Phys Surg 2014; 32: 61-65)

Introduction:

Neonatal septicemia is a clinical syndrome of systemic illness accompanied by bacteremia occurring in the first 28 days of life[1]. Neonatal sepsis may be categorized as early onset, late onset and late onset sepsis[2].Early onset sepsis occurs after birth to 7 days of life and is acquisition of microorganisms from the mother. It may occur through transplacental infection or an ascending infection from the mother's genitourinary tract. The infant may acquire the microbe by passage through a microbes colonized at birth canal during delivery. In global perspective the microorganisms most commonly associated with early onset of infection include group B *Streptococci , Escherichia coli, Heomophilus influenzae, Listeria monocytogenes* [3]. Late onset sepsis occurs at 7-28 days of life and is acquired from the care giving environment. Organisms that have been implicated in causing late onset sepsis include coagulase-negative *Staphylococci*, *Staphylococci aureas*, *E.coli*, *Klebsiella*, *Pseudomonas*, *Enterobacter*,

a. Dr. Chiranjib Barua, Associate Prof. Neonatalogy, Chittagong Medical College.
b. Prof. Md. Nurul Anwar, Dean, Faculty of Biological Sciences, Chittagong University.
c. Prof. Md. Shahidullah, Fellow Neoborn Medicine, Pro-VC, Chairman, Neonatalogy, BSMMU.
d. Prof. Shahadat Hossain, Ex Head of the Dept. of Paed., USTC
e. Dr. Sharmila Barua, Asso. Prof., Gynae, Chittagong Medical College.
f. Dr. Nobel Chowdhury, Medical Officer, Chittagong Medical College Hospital.

Address of Correspondence: Dr. Chiranjib Barua, Associate Prof. Neonatalogy, Chittagong Medical College.

Received: 1 January, 2013 **Accepted:** 19 March, 2014

Candida, *Streptococcus* and anaerobes [4]. Non typeable H. influnzae sepsis has been identified in neonates, especially premature neonates .Candida are increasing important cause of late onset neonatal sepsis [2].

Early onset sepsis occurs after birth to 7 days of life and is acquisition of microorganisms from the mother[1,2]. Late onset sepsis occurs at 7-28 days of life and is acquired from the care giving environment. The incidence of neonatal sepsis is 5 to 8 per 1000 birth, the highest rates occur in LBW, perinatal asphyxia, maternal infection and congenital abnormality babies[2]. Culture positive neonatal sepsis in the USA is 0.7% but in very low birth weight infants under prolonged intensive care the rate of culture proven sepsis may be high as 30%[5].In the developing world neonatal sepsis is one of the commonest cause of perinatal mortality in the developing world[6]. Mortality rate of early onset neonatal sepsis is 2 to 40% and late onset neonatal sepsis is 2 to 20%[2]. Definitive diagnosis of neonatal sepsis is based on positive blood or cerebrospinal fluid(CSF) culture, which both take at least 24 to 48 hours and are often falsely negative. Determination of C-reactive protein (CRP), Total Leucocyte Count and Platelet Count have been used to establish or rule out neonatal infection and to aid the decision of to terminate unwanted antibiotic therapy[7, 8, 9, 10]. CRP produced by the liver under the influence of IL1 when inflammation is present. CRP rises up to 50,000-fold in acute inflammation, such as infection. It rises above normal limits within 6 hours, and peaks at 48 hours. Its half-life is constant, and therefore its level is mainly determined by the rate of production.

Aims And Objectives:

General objective: Reduction of neonatal morbidity and mortality by early diagnosis of Neonatal sepsis.

Specific objectives:

1) To find out the role of CRP, Total Leucocyte Count, Platelet Count and blood culture in early diagnosis of neonatal sepsis.
2) To detect the significance of serial CRP in diagnosis of neonatal sepsis.
3) To find out the prognostic value of CRP in neonatal sepsis.

Inclusion criteria:1) Preterm & Term neonates.2) Normal birth weight, low birth weight & very low birth weight neonates.3) Clinical features of sepsis.

Criteria of control group: Term/Pre-term, normal birth weight/low birth weight, without any evidence of sepsis, asphyxia or history of maternal infection.

Exclusion criteria: 1) Very sick neonates 2) Congenital abnormality 3) Perinatal asphyxia 4) Birth trauma.

Procedure: First blood sample (3 ml) collected at the day of admission (excluding 1st 24 hours of life) and another sample (1.5 ml) of blood drawn at 5th of day of admission. Proper hand washing was done with soap and water and subsequently hands were dried by using sterile (autoclaved) small towel. 3ml of blood from each patient were collected from single venipuncture. One ml of blood was taken into a clean and dry test tube for estimation of C-reactive protein. Another 1ml of blood was taken in a dry clean small vial containing anticoagulant for estimation of leucocyte and platelet count and rest 1ml of blood was introduced at bed side into blood culture bottle. Second sample of blood sent for CRP estimation only. CRP value 6 mg/L or more was taken as CRP positive.

Ethical aspects: Permission from parents were taken before enrolling each baby in this study and discussed clearly.

Statistical Analysis: Data are presented as the percentage of total number of observations. SPSS for windows (Vs.11) was used for the analysis of data t-test and x2 – test were used where appropriate .P<0.05 was used as the minimum level of significance.

Results:

Table-I

Distribution of weight in study groups and comparison between normal birth wt. & low birth wt. babies

Birth weight	Case (n=150)	Control (n=150)	P value
1000- <1500 gm	48 (32%)	9 (6%)	
1500- <2500 gm	60 (40%)	48 (32%)	< 0.001
≥2500 gm	42 (28%)	93 (62%)	
<2500 vs ≥2500 gm	5.93/p<0.001		

In this table, it was seen that infections occurred more frequently in <2500gm group Chi-Square test value is 5.93, P value < 0.001 which is statistically significant.

Table-II

Platelet count in case and control group

Platelets count	Case (n=150)	Control (n=150)
Normal (150000-300000/mm$^{3)}$	108(72%)	150(100%)
Thrombocytopenia (< 150000/ mm^3)	42(28%)	0

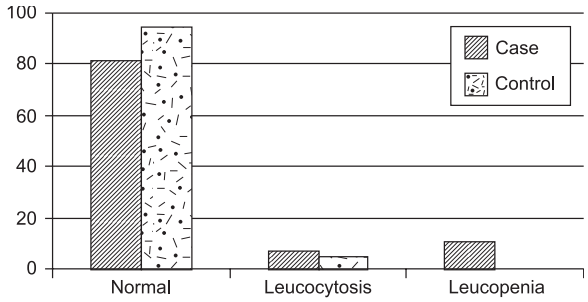


Fig.-1: *Leucocyte status in case and control groups*

Fig.-1 shows leucocytosis, leucopenia and normal leucocyte counts. Seven percent leucocytosis and 11% leucopenia were recorded in the present study but 82% cases showed normal leucocyte count.

Table-III

Blood culture in case and control groups

Culture in Study Groups			Study Groups		Total		
			Case	Control		x^2 test	P value
Culture	Positive	Count	23	0	23		
		% within Study Groups	15.3%	0.0%	7.7%	24.91	<0.001
	Negative	Count	127	150	277		
		% within Study Groups	84.7%	100.0%	92.3%		
Total		Count	150	150	300		
		% within Study Groups	100.0%	100.0%	100.0%		

Table III shows out of 300 enrolled neonates 23 (15.3%) cases were culture positive. P value is<.001 which is statistically highly significant.

Table-IV

Organisms isolated from Blood Culture in case group(n=23)

Organism isolated	Number	Percentage
Klebsiella	13	56.52
E. coli	7	30.43
Staphy. Aureus	3	13.05

Table-IV Shows that Gram Negative Klebsiella was the commonest organism isolated in blood culture. E. coli and Staphylococcus aureus were also common organisms.

Table-V

C-reactive protein (CRP) in study groups.

CRP	Study Groups		Study Groups		Total		
			Case	Control		x^2 test	P value
CRP	Positive	Count	121	25	146		
		% within Study Groups	80.67%	16.7%	48.7%	122.96	< 0.001
	Negative	Count	29	125	154		
		% within Study Groups	19.33%	83.3%	51.3%		
Total	Count	150	150	150	300		
		% within Study Groups	100.0%	100.0%	100.0%		

Table-V shows that majority of cases (80.67%) were CRP positive . Only 19.33% cases were CRP negative.

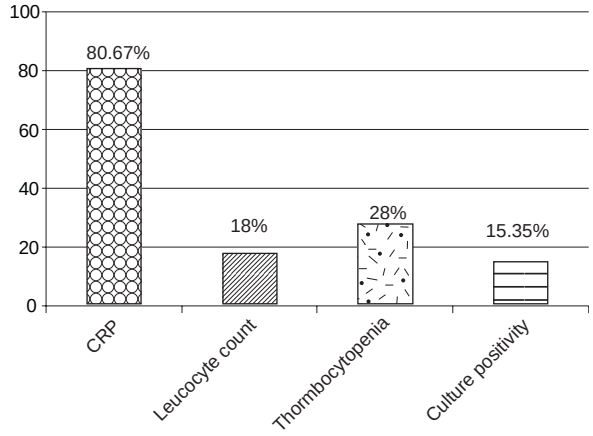


Fig.-2: *Comparative scenario among CRP, leucocyte count, thrombocytopenia and blood culture.*

Fig.-2 shows among three lab. investigations CRP positivity was high (80.67%) thrombocytopenia(28%), leucocytosis and leucopenia (18%). Only 15.35% cases were culture positive.

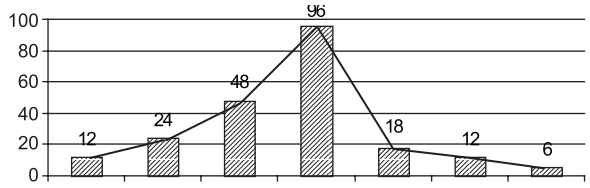


Fig.-3: *CRP in day 1 and day 5(mg/dl)*

Histogram shows high CRP values gradually decline to normal in case group in day 1 and day 5. It indicates prognostic significance of CRP.

Discussion:

Septicemia was found more common in male (55%) than female. male-female ratio was 1.2:1[12]. Neonatal sepsis observed more in low and very low birth weight babies[1, 12]. Out of 150 cases of suspected neonatal sepsis 80.67% had raised CRP. In culture proven sepsis 100% cases had raised CRP[13, 11]. In this study, at initial blood sample CRP positive was 70.39% and in second sample 9.31% were CRP positive but in control group only 16.7% had raised CRP. Forty three per cent of initial sample CRP values were 18mg/dl or more and decline to 12mg/dl or to 6mg/dl in second sample who were under antibiotic treatment. Such finding indicates prognostic significance of CRP[15]. Leucocytosis was observed in 7% of cases and leucopenia was found in 11% of cases. Though in 82% patients leucocyte count was within the normal limit. Significance of leucocyte count in the diagnosis of sepsis is low. In control group, 95% had normal leucocyte count and only 5% had leucocytosis[14] but no leucopenia. Thrombocytopenia was observed in 28% cases. Thrombocytopenia was not found in control group[2,17]. Sensitivity of CRP was 80.67% and specificity was 78.44%, sensitivity of blood culture was recorded 15.33% though specificity was high 100%. Sensitivity of leucocyte count (leucocytosis and leucopenia) was18% but specificity also low (20.68%). So, CRP was more sensitive investigation tool than other laboratory tests. A negative CRP is important than a positive CRP value in that it excludes infection with a high certainty. Out of 150 neonates with suspected septicemia, the present study showed only 15.33% had growth in blood culture. In control group blood culture yielded no growth[14]. Among the isolated organisms, 87% were gram-negative and rest13% were gram positive. The predominance of gram negative organisms in neonatal septicemia is similar to the observation of other investigations from Bangladesh, India, Pakistan and Nepal[15,16]. Klebsiella was the commonest pathogen isolated (56.52%) followed by E. coli[6,15,13].

Conclusion: In neonatal sepsis, there is no significant change of leucocyte count but thrombocytopenia is not uncommon. CRP is easier to measure, cheaper and the result is available earlier. Serial CRP is one of the most sensitive tests for early diagnosis of neonatal sepsis especially in developing countries.

References:

1. Gotoff SP. Infection of the neonatal infant. In: Behrman RE, Kliegman RM, Jenson HB, eds. 2000. Nelson textbook of pediatrics. 16th ed. Philadelphia: WB Saunders Company. 538-549.
2. Mery TC,oct.2009. Neonatal sepsis.National Institute of Health . Online Medical Library.
3. Placzek MM and Whitelow A;1983. Early and late neonatal septicemia. Arch Dis Child ; 58: 728-731.
4. Stoll BJ, Gordon T and Korones SB;1996. Early onset sepsis in very low birth weight neonates: a report from the National Institute of Child Health and Human Development Neonatal Research Network. Journal of Pediatric; 129:72-80.
5. Peter D;1999. Infection in the newborn. In: Rennie Jm, Roberton NRC, eds. Textbook of Neonatology. 3rd ed. London: Churchill Livingstone : 1109.
6. Gupta P, Murali MV, Faridi MMA, Kaul PB, Ramchandran VG and Talwar V;1993.Clinical profile of Klebsiella septicemia in neonates. Indian J Pediatr ; 60: 565-572.

7. Tessin I, Trollfors B and Thringer K.; 1990. Incidence and aetiology of neonatal septicemia and meningits in Western Sweden 1975-86. Acta Paediatr Scand ; 79: 1023-1030.

8. Boo NY and Chor CY ; 1994. Six-year trend of neonatal septicemia in a large Malaysian maternity hospital. *J Pediatr Child Healt*, 30: 23-27.

9. Guerina NG;1998. Bacterial and fungal infection. In: Cloherty JP and Stark AR, eds. Manual of neonatal care. 4th ed. Philadelphia: Lippincott-Raven Publishers, 271-299.

10. Polin RA and Geme JWS;1992. Neonatal sepsis. Adv Paediatr Infect Dis;7: 25-60.

11. Magudumana MD, Ballot DE, Copper PA, Cory BJ, Viljoen E and Carter AC;2000. Serial IL-6 measurements in the early diagnosis of neonatal sepsis. J Trop Pediatr ; 46: 267-277.

12. Reghavan M, Mondal GP, Bhat BV and Srinivasan S;1992. Perinatal risk factors in neonatal infections. Indian J Pediatr ; 59: 335-340.

13. Chiesa C, Panero A, Osban JF, Simonetti AF,2004. Diagnosis of Neonatal Sepsis.Clinical Chemistry ; 50: 279-287.

14. Chaturvedi P, Agrawal M and Narang P; 1989. Analysis of blood culture isolates from neontaes of a rural hospital. Indian Pediatr ; 26: 460-465.

15. Bhutta ZA, Naqbi SH, Muzaffar T and Farroqui BJ; 1991. Neonatal sepsis in Pakistan. *Acta Paediatr Scand* ; 80: 596-601.

16. Karki BM and Parija SC.; 1999. Analysis of blood culture isolates from hospitalized neonates of Nepal. SE Asia J Trop Med Pub Health . 30: 546-548.

17. Ayenger V, Madhulika and Vani SN;1991. Neonatal sepsis due to vertical transmission from maternal genital tract. Indian J Pediatr , 58: 661- 664.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Maternal Outcome of Prolonged Pregnancy

P AKHTER[a], M SULTANA[b], M HOQUE[c], S SULTANA[d], R KHATUN[e], S RANI[f]

Summary:

The probability of a pregnancy terminating in a full-term, healthy live birth is a powerful indicator of the health status of its women, and of the quality of health care available to them during pregnancy and birth. The present study conducted to find out the maternal outcome of prolonged pregnancy. This study carried out in the department of obstetrics, Sir Salimullah Medical College and Mitford Hospital, Dhaka, between the period of February 2003 and December 2003. Patients admitted in labour ward having the history of post dates but not in labour and some were admitted during first stage of labour. Patients who were sure about their Last Menstrual Period (LMP) and those patients who had regular menstrual cycle were included in the study. Total 139 respondents were included in the study. Among them 114 (82.01%) were in the age group of 18 to 29 years and 25 (17.99%) were in the age group of ≥30 years. Among the respondents 67 (48.2%) were primi gravida and 72 (51.8%) were multi gravida. Among the respondents 92 (66.2%) were in the 1st stage of labour, 7 (5.0%) were in the 2nd stage of labour and rest 40 (28.8%) were not in labour. Mode of delivery of highest number of respondents was caesarian section (54.0%) followed by normal vaginal delivery (39.7%). Other mode of delivery were ventouse and forceps and they were 07(05.0%) and 2(1.4%) respectively. Out of 75 respondents under gone caesarian section, indication of C/S was fetal distress in 1st stage of labour, prolong 1st stage with maternal distress, failed induction, cephalopelvic disproportion (CPD) and breech presentation with big baby were 32.0%, 25.3%, 24.0%, 16.0% and 2.7% respectively. Maternal morbidity like PPH, UTI, puerperal sepsis and wound infection were 10.0%, 14.40%, 3.60% and 5.70% respectively. In postdated pregnancy maternal morbidity is common finding. It also has more operative interference.

Key word: Postdated pregnancy; Maternal morbidity.

Abbreviation: LMP: Last Menstrual Period; CPD: Cephalopelvic disproportion; PPH: Post partum haemorrhage; UTI: Urinary tract infection.

(J Banagladesh Coll Phys Surg 2014; 32: 66-70)

Introduction:

Pregnancy is a time when women's health is placed at risk; however, health professionals providing prenatal care can reduce that risk by monitoring women's health regularly and offering preventive services[1]. Every year more than 200 million women become pregnant[2]. Most pregnancies of healthy mothers end with the birth of a live baby. But, in many cases, childbirth is not the joyous event as it should be but a time of pain, fear, suffering and even death[3,4]. The probability of a pregnancy terminating in a full-term, healthy live birth is a powerful indicator of the health status of its women, and of the quality of health care available to them during pregnancy and birth. Improvement of pregnancy outcome is thus an important area of action for those concerned with the improvement of women's health[5]. Although maternal mortality is a significant global health issue, achievements in mortality decline to date have been inadequate[6]. During the last decade the high number of maternal deaths has caused growing public concern. Ninety nine per cent of the deaths occur in developing countries and various strategies have been promoted to reduce maternal mortality[7, 8]. South Asia accounts for half of the world's annual maternal deaths, although it contributes only 29% of the deliveries in the world. Nearly three and half million births occur in Bangladesh every year and most of the deliveries are conducted at home by untrained persons[9]. In the late 1980s, one out of every 33 women in Bangladesh was estimated to have died of such complications, compared with nearly one out of 10,000 women in northern Europe. In 1987, the

a. Dr Parul Akhter, Junior Consultant (Gynae), 200 Beded Hospital, Narayanganj

b. Dr. Masuda Sultana, Junior Consultant (Gynae), UHC, Meghna, Comilla.

c. Dr. Mahbuba Hoque, Junior Consultant (Gynae), Sadar Hospital, Narsingdi.

d. Dr. Sharmin Sultata, Gynae outdoor. 200 Beded Hospital, Narayanganj

e. Dr. Mst. Rahima Khatun, Junior Consultant (Gynae), Sadar Hospital, Satkhira.

f. Dr. Seema Rani Dabee. Junior Consultant (Gynae), 20 Beded Hospital, Aminbazar, Savar, Dhaka.

Address of Correspondence: Dr. Parul Akhter, Junior Consultant (Gynae), 200 Beded Hospital, Narayanganj. Mobile: 01711648370, E-mail: drparulakhter@yahoo.com

Received: 28 February, 2013 **Accepted:** 13 January, 2014

international Safe Motherhood Initiative was launched to address this problem. Unfortunately, progress has been slow, partly because of a lack of consensus on how best to reduce maternal deaths[10].

The timely onset of labor and birth is an important determinant of perinatal outcome[11]. The World Health Organization defines a post-term pregnancy as one that has extended to or beyond 42 weeks (294 days) of gestation. Resources and maternal wishes need to be considered when managing a prolonged pregnancy[12,13,14,15,16]. Prolonged pregnancy is associated with an increased risk of fetal and maternal complications[11, 15]. The cause of prolonged pregnancy is not clear and may represent simple etiological variation. Post-term pregnancy is more common in primigravid women and a previous prolonged pregnancy gives a relative risk of 2.2 for subsequent pregnancies to be prolonged[13]. In most developed countries, prolonged pregnancy is now managed by planned delivery[17]. It is recognized as a high-risk problem faced by obstetricians. Perinatal morbidity and mortality are increased significantly and, for that reason, most obstetric units offer routine induction of labour between 41 and 42 weeks of gestation to minimize the adverse perinatal risks[13, 14,16]. Most women with a previous caesarean section have repeat caesarean delivery if the pregnancy becomes prolonged, as induction is associated with an increased risk of uterine rupture[17].

Methodology:

The objective of the present study was to determine the maternal outcome of prolonged pregnancy. This study carried out in the department of obstetrics, Sir Salimullah Medical College and Mitford, Hospital, Dhaka, between the period of February 2003 and December 2003. During this period, patients admitted in labour ward were taken for this study. Patients were admitted in labour ward having the history of post dates but not in labour and some were admitted during first stage of labour. Patients who were sure about their L.M.P and those patients who had regular menstrual cycle were included in the study. Patients who were unable to give accurate history of their L.M.P Patients who had infrequent menstruation prior to existing pregnancy, Patients of other high risk groups e.g. PET, Ecclampsia, Heart disease, Diabetics, Renal disease, IUGR, Patients with systemic hypertension, Multiple Pregnancies and congenital abnormalities were excluded. Pregnancy occurring during lactational amenorrhoea, pregnancy with venereal diseases and pregnancy with blood group incompatibility were also excluded from the study.

Results:

Total 139 respondents were included in the study. Among them 114 (82.01%) were in the age group of 18 to 29 years and 25 (17.99%) were in the age group of e”30 years. Duration of pregnancy of 80 (57.6%) was 41 weeks, 39 (28.0%) was 42 weeks, 17 (12.2%) was 43 weeks and rest 3 (2.2%) was 44 weeks. Among the respondents 67 (48.2%) were primi gravida and 72 (51.8%) were multi gravida. Clinically ninety nine (71.2%) respondents presented with adequate liqour amnii and 40 (28.8%) were presented with scanty liqour amnii. Ultrasonographically 40 (56.3%) respondents presented with adequate liqour amnii, 15 (21.1%) presented with inadequate liqour amnii and 16 (22.6%) were presented with scanty liqour amnii. Among the respondents 92 (66.2%) were in the 1st of labour, 7 (5.0%) were in the 2nd stage of labour and rest 40 (28.8%) were not in labour. Among the respondents not in labour, twenty four (60.0%) had favourable cervix and 16 (40.0%) had unfavourable cervix. Sweeping, sweeping + ARM and prostaglandin were the methods of induction among the women not in labour and they were 8 (20.0%), 16 (40.0%) and 16 (40.0%) respectively. Mode of delivery of highest number of respondents was caesarian section (54.0%) followed by normal vaginal delivery (39.7%). Other modes of delivery were ventouse and forceps and they were 07(05.0%) and 2 (1.4%) respectively. Out of 75 respondents under gone caesarian section, indication of C/S was fetal distress in 1st stage of labour, prolong 1st stage with maternal distress, failed induction, cephalopelvic disproportion (CPD) and breech presentation with big baby were 32.0%, 25.3%, 24.0%, 16.0% and 2.7% respectively. Maternal morbidity PPH, UTI, puerperal sepsis and wound infection were 10.0%, 14.40%, 3.60% and 5.70% respectively.

Table-I

Characteristics of the respondents in the study group.

Characteristics	Frequency	Percent
Age (years)		
18-29	114	82.01
≥30	025	17.99
Duration of pregnancy (weeks)		
41	80	57.6
42	39	28.0
43	17	12.2
44	03	02.2
Gravida		
Primi gravida	67	48.20
Multi gravida	72	51.80
Amount of liqour amnii (Clinical)		
Adequate	99	71.20
Scanty	40	28.80
Amount of liqour amnii (USG) (n=91)		
Adequate	40	56.3
Inadequate	15	21.1
Scanty	16	22.6
Stages of labour		
1st stage	92	66.2
2nd stage	07	05.0
Not in labour	40	28.8
Condition of cervix among the women not in labour (n=40)		
Favourable	24	60.0
Not favourable	16	40.0
Methods of induction among the women not in labour (n=40)		
Sweeping	08	20.0
Sweeping + ARM	16	40.0
Prostaglandin	16	40.0

Table-II

Distribution of mode of delivery among the study group.

	Frequency	Percentage
Mode of delivery among cases (n=139)		
NVD	55	39.7
Ventouse	07	05.0
Forceps	02	01.4
C/S	75	54.0
Indications of cesarean section (n=75)		
Fetal distress in 1st stage of labour	24	32.0
Prolong 1st stage with maternal distress	19	25.3
Failed induction	18	24.0
Cephalopelvic disproportion (CPD)	12	16.0
Breech presentation with big baby	02	02.7

Table-III

Distribution of maternal morbidity among the study group.

	Frequency	Percentage
Maternal morbidity (n=139)		
PPH	14	10.0
UTI	20	14.4
Puerperal sepsis	05	03.6
Wound infection	08	05.7

Discussion:

Complications of pregnancy and childbirth are the leading cause of premature death among women in developing countries[10]. Prolonged pregnancies are associated with both fetal and maternal complications. A variety of management practices can be utilized to mitigate the risk of these complications. Prolonged pregnancies, although less common in the era of ultrasound dating, are associated with fetal and maternal risks[18].

In the present study total 139 respondents with prolonged pregnancy were included. Among them 114 (82.01%) were in the age group of 18 to 29 years and 25 (17.99%) were in the age group of e”30 years. Duration of pregnancy of 80 (57.6%) was 41 weeks, 39 (28.0%) was 42 weeks, 17 (12.2%) was 43 weeks and rest 3 (2.2%) was 44 weeks. Among the respondents 67 (48.2%) were primi gravida and 72 (51.8%) were multi gravida. Clinically ninety nine (71.2%) respondents presented with adequate liqour amnii and 40 (28.8%) were presented with scanty liqour amnii. Ultrasonographically 40 (56.3%) respondents presented with adequate liqour amnii, 15 (21.1%) presented with inadequate liqour amnii and 16 (22.6%) were presented with scanty liqour amnii.

The first decision that must be made when managing an impending post term pregnancy is whether to deliver. In certain cases the decision is straightforward. However, frequently several options can be considered when determining a course of action in the low-risk pregnancy. The certainty of gestational age, cervical examination findings, estimated fetal weight, patient preference, and past obstetric history must all be considered when mapping a course of action[19]. Among the respondents 92 (66.2%) were in the 1st of labour, 7

(5.0%) were in the 2nd stage of labour and rest 40 (28.8%) were not in labour. Among the respondents not in labour, twenty four (60.0%) had favourable cervix and 16 (40.0%) had unfavourable cervix.

Women undergoing labor induction because of prolonged pregnancy should be sufficiently informed regarding the risks of a cesarean section or a vacuum extraction[20]. Sweeping, sweeping + ARM and prostaglandin were the methods of induction among the women not in labour and they were 8 (20.0%), 16 (40.0%) and 16 (40.0%) respectively. **Abotalib et al.[21]** in a study showed that labor induction and operative delivery rates were significantly higher in prolonged pregnancies. However, there were no significant differences between the patients who were induced and those who had spontaneous labor among the prolonged pregnancies.

Prolonged pregnancy is a common indication for planned delivery[17]. In the present study the mode of delivery of highest number of respondents was caesarian section (54.0%) followed by normal vaginal delivery (39.7%). Other modes of delivery were ventouse and forceps and they were 07(05.0%) and 2(1.4%) respectively. Out of 75 respondents under gone caesarian section, indication of C/S was fetal distress in 1st stage of labour, prolong 1st stage with maternal distress, failed induction, cephalopelvic disproportion (CPD) and breech presentation with big baby were 32.0%, 25.3%, 24.0%, 16.0% and 2.7% respectively. Abotalib et al.[21] in a study showed that operative delivery rates were significantly higher in prolonged pregnancies. Studies by Hannah et al.[22], Knox et al.[23] and Sanchez-Ramos et al.[24] showed that elective induction of labor not only is rate of cesarean delivery not increased in women who were randomized to routine induction of labor, but also more cesarean deliveries were performed in the noninduction groups, and the most frequent indication was fetal distress[19]. Risk factors intrinsic to the patient, rather than labor induction itself, are the cause of excess cesarean deliveries in women with prolonged pregnancies[25].

The maternal risks of post term pregnancy are often underappreciated[19]. In the present study maternal morbidity such as PPH, UTI, puerperal sepsis and wound infection were 10.0%, 14.40%, 3.60% and 5.70% respectively. In studies by Rand et al.[26], Campbell et al.[27], Alexander et al.[28], and Treger et al.[29] showed an increase in labor dystocia (9-12% vs 2-7% at term), an increase in severe perineal injury (3rd and 4th degree perineal lacerations) related to macrosomia (3.3% vs 2.6% at term) and operative vaginal delivery, and a doubling in the rate of cesarean delivery (14% vs 7% at term). Increased rate of cesarean delivery is associated with higher risks of complications such as endometritis, hemorrhage, and thromboembolic disease[28, 30].

Conclusion:

Rate of caesarean section in post-dated pregnancy was more than fifty percent, which is higher than the rate in term pregnancy. In the future we need to develop more effective strategies to reduce perinatal morbidity at term by selective delivery based on overall risk, not on gestational age alone.

Limitation of the study:

Because of illiteracy and lack of awareness of the patient, inaccurate LMP was a problem. There is no facility for emergency ultra sonogram in this hospital.

References:

1. Villar J and Bergsjo P. Scientific basis for the content of routine antenatal care. I. Philosophy, recent studies & power to eliminate or alleviate adverse maternal outcomes. ActaObstetricia et Gynecologica Scandinavica 1997; 76(1): 1-14.
2. World Health Organization (WHO). Mother-baby package: a safe motherhood planning guide. Geneva: World Health Organization, 1994 [Draft 3].
3. International Conference on Population and Development, Cairo. Programme for Action. Cairo: United Nations, 1994.
4. World Health Organization (WHO). Maternal mortality in 1995: Estimates developed by WHO, UNICEF & UNFPA. 2001, Ref.WHO/RHR/01.9.
5. Sundari TK. Can health education improve pregnancy outcome? Report of a grassroots action education campaign. The Journal of Family Welfare 1993. 39(1):1-12.
6. Prata N, Passano P, Sreenivas A, Gerdts CE. Maternal mortality in developing countries: challenges in scaling-up priority interventions. Women's Health 2010;6(2):311-327.
7. Berg CJ. Prenatal care in developing counties: The World Health Organization technical working group on antenatal care. Journal of the American Medical Women's Association 1995;50(5): 182-186.
8. Carroli G, Villar J, Piaggio G, Khan-Neelofur D, Gulmezoglu M, Mugford M, Lumbiganon P, Farnot U, Bersgjo P. WHO Antenatal Care Trial Research Group. WHO systematic review

of randomised controlled trials of routine antenatal care. Lancet 2001; 357(9268): 1565-1570.

9. Islam MT, Hossain MM, Islam MA, Haque YA. Improvement of coverage and utilization of EmOC services in southwestern Bangladesh. International Journal of Gynecology and Obstetrics 2005; 91: 298-305.

10. Maine D, Akalin MZ, Chakraborty J, de Francisco A, Strong M. Why Did Maternal Mortality Decline in Matlab? Studies in Family Planning 1996; 27(4):179-187.

11. Norwitz ER. Snegovskikh VV, Caughey AB. Prolonged Pregnancy: When Should We Intervene? Clinical Obstetrics & Gynecology 2007; 50(2): 547-557.

12. Delaney M, Roggensack A, Leduc DC, Ballermann C, Biringer A, Dontigny L. Guidelines for the Management of Pregnancy at 41+0 to 42+0 Weeks. J Obstet Gynaecol Can 2008; 30(9): 800–810.

13. Luckas MJM and Walkinshaw SA. Prolonged pregnancy. The Ohstetriciarz G Gynaecologist 2000;2(1): 39-41.

14. Olesen AW, Basso O, Olsen J. Risk of recurrence of prolonged pregnancy. *BMJ* 2003; 326*:* 476.

15. Olesen AW. Prolonged pregnancy: methods, causal determinants and outcome. Dan Med Bull 2004; 51:148.

16. Siozos C and Stanley KP. Prolonged pregnancy. Current Obstetrics and Gynaecology 2005; 15 (2): 73-79.

17. Bailey D. Management of prolonged pregnancy: Yesterday, today and tomorrow. O&G Magazine 2010; 12(2): 20-21.

18. Doherty L and Norwitz ER. Prolonged pregnancy: when should we intervene? Current Opinion in Obstetrics & Gynecology 2008; 20(6): 519-527.

19. Caughey AB and Butler JR. Postterm Pregnancy. Available from:www.emedicine.com, Last updated: 13/09/2010[Access 30/12/2010].

20. Bodner-Adler B, Bodner K, Pateisky N, Kimberger O, Chalubinski K, Mayerhofer K, Husslein P. Influence of labor induction on obstetric outcomes in patients with prolonged pregnancy: a comparison between elective labor induction and spontaneous onset of labor beyond term. Wien Klin Wochenschr 2005; 117(7-8): 287-92.

21. Abotalib ZM, Soltan MH, Chowdhury N, Adelusi B. Obstetric outcome in uncomplicated prolonged pregnancy. International Journal of Gynecology & Obstetrics1996; 55(3): 225-230.

22. Hannah ME, Hannah WJ, Hellmann J, Hewson S, Milner R, Willan A, and the Canadian Multicenter Post-term Pregnancy Trial Group. Induction of Labor as Compared with Serial Antenatal Monitoring in Post-Term Pregnancy - A Randomized Controlled Trial. N Engl J Med 1992; 326:1587-1592.

23. Knox GE, Huddleston JF, Flowers CE Jr. Management of prolonged pregnancy: results of a prospective randomized trial. Am J Obstet Gynecol 1979; 134(4): 376-84.

24. Sanchez-Ramos L, Olivier F, Delke I. Labor induction versus expectant management for postterm pregnancies: a systematic review with meta-analysis. Obstet Gynecol 2003; 101(6): 1312-1318.

25. Alexander JM, MCIntire DD, Leveno KJ. Prolonged pregnancy: induction of labor and cesarean births. Obstet Gynecol. 2001; 97(6): 911-915.

26. Rand L, Robinson JN, Economy KE, et al. Post-term induction of labor revisited. Obstet Gynecol 2000; 96(5 Pt 1): 779-783.

27. Campbell MK, Ostbye T, Irgens LM. Post-term birth: risk factors and outcomes in a 10-year cohort of Norwegian births. Obstet Gynecol. 1997;89(4):543-548.

28. Alexander JM, McIntire DD, Leveno KJ. Forty weeks and beyond: pregnancy outcomes by week of gestation. Obstet Gynecol 2000;96(2):291-294.

29. Treger M, Hallak M, Silberstein T, et al. Post-term pregnancy: should induction of labor be considered before 42 weeks? J Matern Fetal Neonatal Med 2002; 11(1):50-53.

30. Eden RD, Seifert LS, Winegar A. Perinatal characteristics of uncomplicated postdate pregnancies. Obstet Gynecol 1987; 69(3 Pt 1): 296-299.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Prevalence and Predictor of Nonalcoholic Steatohepatitis (NASH) in Nonalcoholic Fatty Liver Disease (NAFLD)

S ALAM[a], M ALAM[b], SMNE ALAM[c], ZR CHOWDHURY[d], J KABIR[e]

Summary:

Fatty liver is a common cause of chronic liver disease in developed as well as developing countries.We have designed this study to estimate the prevalence and predictors for non alcoholic steatohepatitis (NASH) in non alcoholic fatty liver disease (NAFLD). We have included 493 patients with sonographic evidence of fatty change in liver and 177 of them had done liver biopsy for histopathological study. Other causes of liver disease and alcohol consumption were excluded. Metabolic syndrome and biochemical and anthropometric evaluation was done. Females were predominating 250 (57.0 %). Centrally obese 422 (96.2 %) was more than over all obesity330 (75.1%). NASH was absent in 10 (5.6%) cases and diagnostic of NASH was 75 (42.4 %).Presence of diabetes could significantly (p = 0.001) predicted NASH. Age, sex, BMI, waist circumference, Serum HDL,triglyceride, insulin resistance index, hypertension, metabolic syndrome could not predict NASH. Serum GGT level was significantly (p = 0.05) higher in NASHwith a sensitivity of 45 % and specificity of 68 % only. Serum ALT and AST level could not detect NASH. Females were predominant sufferer of NAFLD in Bangladesh. Prevalence of NASH was much higher42.4%. Diabetes was the main predictor of NASH. GGT was the only biochemical indicator of NASH. We recommend liver biopsy in NAFLD with diabetes and raised GGT.

(J Banagladesh Coll Phys Surg 2014; 32: 71-77)

Introduction:

Nonalcoholic fatty liver disease (NAFLD) is a clinico-histopathological entity with histological features that resemble alcohol-induced liver injury. By definition, occurs in patients with little or no history of alcohol consumption[1]. NAFLD is the most common liver disease in western countries, affecting 20-30% of the general population[2,3]. It encompasses a histological spectrum that ranges from fat accumulation in hepatocytes without concomitant inflammation or fibrosis (simple hepatic steatosis) to hepatic steatosis with a necro-inflammatory component (steatohepatitis) that may or may not have associated fibrosis. The latter condition, referred to as nonalcoholic steatohepatitis (NASH), may progress to cirrhosis in up to 20% of patients[4]. Reports have also suggested that the prevalence of NAFLD among Asian Indians is comparable to that seen in the West[5, 6]. Average age for NASH patients is 40-50 years and for NASH-related cirrhosis it is 50-60 years. NASH probably causes around 80% of cases of cryptogenic cirrhosis which accounts for 10-20% of all cirrhosis and progresses to advanced fibrosis in 32 to 37% of patients[7].

In parallel with the epidemic of obesity and metabolic syndrome worldwide, the prevalence of NAFLD in Asian countries has increased rapidly with a trend to younger patients during the last two decades. The prevalence of NAFLD was about 15% in adults in Shanghai and Hong Kong[8]. NAFLD has been associated with insulin resistance and hyperinsulinaemia, even in lean subjects with normal glucose tolerance [9]. Diabetes mellitus may be an independent predictor of NASH, including cirrhosis and hepatocellular carcinoma[10]. NAFLD is now recognized as the hepatic component of the metabolic syndrome, which includes hyperlipidemia, glucose intolerance, obesity, and systemic hypertension. Predictors of NASH increase with the number of components of the metabolic syndrome[11].The contrasting clinical course of NASH versus non NASH fatty liver (NNFL) indicates that these

a. Dr. Shahinul Alam, Associate Professor, Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Postal Code-1000.Bangladesh.

b. Dr. Mahabubul Alam, Medical Officer. Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh.

c. Dr. Sheikh Mohammad Noor-E-Alam, Resident, Department of Hepatology, BangabandhuSheikhMujibMedicalUniversity, Shahbag, Dhaka, Bangladesh.

d. Dr. ZiaurRahmanChowdhury, Research Assistant, Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh.

e. Dr. Jahangir Kabir, Resident, Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh.

Address of Correspondence: Dr. Shahinul Alam, Associate Professor, Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Postal Code-1000.Bangladesh. email: shahinul67@yahoo.com

Received: 28 March, 2013 **Accepted:** 10 December, 2013

two conditions diverge early in the course of NAFLD although some patients probably transition from NNFL to NASH. Progression to cirrhosis is usually preceded by longstanding histological NASH and is infrequent in NNFL. Longitudinal studies with serial biopsies have shown that about one-third of NASH patients develop advanced fibrosis (stage 3 or 4 fibrosis) over 5-10 years from the time of the initial diagnosis[12]. Although usually relatively slow, progression to cirrhosis can occur in as little as 2–3 years. NASH is a common cause of 'cryptogenic' cirrhosis, which accounts for 10 – 20% of all cirrhosis[13]. Among patients diagnosed with NASH–related cirrhosis, the risk of developing a major complication of portal hypertension is 17, 23 and 52% at 1, 3 and 10 years, respectively. Among patients with early stage NASH, overall mortality over 10–15 years is about 10–12%, being significantly higher in NASH versus NNFL, compared to the general population. The risk of developing decompensated cirrhosis is 5–10% and for hepatocellular cancer it is 1–2%. There is a tenfold risk of cirrhosis relative to the general population[14].

A complete diagnosis of fatty liver disease ideally should define the histology, including the stage and grade of the disease as well as its etiology. In Bangladesh NAFLD is never been or insufficiently addressed in the field of medical research and practice. NASH is a potentially dangerous condition which requires medical intervention. The prevalence of NASH and potential risk factors for it is not yet explored here. We have designed this study protocol to estimate the prevalence of NASH in NAFLD and predictor of NASH in the perspective of Bangladeshwhich will be helpful future scientific knowledge and intervention.

Materials and Methods:

Study population:

We have included initially 439 patients at outpatient department of Hepatology in the University Hospital during the period of March 2010- December 2012 for fatty filtration in liver with ultrasonography. Exclusion criteria consisted of significant alcohol abuse (< 20g daily), evidence of hepatitis B and C and of drug induced fatty liver and other specific liver diseases: Hemochromatosis, Wilson's disease or autoimmune liver disease. These patients underwent clinical evaluation, anthropometric measurements, and blood tests. Liver biopsy was done after randomization in 190 patients but 4 biopsy samples were inadequate to comment for histopathology 4 patients withdrawn themselves from the study. The study was approved by the Institutional Review Board and all individuals provided written informed consent prior to enrollment in the study. Metabolic syndrome was defined according to Asian criteria,[15] and three of the five listed criteria were considered: waist circumference (WC) e"80 cm for women and e"90 cm for men, serum triglyceride ≥150 mg /dl (1.7 mmol/l) , serum HDL cholesterol <50 mg/dl (1.3 mmol/l) for women and <40 mg/dl (1 mmol/l) for men, elevated blood pressure (systolic blood pressure ≥130 and or diastolic blood pressure ≥85 mmHg or drug treatment for hypertension) and plasma glucose concentration e" 100 mg/dl (5.6 mmol/l) or drug treatment for diabetes.

Clinical and Biochemical evaluation:

All the patients were clinically evaluated: Blood pressure, Body mass index (BMI) and waist circumference was recorded for every patient. Liver function tests were performed prior to the liver biopsy. Blood samples were obtained under fasting conditions and the following tests were performed using standard laboratory methods: alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase, Gamma glutamyltranspeptidase (GGT) international normalized ratio (INR), blood glucose fasting and 2 hours after breakfast, lipid profile, Insulin level was assessed using the method of indirect chemiluminescence (MEIA). Insulin resistance was calculated according to the HOMA index (Homeostatic Metabolic Assessment).

Histological assessment

Liver biopsy specimens of 182 were analyzed by pathologist blinded to the patients' clinical and biochemical results. Histopathology was done in the department of Pathology BSMMU. The diagnosis of NASH was based on the Brunt et al criteria, [16]modified by Kleiner et al[17]. In this scoring system, the degree of disease activity in NAFLD was evaluated using the NAFLD Activity Score (NAS), which was calculated as the unweighted sum of the scores for steatosis (0–3), lobular inflammation (0–3), and hepatocyte ballooning (0–2) and thus ranged from 0 to 8. A NAS of 5 or more was diagnosed as "definitive NASH", NAS of 2 or

less as "non-NASH," and 3 or 4 as "borderline NASH." Other than NASH, was considered as NNFL. Hepatic fibrosis staging was as follows: 0 = no fibrosis; 1 = zone 3 fibrosis only; 2 = zone 3 and portal/periportal fibrosis; 3 = bridging fibrosis; and 4 = cirrhosis.

Statistical analysis

Results are presented as mean ± standard deviation (SD) for quantitative data and as numbers or percentages for categorical or qualitative data. Statistical differences in quantitative data were determined using t test or one way Anova test. Qualitative data were compared using the x^2 test. Multivariate regression analysis was done to explore the strongest predictor of NASH including the variables with significance in univariate analysis. For all tests, significance was achieved at $p < 0.05$.

Results:

Patient Characteristics:

Total of 439 patients wereincluded in this study. Females were 250 (57.0 %) and males were 189 (43.0 %). Mean age of the sample was 40.8 ± 10.2 years. Most of the population was house wife 217 (50.3 %), others were service holder 84 (19.5 %), business man 69 (16.0 %) and students were 59 (13.7 %). Hypertension and diabetes were prevailing in 83 (18.8 %) and 74 (16.8 %) respectively but metabolic syndrome was 188 (42.9 %). Triglyceride was high in 320 (72.8 %). BMI was normal in 51 (11.7 %), over weight 58 (13.2 %), Obese I 237 (53.9 %) and obese II 93 (21.2 %) according to criteria for Asian[18]. Most of the patients were centrally obese 422 (96.2 %) having waist circumference above normal. ALT, AST and GGT level were 54.1 ± 54.4, 45.1± 51.8 and 46.6 ± 33.7 u/l respectively. Insulin resistance index were higher than normal in 218 (49.6 %).

Histological Changes: Histopathological reports of 182 patients were available but 5 of them did not have fatty change on microscopy. We have included 177 patients for further analysis. There was no significant difference between biopsied and non- biopsied patient regarding clinical, anthropometric and biochemical variables. Steatosis of < 33% was 73(41.2%), 33 – 66 % was 82 (46.4 %) and > 66 % was 22 (12.4%). Lobular inflammation was absent in 10 (5.6 %), mild in 93 (52.5 %), moderate in 70 (39.5 %) and severe in 4 (2.3 %). Ballooning was absent in 5 (2.8 %), few ballooning in 138 (78.0 %) and prominent ballooning in 34 (19.2%) (Figure I). No fibrosis was seen in 28 (15.8%), stage I in 94 (53.3%), stage II in 40 (22.5 %) and stage III in 15 (8.3%). None had stage IV fibrosis(Table I).

According to NAS scoring system NASH was absent in 10 (5.6%) cases, borderline NASH was 92 (52.6%) and diagnostic of NASH was 75 (42.4 %). So NNFL was 102 (57.6%) and NASH was 75 (42.4%).

Predictors of NASH:

Prevalence of NASH in NAFLD was 75 (42.4%). There were no significant difference of age, BMI, waist circumference, Serum HDL and triglyceride level, insulin resistance index,sex, hypertension, metabolic syndrome did not differed in NASH and Non NASH. Mean age, BMI and waist circumference was similar in NNFL and NASH patients. Mean triglyceride was higher in NASH and mean HDL was lower in NASH but could not establish statistically significant value. Presence of diabetes could significantly ($p = 0.001$) differentiate NASH from NNFL. Serum ALT and AST level could not detect NASH in NAFLD. But serum GGT level was significantly ($p = 0.05$) higher in NASH than that of NNFL (Table II). GGT level for NASH was (51.7 ± 32.8) U/L and for NNFL was (40.4 ± 22.6) U/L. Multivariate regression analysis also explore that presence of diabetes could influence the development of NASH ($p= 0.04$) and GGT could differentiate NASH from NNFL ($p= 0.01$) (table III). But area under the curve is 59.3 % for GGT to differentiate NASH, with a sensitivity of 45 % and specificity of 68 % only for 44.5 U/L (Figure II).

Table-I

Histopathological features of biopsied patients

Variable	Number	Percent
Lobular inflammation		
Absent	10	5.6
Mild	93	52.5
Moderate	70	39.5
Severe	4	2.3
Ballooning		
Absent	5	2.8
Few	138	78.0
Prominent	34	19.2
Fibrosis		
Absent	28	15.8
Stage I	94	53.3
Stage II	40	22.5
Stage III	15	8.3
NASH	75	42.4

Table-II

Clinical, anthropometric and biochemical differences of NNFL and NASH

Variable	NNFL N=102	NASH N=75	*P*value
Age (yr)Mean ± SD	39.3 ± 9.4	41.0 ± 9.7	0.24
Sex: Male/ female	42/60	31/44	1.00
Body Mass Index (Kg/m^2)	27.8 ± 3.9	27.8 ± 4.6	0.998
Waistcircumference in cm Male	93.0 ± 5.5	93.0 ± 9.8	0.081
Female	95.8 ± 9.9	95.6 ± 11.0	0.927
HDL in mg/dl Male	36.3 ± 8.9	34.2 ± 6.5	0.337
Female	39.8 ± 10.3	39.2 ± 10.3	0.801
Serum Triglyceride mg/dl	225.2 ± 165.8	239.8 ± 111.6	0.509
Insulin Resistance Index	1.8 ± 1.3	1.5 ± 0.7	0.337
Diabetes Present / Absent	13/86	25/48	0.001
Hypertension Present / Absent	17/65	17/48	0.555
Metabolic SyndromePresent/ Absent	41/41	39/32	0.328
ALT U/L	56.9 ± 38.8	56.3 ± 31.8	0.603
AST U/L	46.9 ± 63.7	46.1 ± 22.2	0.916
GGT U/L	40.4 ± 22.6	51.7 ± 32.8	0.05

NASH; Non alcoholic steatohepatitis, NNFL; Non nash fatty liver

Table-III

Multivariate regression analysis for variable detecting NASH

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig
	B	Std. Error	Beta		
(Constant)	1.247	.517		2.411	.018
BMI	.014	.018	.124	.780	.438
Diabetes	.260	.125	.227	2.084	.040
Serum Triglyceride	.000	.000	-.105	-.919	.361
GGT	.005	.002	.289	2.473	.015
Waist Circumference	-.004	.008	-.077	-.491	.624

a. Dependent Variable: nash and nnfl
NASH; Non alcoholic steatohepatitis, NNFL; Non nash fatty liver

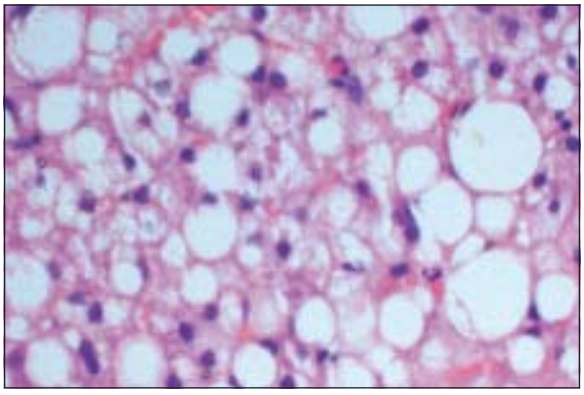


Fig.-1: *Microscopic feature of Nonalcoholic steatohepatitis:steatosis and ballooning degeneration.*

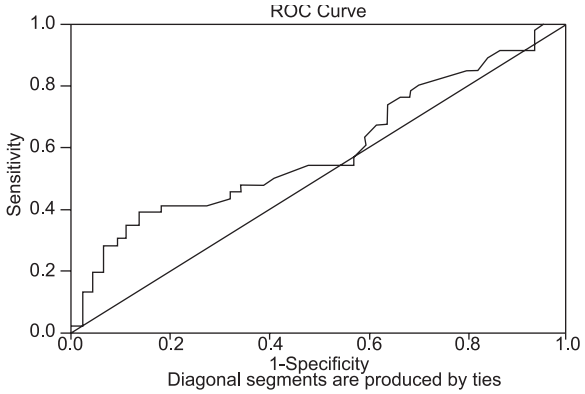


Fig.-2: *Receiver Operating Characteristic curve for GGT to differentiate NASH from NNFL.*

Discussion:

This study is the largest series from Bangladesh on NAFLD. Report of biopsy proven NASH and NNFL is also rare. University hospital is a tertiary care "center of excellence" hospital only and patients are referred from whole over the country. So this study may be the representative of prevalence of NASH in NAFLD of the country. Population based prevalence of NAFLD was not yet done in Bangladesh. Most of our NAFLD patients are of 30 to 50 years; this is similar to several reports from Asia[6,19-20]. But age could not influence the development of NASH. Female preponderance in NAFLD is dissimilar from reports from developed counties. Many recent studies have reported that male gender is a risk factor for fatty liver disease[21]. For example, in a study of 26,527 subjects undergoing medical checkups; the prevalence of NAFLD was 31% in men and 16% in women[22]. This female preponderance 250 (57.0 %) in our study may be the social conservative attitude which bounded most of our ladies to stay home for house hold activities without job leading to sedentary life style. Similar female preponderance was observed in one population studies from India[23]. But in accordance with previous studies sex did not influenced the development of NASH in NAFLD[22].

Centrally obese was 422 (96.2 %) outnumbered the overall obesity 330 (75.1%). The prevalence of NAFLD was increased according to the increase of BMI or abdominal circumference reported from Japan[24]. But other report concluded that waist circumference is an independent predictor of advance histological changes in NAFLD than BMI[25, 26]. But waist circumference was similar in NASH and NNFL in our series. It could be explained by that waist circumference indicate visceral obesity but no influence on pathogenesis of NASH at the stage of 2nd hit. Hypertriglyceridemia was very common 320 (72.8 %) in this study with no difference between NASH and NNFL. TG was long been considered as major factor in the development of NAFLD,[5-8] but there is mounting evidence that such non-TG lipid molecules are implicated in the pathogenesis of NASH by the process of lipotoxicity. Conversely, formation of TG may actually be a cytoprotective mechanism in liver[27, 28]. Our study revealed similar role of TG in NAFLD.

Our study explored that prevalence of NASH was 75 (42.4%) in NAFLD which is much higher. It is alarming for the country like Bangladesh. It was neither addressed previously nor considered anyway. In previous review, NAFLD wasfoundhighly prevalent (15% to 45%) in modern societies, only 10% to 25% of cases develop NASH, hepatic fibrosis leading to cirrhosis, end-stage liver disease or hepatocellular carcinoma[29].In other studies prevalence of NASH was 10 to 30 % in NAFLD [30] and it is less in Asian than that of European[31.32] . We were unbiased in selecting patient for liver biopsy and it was irrespective of clinical, biochemical and anthropometric status of the study population. So it is the representative of prevailing situation in the society. This finding warrants further extensive study on prevalence of NASH in Bangladesh and awareness of clinician is essential to diagnose NASH and to advice possible intervention as early as possible.

Presence of diabetes signified the presence of NASH in our study population (p= .001). Metabolic syndrome was prevailing in 188 (42.9%) population. NAFLD is strongly associated with insulin resistance (IR) and other components of the metabolic syndrome, like T2DM, central obesity, hyperlipidemia, and hypertension[33]. The pathogenesis of NASH appears to be a multiple hit process. The initial insult is the development of macrovesicular steatosis with the accumulation of hepatic fat from decreased hepatic free fatty acid oxidation and D or increased hepatic de novo lipogenesis, and D or decreased lipid export from the liver. Although IR can contribute to this dysregulation of lipid metabolism, once fatty liver develops, it can worsen hepatic IR and diabetes, contributing to a vicious cycle[34].

Serum ALT and AST levels were similar in NASH and NNFL in this study. But GGT were significantly (P= 0.05) higher in NASH than that of NNFL. NASH has been associated with slight elevation of liver enzymes mostly ALT[35]. In other reports NAFLD patient typically present with asymptomatic serum aminotransferase elevations of 2-3 times the normal[36] · This difference was due to different selection criteria. GGT is a sensitive indicator of liver damage[37]. Excess deposition of fat in the liver is associated with an elevated serum GGT[38]. Recent reports suggest that an increased GGT level is a risk factor for advanced fibrosis in NAFLD and, with weight loss, a decrease in GGT activity is predictive of improved lobular inflammation and fibrosis of liver[39].

The limitation of the study was that we had not done it at the community level rather at a tertiary level hospital of the country.

In conclusion, Females were predominant sufferer of NAFLD in Bangladesh. Prevalence of NASH was much higher in NAFLD. Diabetes was the main culprit in developing NASH in NAFLD. GGT was the only biochemical predictor of NASH but with low sensitivity and specificity. We recommend liver biopsy in NAFLD with diabetes and raised GGT.

References:

1 Ludwig J, Viggiano TR, McGill DB, Oh BJ.Nonalcoholic steatohepatitis: mayo Clinic experience with a hitherto unnamed disease. Mayo Clin Proc1980; 55: 434-38.

2. Browning JD, Szczepaniak LS, Dobbins R, Nuremberg P, Horton JD, Cohen JC, et al. Prevalence of hepatic steatosis in an urban population in the United States: impact of ethnicity. Hepatology 2004; 40: 1387-95

3. Angulo P. GI epidemiology: nonalcoholic fatty liver disease. Aliment PharmacolTher 2007; 25: 883-9.

4. Matteoni CA, Younossi ZM, GramlichT, Boparai N, Liu YC, McCullough AJ.Nonalcoholic fatty liver disease: a spectrum of clinical and pathological severity. Gastroenterology1999; 116:1413.

5. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al. For the Consensus Group. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. J Assoc Physicians India 2009; 57: 163-70.

6. Amarapurkar D, Kamani P, Patel N, Gupte P, Kumar P, Agal S, et al. Prevalence of non-alcoholic fatty liver disease: population based study. Ann Hepatol. 2007;6:161.

7. Grattagliano I, Portincasa P, Palmieri VO, Palasciano G.Managing nonalcoholic fatty liver disease: recommendations for family physicians. Can Fam Physician 2007; 53: 857-63.

8. Fan JG, Farrell GC. Epidemiology of non-alcoholic fatty liver disease in China. J Hepatol 2009; 50: 204-10.

9. Marchesini G, Bugianesi E, Forlani G, Cerrelli F, Lenzi M, Manini R, et al. Nonalcoholic fatty liver, steatohepatitis, and the metabolic syndrome. Hepatology 2003; 37: 917-923.

10. Angulo P, Keach JC, Batts KP, Lindor KD.Independent predictors of liver fibrosis in patients with nonalcoholic steatohepatitis. Hepatology 1999; 30:1356-62.

11. Marchesini G, Brizi M, Bianchi G, Tomassetti S, Bugianesi E, Lenzi M,et al. Nonalcoholic fatty liver disease: a feature of the metabolic syndrome. Diabetes2001; 50:1844-1850.

12. Argo CK, Northup PG, Al-Osaimi AM, Caldwell SH. Systematic review of risk factors for fibrosis progression in non-alcoholic steatohepatitis. J. Hepatol. 2009; 51:371 - 379.

13. Powell EE, Cooksley WG, Hanson R. The natural history of nonalcoholic steatohepatitis: a follow-up study of forty - two patients for up to 21 years. Hepatology 1990; 11: 74 - 80.

14. Hui JM, Kench JG,Chitturi S. Long-term outcomes of cirrhosis in nonalcoholic steatohepatitis compared with hepatitis C. Hepatology 2003; 38: 420-427.

15. Chitturi S, Farrell GC, Hashimoto E, Saibara T, Lau GK, Sollano JD. The Asia-Pacific Working Party on NAFLD. Non-alcoholic fatty liver disease in the Asia-Pacific region: Definitions and overview of proposed guidelines. J GastroenterolHepatol. 2007; 22:778-87.

16. Brunt EM, Janney CG, Di Bisceglie AM, Neuschwander-Tetri BA, Bacon BR. Nonalcoholic steatohepatitis: a proposal for grading and staging the histological lesions. Am J Gastroenterol 1999; 94:2467-74.

17. Kleiner DE, Brunt EM, Van Natta M, Behling C, Contos MJ, Cummings OW, et al. Design and validation of a histological scoring system for nonalcoholic fatty liver disease. Hepatology 2005; 41:1313-1321.

18. Annurad E, Shiwaku K, Nogi A, Kitajima K, Enkhmaa B, Shimono K , et al. The New BMI criteria for Asians by the Regional Office for the Western Pacific Region of WHO are suitable for the screening overweight to prevent metabolic syndrome in Elder Japanese Workers. J occup Health 2003; 45: 335-343.

19. Li H, Wang YJ, Tan K, Zeng L, Liu L, Liu FJ, et al. Prevalence and risk factors of fatty liver disease in Chengdu, Southwest China. HepatobiliaryPancreat Dis Int. 2009;8:377-82.

20. Park SH, Jeon WK, Kim SH, Kim HJ, Park DI, Cho YK, et al. Prevalence and risk factors of non-alcoholic fatty liver disease among Korean adults. J GastroenterolHepatol. 2006;21:138-43.

21. Vernon G, Baranova A, Younossi ZM. Systematic review: the epidemiology and natural history of non-alcoholic fatty liver disease and nonalcoholic steatohepatitis in adults. Aliment Pharmacol Ther 2011; 34: 274-85.

22. Adams LA, Lymp JF, St Sauver J, Sanderson SO, Lindor KD, Feldstein A, et al. The natural history of nonalcoholic fatty liver disease: a population-based cohort study. Gastroenterology. 2005;129: 113-21.

23. Mohan V, Farooq S, Deepa M, Ravikumar R, Pitchumoni CS. Prevalence of non-alcoholic fatty liver disease in urban south Indians in relation to different grades of glucose intolerance and metabolic syndrome. Diabetes Res ClinPract. 2009; 84:84-91.

24. Hamaguchi M, Takeda N, Kojima T, Ohbora A, Kato T, Sarui H, et al. Identification of individuals with non-alcoholic fatty liver disease by the diagnostic criteria for the metabolic syndrome. World J Gastroenterol 2012; 18:1508-1516.

25. Manco M, Bedogni G, Marcellini M, Devito R, Ciampalini P, Sartorelli MR, et al. Waist circumference correlates with liver fibrosis in children with nonalcoholic steatohepatitis. Gut. 2008;57: 1283-87.

26. Sato K,Dohke M, Mizutome N, Kimura R, Makuri A, Matsui A, et al. Evaluation of metabolic syndrome with respect to the waist circumference and visceral fat area. Ningen Dock. 2008;23:558-563.

27. Neuschwander-Tetri BA.Nontriglyceride hepatic lipotoxicity: the new paradigm for the pathogenesis of NASH. Curr Gastroenterol Rep 2010;12:49-56.

28. Neuschwander-Tetri BA. Hepatic lipotoxicity and the pathogenesis of nonalcoholic steatohepatitis: the central role of nontriglyceride fatty acid metabolites. Hepatology 2010;52:774-88.

29. Farrell GC, RooyenDv, Gan L , Chitturi S. NASH is an Inflammatory Disorder: Pathogenic, Prognostic and Therapeutic Implications. Gut Liver 2012;6:149-171.

30. McCullough AJ. The epidemiology and risk factors of NASH. In:FarrellGC,GeorgeJ,de la Hall P, et al., eds. Fatty Liver Disease: NASH and Related Disorders. Malden, MA: Blackwell Publishing; 2005:23-37.

31. Farrell GC, Larter CZ. Nonalcohlic fatty liver disease: form steatosis to cirrhosis. Hepatology 2006; 43: s99-s112.

32. Ratziu V, Poynard T. Assesing the outcome of nonalcohlic steatohepatitis? It's time to get serious. Hepatology 2006; 44:802-5.

33. Clark JM, Brancati FL, Diehl AM. The prevalence and etiology of elevated aminotransferase levels in the united states. Am J Gastroenterol 2003; 98: 960-67.

34. Edmison J, McCullough AJ. Pathogenesis of non-alcoholic steatohepatitis: human data. Clin Liver Dis 2007; 11: 75-104.

35. Bayard M, Holt J, Boroughs E. Nonalcoholic fatty liver disease. Am Fam Physician2006; 73: 1961-8.

36. Marceau P, Biron S, Hould FS, Marceau S, Simard S, Thung SN, et al. Liver pathology and the metabolic syndrome X in severe obesity. J ClinEndocrinolMetab 1999;. 84: 1513-7.

37. Lee DH, Blomhoff R, Jacobs DR Jr. Is serum gamma glutamyltransferase a marker of oxidative stress? Free Radic Res 2004; 38: 535-9.

38. Saito T, Nishise Y, Makino N, Haga H, Ishii R, Okumoto K, et al. Impact of metabolic syndrome on elevated serum alanine aminotransferase levels in the Japanese population. Metabolism 2009; 58: 1067-75.

39. Kim HC, NamC, Jee SH, Han KH, Oh DK, Suh I. Normal serum aminotransferase concentration and risk of mortality from liver diseases: prospective cohort study. BMJ 2004; 328: 983.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Efficacy of Temporalis Myofascial Flap in Maxillofacial Reconstruction

MS SHIKDER[a], MM HASAN[b], AA MASUD[c], IA HAIDER[d], MU AHMED[e]

Summary:

Temporalis myofascial flap holds great promise for the reconstruction of various defects of the maxillofacial region for its dependable blood supply, proximity to the maxillofacial region, possibility to mobilize it to the oral cavity and its fanned out nature.

The purpose of the study was to evaluate the efficacy of temporalis myofascial flap in maxillofacial reconstruction.This cross sectional study was carried out in the Department of Oral and Maxillofacial Surgery, Dhaka Dental College and Hospital from January 2011 to December 2011. Nineteen patients (10 male and 9 female), age ranging from 19 to 55 years with medium to large defect (>8cm)were selected for this study. After surgical resection of the pathological lesion, the TMF was exposed by a hemi-coronal incision with a preauricular extension. The muscle was rotated to oral cavity and sutured with defect margin. Post-operatively the patient were clinically evaluated at 1, 2, 3 weeks and 1 and 3 months. Complete flap take was observed in 84% cases where as partial flap take was seen in 16% cases. Mouth opening restriction were 2-3 mm in 10 cases and 5-8 mm in 5 cases.Temporalis myofascial flap is a useful, reliable and versatile option for reconstruction of moderate to large sized defects. This flap provide abundant tissue, with minimum to no functional morbidity or esthetic deformity in donor site.

Key words: TMF-temporalis myofascial flap.

(J Banagladesh Coll Phys Surg 2014; 32: 78-83)

Introduction:

Reconstruction should strive to restore the maxillofacial form, versatility of tissues, oral competence and oral cavity function and thus allowing the patients to meet the social needs.[1] Various defects in orofacial region due to tumor extirpation, trauma or congenital defect requires subsequent reconstruction. The choice of reconstructive technique depends upon numerous factors related to each individual case. These factors include the site, size and composition of the defect. A large variety of techniques are available for reconstruction of the defects. Each technique has its advantages and limitations. The simplest surgical procedure with good surgical, functional and esthetic results and having minimum morbidity should be adopted. Several soft tissue flaps can be used for the reconstruction of defect in the orofacial region. These include local, regional and free flaps. Local flap is enough to reconstruct the small defect but for moderate to large defect regional or free flaps are often necessary. Of the various regional flaps temporalis myofascial flap provides one of the best option because of its reliable vascularity, adequate bulk and proximity to the defects in orofacial region.[2] Since the viability of the flap is excellent, post operative radiotherapy can be taken up much earlier than any types of reconstruction.[3]

The apparent advantages of temporalis myofascial flap (TMF) is evident from other studies in different countries as it is easy in performing the technique, proximity of the flap to the oral cavity, minimal or no donor side morbidity and minimal post operative complication. But there is no systematic study to verify its potential and outcome in Bangladesh is available for reconstruction of the defects. In a study John F arvier showed that for the repair of the adult cleft palate the transantral transfer of TMF is a single stage procedure with low morbidity and few complication.[4] The present study was designed to find out the efficacy of temporalis myofascial flap for reconstruction of various defects of maxillofacial region after tumour surgery in the department of Oral and Maxillofacial Surgery (OMS), Dhaka Dental College & Hospital (DDCH).

a. Dr. Muhammad Sohag Shikder, Maxillofacial Surgeon.

b. Dr. Md. Mokerrom Hasan, Lecturer, Dept. of Oral and Maxillofacial Surgery, DDCH.

c. Dr. Abdullah Al Masud, Assistant Professor, Dept. of Oral and Maxillofacial Surgery, DDCH.

d. Dr. Ismat Ara Haider, Associate Professor, Dept. of Oral and Maxillofacial Surgery, DDCH.

e. Professor Dr. Mohiuddin Ahmed, Professor and Head, Dept. of Oral and Maxillofacial Surgery, DDCH.

Address of the correspondent: Dr. Md. Sohag Shikder, House-DCC-95, 4th floor, Sharif Miar Bari, Poolpar, Ibrahimpur, Kachukhet Cantonment, Dhaka, E-mail shikderd36@yahoo.com Mob: 01716604751

Received: 11April, 2013 **Accepted:** 8 February, 2014

The aim of this study is to evaluate the effectiveness of temporalis myofascial flap in reconstruction purpose depending on size of the defect, length of the flap and operating time.

Material and Methods:

This Prospective type of cross-sectional study was carried out in the department of oral and maxillofacial surgery, Dhaka Dental College and Hospital from January 2011 to December 2011. Patient admitted in the Department of Oral and Maxillofacial Surgery, Dhaka Dental College and Hospital with orofacial tumor and deformity requiring ablative surgery were selected for this study. Surgical planning was done and the defect was categorized as medium to large defect (medium defect 4-8 cm, large defect >8cm). Total of 19 patients were selected including tumor and cleft palate defect.

A detailed medical history of the selected patient and thorough clinical examination was done. In all cases site and size of the lesion and probable defect was measured preoperatively. After surgical resection of the pathological lesion the defect to be reconstructed was measured. The TMF was exposed using a hemicoronal incision with a pre-auricular extension. Dissection was carried out at a subgaleal level. It was then mobilized subperiosteally from its deep origin in the temporal fossa. Anteriorly the muscle was elevated from lateral aspect of the orbit and inferiorly down to the temporal crest. The temporal fascia was then incised transversely 2 cm above the zygomatic arch so as not to injure the zygomatic branch of facial nerve. The zygomatic arch was exposed and divided as far forward and as far posteriorly as possible to allow the muscle to be rotated in to the oral cavity. The muscle was sutured to the defect margin of the oral cavity with the temporal fascia facing the oral cavity. The donor site was closed in layers over a suction drain. Post-operatively all the patients were clinically evaluated after 1st, 2nd, 3rd week, 1st and 3rd month. Post-operatively patient was evaluated on the basis of flap success (flap color, epithelialization, and adaptation), flap failure (Infection, wound dehiscence, sloughing and fistula), complication (mouth opening/trismus, facial deformity) and donor site morbidity (Infection, deformity). Per-operative evaluation was done with defect size, flap size and operating time.

In post-operative evaluation flap viability was assessed by pink color, pale color and blue with pink color. Epithelialization was assessed by homogenosity of the flap with adjacent mucosa.

In complication, mouth opening was measured by inter incisal opening with scale and facial deformity was judged comparing with opposite side of the face with visual examination.

Donor site morbidity was expressed by infection which indicated any sort of secretion from donor site and deformity was assessed with opposite temporal fossa. The data was collected in preformed questionnaire. The data was analyzed with statistical software (spss va 20) and was represented with table, diagram.

Result:

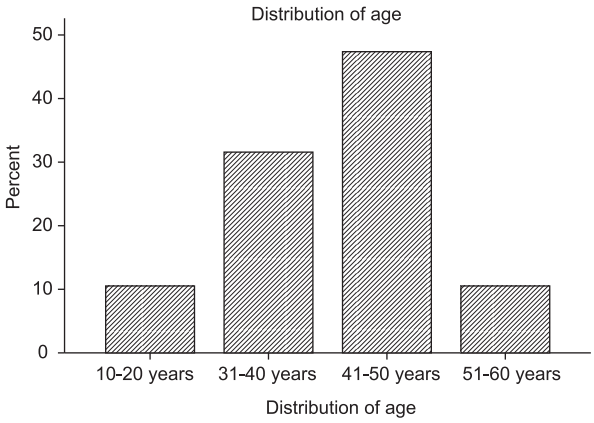


Fig-.1: *A bar diagram showing distribution of age. The age range of the study population was 10-60 years but the lowest age was 19 and the highest was 55 years. The highest percentage (47.4%) of the subjects was from the age group 41 to 50. There was no subject available for the age group 21-30 years.*

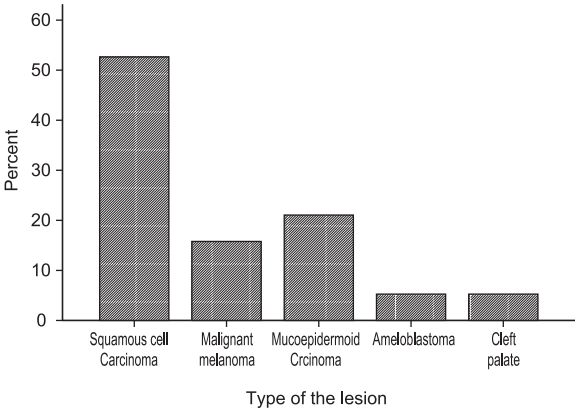


Fig-.2: *A bar diagram showing the type of lesion. Sq. cell carcinoma is more frequent (52.6%) among all other lesions. Then comes mucoepidermoid carcinoma and malignant melanoma accordingly. Ameloblastoma and cleft palate accounts the least percentage.*

Table-I

Post operative outcome of the subjects after 3 weeks and 3 months post operatively.

Postoperative outcome		Postoperative duration	
		After 3 weeks	After 3 months
Flap color	Pink	18 (94.7%)	19(100%)
	Pale-pink	1(5.3%)	00(0%)
Blue with pink	0(0%)	00(0%)	
Epithelialization	Yes	8(42.1)	19(100%)
	No	11(57.9)	00(0%)
Adaptation	Yes	17(89.5%)	19(100%)
	No	02(10.5%)	00(0%)
Post-op. infection	Yes	00(0%)	00(0%)
	No	19(100%)	19(100%)
Sloughing	Yes	11(57.9%)	00(0%)
	No	08(42.1%)	19(100%)
Wound dehiscence	Yes	00(0%)	00(0%)
	No	19(100%)	19(100%)
Fistula	Yes	01(5.3%)	00(0%)
	No	18(94.7%)	19(100%)
Facial deformity	Yes	03(15.8%)	00(0%)
	No	16(84.2%)	19(100%)
Donor site infection	Yes	00(0%)	00(0%)
	No	19(100%)	19(100%)
Donor site deformity	Yes	19(100%)	19(100%)
	No	00(0%)	00(0%)

Table-I: shows post operative outcome with different parameters after 3 weeks and after 3 months. All subjects show pink color flap, proper adaptation without any fistula but show donor site deformity in all cases after 3 months.

Table-II

One-Sample statistics

	N	Mean	Std. Deviation	Std. Error Mean
Preoperative mouth opening	19	32.1053	7.68038	1.76200
Postoperative mouth opening (after 3 months)	19	30.0000	4.83046	1.10818

Table-II Shows mean pre & post operative mouth opening with standard deviation. It showed that mean preoperative mouth opening was 32.10 mm and SD±7.68 where as mean post operative mouth opening was 30.00 mm and SD±4.83. that is mean difference was 2.1 mm.

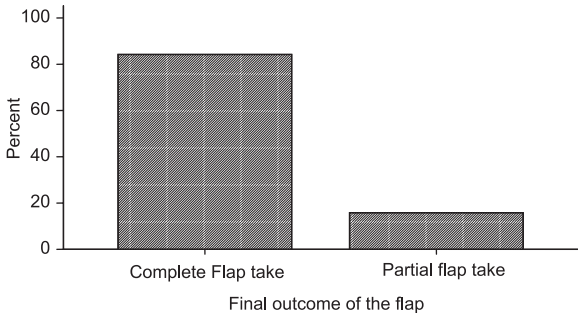
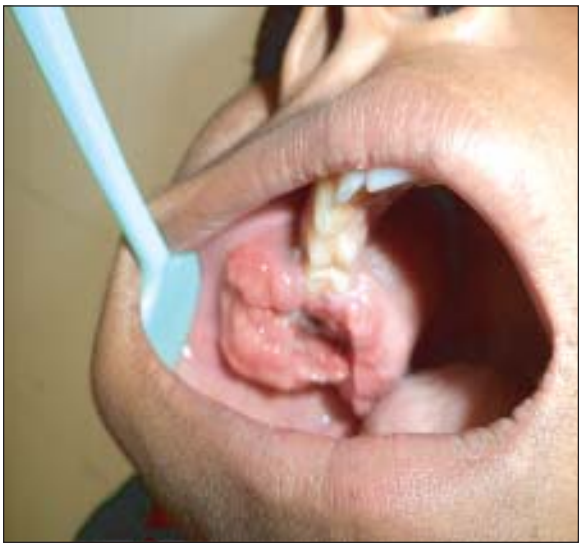
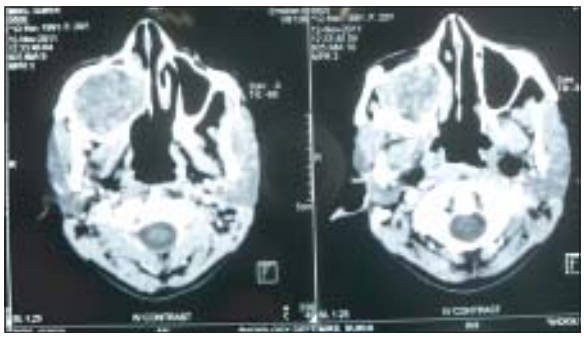


Fig.-3: *A bar diagram showing final outcome of the flap where complete take is observed in 84% cases and partial take was observed in 16% cases.*

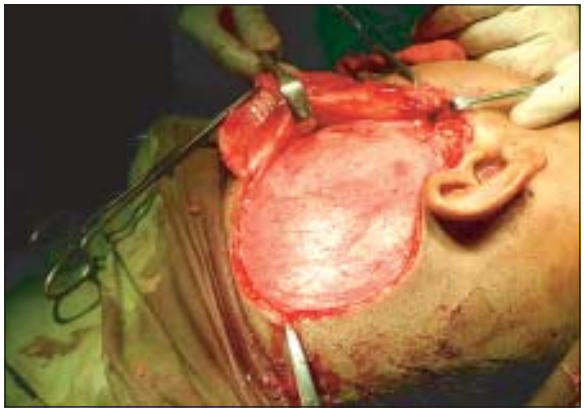
Illustration



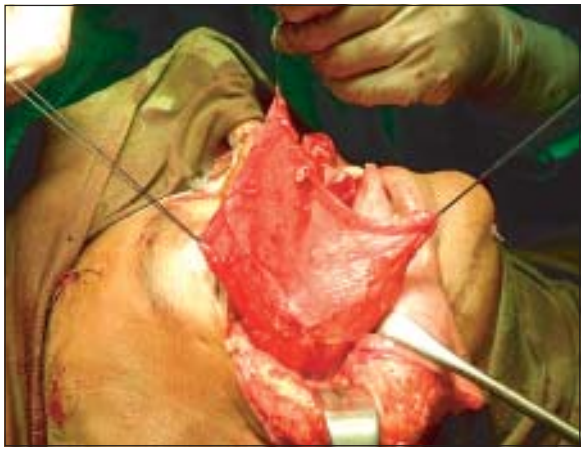
Pre operative: A case of Right maxillary ameloblastoma involving buccal and palatal aspect of right maxilla from right upper fist premolar to retromolar region.



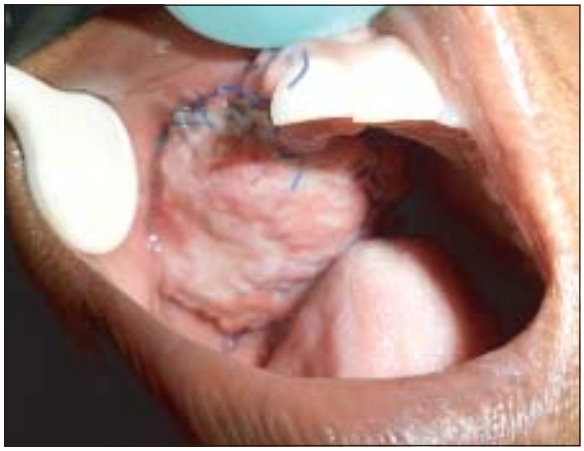
Pre operative: Axial section of Radiograph showed involvement of right maxilla with mixed density of right maxillary sinus but no erosion of buccal or posterior wall and lateral wall of the nose is seen



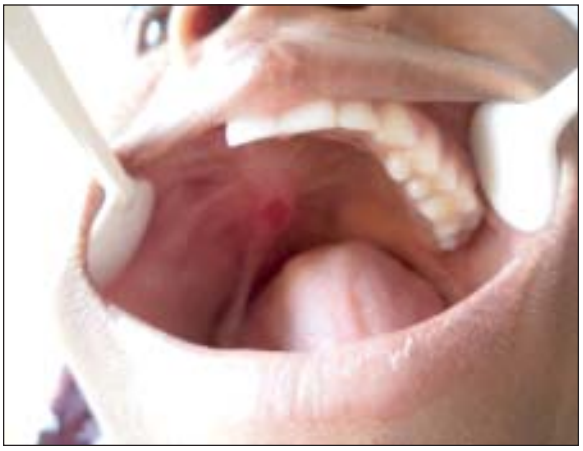
Per operative: Incision and exposure of TMF with hemicoronal incision with preauricular extension



Per operative: Transfer of TMF with in the oral cavity through the resection of zygomatic arch.



Post operative: After three weeks proliferative stage of TMF with sloughing of superficial fascia



Post operative: After three months complete healing of TMF with epithelialisation

Discussion:

Surgical treatment of malignancies in the maxillofacial region has improved with advances in reconstructive techniques with a thorough knowledge of the anatomy of the flaps and their clinical application. Many different reconstructive procedures are available for this purpose. Local flaps can be used successfully for small defects. However, for moderate to large defects free or regional flaps are often necessary to reconstruct the defects.[2] Microvascular free flap might be the best choice for reconstruction but difficult to perform due to limited facilities in the place of study (large patient que, logistics support) and expertise. Where as distant flaps are not always a good option for maxillofacial defects particularly for palate, check due to their long distance, excessive bulk and donor site morbidity.

The TMF is simple with short harvesting time, versatile, regional proximity, mobility of a thin tissue mass on a vascular pedicle and lacks functional morbidity at donor site with acceptable cosmesis which are significant advantages of the use of this flap.[5] This study used TMF for maxillofacial defect in 19 patients, age ranges from 19 to 55 years. Among them 41-50 years age groups(Fig.1) comprises the highest percentage (47.4%). Among the study subject 52.63% were male and 47.37% were female and male female ratio was 10:9. Estelles Freriol *et al.* (2005) used TMF in 22 patients where male were 14 and female were 8 with a age range between 37 to 73 years.[6]

Among 19 cases most of the cases (52.6%) were squamous cell carcinoma then comes mucoepidermoid

carcinoma ,malignant melanoma and ameloblastoma (Fig.2). Most involved site was palate particularly midpalate. In 38 cases of Hoyo *et al*. for TMF reconstruction, 23 cases were sq. cell carcinoma and most involving site was maxillary sinus.[7] The study result of Hoyo *et al.* showed that after surgical resection of maxillary carcinoma TMF might be the treatment of choice for reconstruction which supports the presented study.

TMF flap can be used for the reconstruction of mandibular soft tissue upto the canine region.[8] In the present study most of the site of the defect was in the midpalate and cheek.

Operating time is an important factor for both the patient and for the surgery out come. In a tertiary hospital like DDCH where patient flow is higher, operating time needed to be averaged to meet the high patient flow. In all of the study cases mean operating time was 126.31 minutes with a SD of 12.56 minutes. Distant flap and microvascular flap procedure would have been more elaborate and time consuming.

Viability of the flap was observed by color of the flap. A flap that had intact blood supply with good perfusion showed normal color that is pink while flap with poor perfusion showed pale color and flap with venous congestion showed bluish color.[1] In the present study after from 1st POD to 3 weeks 94.7% showed pink color which became 100% after 3 months. Ahmed *et al.* got 90% success rate of flap while 10% of failure due to diabetes.[1] Abu baker in 2002 also get 100% success rate with his study.[2]

Flap adaptation at the defect margin is an important criteria for flap success. In 3 weeks follow up 17 (89.5%) showed good adaptation at the flap margin but at 3 months follow up all the cases 19(100%) showed good adaptation. Regarding epithelialisation of the flap 42.1% of the cases showed surface epithelialisation within 3 weeks and 100% achieved surface epithelialisation in 3 months follow up. The epithelialization of the flap took place in around 3 weeks time in the study of Mani and Panda.[3] This result differs with the study of Mani and may be due to the patients condition. Sloughing of temporal fascia frequently occurred in the Hoyo study.[7] In our study, fascia sloughing occurred in all (100%) cases up to 3 weeks. Wound dehiscence is major complication of flap outcome. Dehiscence may result in flap failure. But in this study during follow up period wound dehiscence was nil. No dehiscence or fistula occurred in Hoyo study.[7] Hoyo study matches with the present.

Mouth opening frequently reduces after maxillectomy if done without reconstruction. Ahmed *et al*. showed post operative mouth opening restriction only in 2 out of 28 cases, but Naaj *et al*. got mouth opening limitation in most cases in their study.[1,9] Pre and postoperative mouth opening was studied and compared in the present study. It showed that mean preoperative mouth opening was 32.10 mm with SD of 7.68, where as mean post operative mouth opening was 30.00 mm with SD of 4.83. So, mean difference was 2.1 mm which was probably due to inadequate post operative jaw exercise.

Before harvesting any flap donor site morbidity must be kept in mind. All patients had donor site deformity which was also present in the study of Naaj *et al*. due to lack of tissue bulk that had been harvested.[9] Depression of the donor site was found in all of the (100%) cases upto 3 months follow-up period. But the deformity was masked in all cases by the hair line. This is an advantage of TMF comparing to others.

Flap success depends on all the factors described above. Temporalis muscle flap success rate was 80% with two minor complication that resolved without damage to the flap in a study.[9] Only one patient out of 26 had complete flap necrosis in Colmenero study.[10] In Hoyo study there was no partial or total flap necrosis in 38 cases[7]. In the present study complete flap take occurred in 84% of the cases after complete follow up and only 6% showed partial flap take and it was due to the marginal necrosis of the flap.

Conclusion:

Temporalis myofascial flap is an axial pattern flap, which is an excellent choice for oral and maxillofacial reconstruction, especially defects of palate and cheek. Temporalis myofascial flap is far less bulky, more pliable, non-hair bearing and in close proximity to the oral cavity. Use of this muscle flap does not impair mandibular function. The depression is minimal and the hair covers most of the donor area.

References:

1. Ahmad S, Warraich R A, Abid H. The versatility of Temporalis muscle flap in reconstruction of maxillofacial region. Annals 2009; 15: 1.

2. Abubaker A O, Mustafa B, Abouziga. The temporalis muscle flap in reconstruction of intra oral defects: An appraisal of technique. Oral surg oral Med oral Pathol oral Radiol Endod *2002;* 94: 24-30.

3. Mani V, Panda A K. Versatility of temporalis myofascial flap in maxillofacial reconstruction-analysis of 30 cases. Int J Oral Maxillofac Surg 2003; 32: 368-372.

4. John F. Arvier, Motiur R. Molla, S. M. Iqbal Shaheed, Kerry Lanza Barry Fitzpatrick Trans-antral temporalis transfer for the repair of adult cleft palates. Australian Dental Journal 1997;42:(5):307-14

5. Demas P N, Sotereanos G C. Transmaxillary temporalis transfer for reconstruction of a large palatal defect. J Oral Maxillofac Surg 1989; 47: 197-202.

6. Eestelles Ferriol J E, Liatas M C, Ferrer Ramirez M J. Temporalis myofascial flap: technique description and results in our patient. Acta otorrinolarissgol Esp 2005*;* 56: 257-260.

7. Dell hoyo J A, Sanroman J F, Gil-diez J L. The temporalis muscle flap. J Oral Maxillofac Surg 1994; 52: 143-147.

8. Bradley P, Brockbank J. The temporalis muscle flap in oral reconstruction. J Max-Fac Surg1981; 9: 139-145.

9. Naaj I A, Leiser Y, Liberman R, Peled M. The use of temporalis myofascial flap in oral cancer patient. J.oral maxillofac surg; 68: 578-583.

10. Colmenero C, MartorellA V, Colmenero B, SierraI I. Temporalis myofascial flap for maxillofacial reconstruction. J Oral Maxillofac Surg 1991; 49: 1067-1073.

Doctor Patient Communication: A Review

T BEGUM

Abstract:

Communication between patients and health professionals is seen as the core clinical function in building a therapeutic doctor-patient relationship, which is the heart and art of the medicine. Patients' satisfaction is strongly influenced by the quality of the communication that occurs. Effective communication is the basis of mutual understanding and trust. This paper aims to raise awareness on the important issues involved in doctor-patient and inter-professional communication among the medical professionals.

(J Banagladesh Coll Phys Surg 2014; 32: 84-88)

Introduction:

The word 'communicate' comes from the Latin, 'to impart, to share'.[1] It is the act by which information is shared between human. It is a mutual dialogue and through communication we relate and interact with other people.

The doctor-patient relationship is a complex one. The patient enters into this relationship usually in a distressed state and desires to be more comfortable, emotionally and spiritually relieved by the outcome of the interaction. The physician brings to the relationship a technical knowledge of organ systems and disease process, clinical experience, medical judgment and in most cases, empathy and understanding of the patients' needs and concerns.[2,3]

Good communication encourages meaningful and trusting relationships between health care professionals and their patients.[3] The ability to communicate competently with patients has been considered as a pre-condition of qualification for all health care professionals if they are to deliver patient care. [4,5]

According to Tattersall communication is a vital part of care.[6] A doctor's communication and inter-personal skills encompass the ability to gather information in order to facilitate accurate diagnosis, counsel appropriately, give therapeutic instructions and establish caring relationships with patients.[7,8] These are the core clinical skills in the practice of medicine, with the ultimate goal of achieving the best outcome and patient satisfaction.[9]

Address of Correspondence: Prof. Tahmina Begum, Professor & Head, Department of Paediatrics, BIRDEM, Dhaka, Mobile: 0171500888

Received: 18 November, 2013 **Accepted:** 11 March, 2014

There is increasing awareness among doctors, patients, researcher and educator that effective doctor patient communication is important in achieving desired health outcome.[9,10] As communication skills are important qualities of a doctor, it has been shown that patient satisfaction, compliance, recall and understanding of medical advice and health outcome are largely influenced by the quality of communication between patient and doctor.[9,11] Doctors need to learn essentials of good communication more than other professionals because patients are humans with sensitive needs.

Benefits of effective communication:

Communication is essential for all aspects of life. It is important not only to professional – patient interaction but also within the health care team.[4] The 3 main goals of doctor-patient communication are creating a good interpersonal relationship, facilitating exchange of information and including patients in decision making.[10,12,13]

Benefits of effective communication are.[3,8,12-15]

For patients:

- Patients' problem can be identified more accurately
- Helps patient to recall information and comply with treatment instruction. Thereby, providing patient satisfaction
- Increase patient understanding of treatment
- Increase compliance which leads to improved health and better outcomes
- Promote better emotional health for resolution of symptoms and pain control
- Improve quality of care by involving patient in decision making

For doctors:

- Improve doctor patient relationship. The doctor is better able to seek the relevant information and recognize the problems of the patients by way of interaction and attentive listening
- Good doctor-patient communication has the potential to help regulate patients' emotions and allow for better identification of patients' needs, perceptions and expectations
- Relieve doctors of some of the pressures of dealing with the difficult situations encountered in the emotionally demanding profession
- Enhance job satisfaction
- Reduce the incidence of clinical error

Expectations of patient:

'The patient must feel at all times that they are treated with respect'.[15] Common expectations patients have for their physicians are: [5,12,13-21]

Primary expectation-

- Clinical competence

Secondary expectations-

- Professional
- Respectful
- Polite
- Sincere
- Caring attitudes
- Effective communication skills- verbal and non verbal

Medical interview:

The medical interview is the usual communication encounter between the doctor and the patient. It can be classified according to the purpose of the interview into 4 types:

History taking

Consultations

Breaking bad news

Obtaining informed consent

Consultation:

Research over the past few decades has shown that most patients want to be informed about their illness. Patient's primary goal in seeking medical care is to obtain information about their condition or illness as well as treatment option and outcome.[5,6,16,22] Patient's satisfaction is strongly influenced by the quality of the communication that occurs. Compliance of the patients with treatment, recall and understanding of medical advice and health outcomes is significantly related to satisfaction with information provided by the doctors. It is therefore important to communicate information and provide explanation to patient and families and convey proper concern to those who seek help.[5,6,13-15] Studies also showed that patient's dissatisfaction can seriously reduce their compliance with their treatment regimen. It also triggers patient doubts about the competence of their physician.[14,15]

Complaints about doctors and health services are commonly originate because of bad attitude and behavior of doctors, poor communication or because patients expectations have not been met, or both.[3,18]

In medical consultation, patients are likely to retain only 50% of what a physician has told them. Furthermore, only about half of the information they received is remembered correctly. So, we can expect patients to recall correctly about 25% of what we have told them.[4,6] Doctors' way of counseling plays a major role in retention of information.[22]

It demands that communication be understandable to the patient, not only when it is delivered but also after the patient has left the hospital or doctors room. [6]

Breaking bad news: Breaking bad news is one of a physician's most difficult duties. Bad news is any news that seriously and negatively changes the patient's view of his or her future. Bad news is the gap between patient's expectation and reality of patient's medical condition.[23] One cannot tell how bad any bad news is and how badly it may affect the patient's life unless doctors have already some idea of what the patients perception and expectation of the situation, *therefore before you tell ask* (find out what the patient know or think).

During breaking the bad news, SPIKES model can be used:[23,24]

S- Setting: the right physical contact of the interview (sitting down, body language, eye contact etc.) and listening skill (open questions to start with, not interrupting, facilitation etc.)

P- Patient perception: Ask patient to say what he or she knows or suspects about the medical problem and as patient replies listen and accept denial by patient.

I- Invitation: What he or she would like to know?

K- Knowledge: Provide information by using easy language, check understanding and respond to patient.

E- Explore emotions and empathizes: Identify the emotions and the cause of emotions, respond by reflecting back to the patient what he or she said. Empathic response is a technique or skill- not a feeling.

S- Strategy and summary: Involve the patient's support system (family, friends etc.), summarize and clarify the major questions.

'Doctors put too much emphasis on *curing* and not enough on *caring*. Curing costs millions but caring comes from the heart and soul and costs nothing'

People are more concerned with how doctors will communicate with patients, whether they show caring attitude and whether they are board certified. There are many varying ideas about what doctors are like, what they should be doing and what people think of them. In spite of that all people expect that doctor should meet the society's health needs and people's expectation and practice highest quality of medical care.[25]

Communication skills required for the consultation:

In all doctor patient interactions, a variety of communication skills will be required for different phases of the consultation.[3,6,7,8,26-31] These are:

Establish a rapport: Developing rapport is important in doctor- patient relationship as it enables the patient to feel understood, valued and supported.

Identify the reasons for the consultation

Gather information

Structure the consultation.

Build relationship (empathy, body language, active listening)

Provide appropriate information

Check understanding

The main responsibility for effective communication during consultation rests with the doctor. [3]

Medical interviews must, of course retain their emphasis on active listening.[19]

During listening:

- Choose an appropriate physical environment
- Remove distractions
- Make eye contact
- Consider expression and gesture
- Allow patient to talk uninterrupted as this is the key technique in facilitating the interview
- Value others opinions, concerns- shows you mean it
- Summarize, so you understood
- Check on feeling as well as content
- Avoid closed ended questions- allow to expand

Closing the interview:

At the end of interview, it is important for doctor to establish that both doctor and patient understand what occurred and what the plan is going to be.[15]

Factors to be considered during communication:

One must be aware of the following factors during communication.[3,10,11,20,21]

- What we say to each other and how we say it, matters enormously
- An *empathetic* style is sensitive necessary involvement with patients' experience that leads to shared decisions
- Requires planning and thinking in term of outcomes
- Demonstrate dynamism which requires flexibility, responsiveness and involvement
- Follows the helical model (a spiral fashion so that communication gradually evolves through interaction
- Effective communication enables us to become better doctor clinically and effective communication improves patient care and disease outcome
- The main responsibility for effective communication during consultation rests with the doctor
- Developing communication skills is a continuing process in the professional carrier

How to teach communication skills:

There is substantial evidence that communication skills can be taught and learnt.[1,3,32,33,34] Learning involves

change of behavior. To be effective, teaching should include:

- Basics of human communication
- Principles of managing the clinical interview and practice of clinical interview
- Patient doctor communications
- Evidences of current deficiencies in communication, reasons for them and the consequence for patient and doctor
- An evidence base for skills needed to overcome these deficiencies
- A demonstration of the skills to be learnt on real or simulated patient
- Video demonstration
- An opportunity to practice the skills under controlled and safe conditions
- Small group discussion
- Role play
- Constructive feedback on performance and reflection on the reasons for any unconstructive behaviors

Barriers to effective communication:

There are number of barriers to communication - ranging from personal traits to organizational constraints.[5 9,14,15]

Personal:

- Lack of skill and understanding of structure of conversational interaction which encourages two way communication
- Inadequate knowledge of or training in other communication skills including body language and speed of speech
- Non appreciation of the importance of keeping patients adequately informed
- Negative attitude of doctors towards communication. Doctors always remain concerned to treat illness rather than focusing on the patients' holistic needs such as psychological and social well being
- Lack of time, uncomfortable topics, lack of confidence, concerns relating to confidentiality and work overload
- Lack of knowledge about the illness or treatment
- Inconsistency in providing information
- Language barrier
- Tiredness and stress
- Personality differences between doctors and their patients

Organizational:

Factors that contribute to and exacerbate poor communication ate often related to the organizational constraints within which doctor work.

- Lack of time
- Work load
- Interruptions
- Lack of organizational support

Conclusion:

The most successful doctor-patient relationships are those in which both the patient and physician feel comfortable and confident in each other's ability to communicate. In order to deliver effective health care, doctors are expected to communicate competently both orally and in writing with a range of professionals. Therefore, it is essential to ensure that appropriate and effective training opportunities are available to medical students and doctors to develop and refine such skill in order to facilitate interaction with patient, their relatives and with the professionals.

References:

1. Snadden D, Ker JS. Communication skills. In: Dent RA, Harden RM, editors. A Practical guide for medical teacher. 2nd ed. Churchill Livingstone, Edinburgh. 2005. 238-47
2. Lee A, 2013. Doctor Patient Relationship. Available from: http://www.iffgd.org/site/manage your health [Accessed August 22, 2013]
3. British Medical Association Board of Medical Education. Communication Skills Education for Doctors: an update. British Medical Association. London. 2004 Available from: http://www.bma.org.uk [Accessed November, 2004]
4. Weir K. Improving patient- physician communication. Psychology 2012; 43 (10): 36-38
5. Jennifer FH, Nancy L. Doctor- Patient Communication: A Review. The Ochsner Journal 2010; 10: 38-43
6. Tattersall M, Ellis P. Communication is a vital part of care. BMJ 1998; 316:1891-92
7. Duffy FD, Gordon GH, Whelan G et al. Assessing competence in communication and interpersonal skills: the Kalamazzo II report. Acad Med 2004; 79 (6): 495-507

8. Van Zanten M, Boulet JR, McKinley DW et al. Assessing the communication and interpersonal skills of graduates of international medical schools. Acad Med 2007; 82 (10): 65-68

9. Brinkman WB, Geraghty SR, Lanphear BP et al. Effect of multisource feedback on resident communication skills and professionalism: a randomized controlled trial. Arch Pediatr Adolesc 2007; 161 (1): 44-49

10. Boon H, Stewart M. Patient-physician communication assessment instrument: 1986 to 1996 in review. Patient education and counseling 1998; 35: 161-76

11. Dalen JV, Prince CJAH, Scherpluer AJJA et al. Evaluating Communication Skills. Advance in Health Science Education 1998; 3:187-95

12. Arora N. Interacting with cancer patients: the significance of physicians' communication behavior. Soc Sci Med 2003; 57 (5): 791- 806

13. Bredart A, Bouleuc C, Dolbeault S. Doctor-patient communication and satisfaction with care in oncology. Curr Opin Oncol 2005; 17 (14): 351-54

14. Schattner A. The Silent Dimension: Expressing Humanism in each Medical Encounter. Arch Intern Med 2009; 169: 1095-99

15. Haftel J. Ipson I. Patient-Doctor Communication. 2008. Available from: http://www.med.umich.edu/med students/cur Res [Accessed August, 2008]

16. Frager DC, Coyne L, Lyle J et al. Which treatment helps? The patients' perspective. Bull Menninger Clin 1999; 63(3): 388-400

17. Avis M, Bond M, Arthur A. Questioning patient satisfaction: an empirical investigation in two outpatient clinics. Soc. Sc. Med 1997; 44 (1): 85-92

18. Fitzpatric R. Survey of patient satisfaction: Important general consideration. BMJ 1991; 302: 887-889

19. Brown JB, Boles M, Mullooly JP et al. Effect of clinician communication skills training on patient satisfaction: a randomized controlled trial. Ann Intern Med 1999; 131 (11): 822-29

20. Suarez-Almazor ME. Patient-physician communication. Curr Opin Rheumatol 2004; 16 (2): 91-95

21. Consumers value of information on quality when selecting doctor and health plan (editorial). Medical Practice Communication 1997; 4 (3): 3

22. Stewart MA. Effective physician-patient communication and health outcome: a review. CMAJ 1995; 152 (9): 1423-33

23. Baile WF, Buckman R, Lenzi R et al. SPIKES – a six step protocol for delivering bad news: application to the patient with cancer. Oncologist 2000; 5 (4): 302- 11

24. Fentiman IS. Communication with old breast cancer patients. Breast J 2007; 13 (4): 406- 409

25. Parkhouse J. Mirror , mirror on the wall (editorial). Medical Education 1985; 19: 03-94

26. Barrington D, Selagy C. Skills training in medical education: What skills and when should they be introduced. Education for General Practice 1996; 7: 16-22

27. Dunn WR, Hamilton DD. Techniques of identifying competencies needed for doctors. Medical Teacher 1995; 7 (1): 15-25

28. Lane DS, Ross V. The importance of defining physicians' competencies: Lesson from preventive medicine. Acad Med 1994; 69 (12): 972-74

29. Kern DE, Cole KA. More than Doctors' Communication Skills. Medical Education 2005; 39: 442-447

30. Simpson JG, Furnace J, Crosby J et al. The Scottish doctor-learning outcomes for the medical undergraduate in Scotland: a foundation for competent and reflective practitioners. Medical teacher 2002; 24:136-43

31. Dalen JV, Prince CJAH, Scherpluer AJJA et al. Evaluating communication skills. Advance in Health Science Education 1998; 3: 187-95

32. Wenghofer EF, Williams AP, Klass DJ et al. Physian- patient encounters: the structure of performance in family and general office practice. J Contin Educ Health Prof. 2006; 26: 285-293

33. Maguire P. Can communication skills be taught? British Journal of Hospital Medicine 1990; 43 (3): 215-16

34. Martin D. Martins' map: a conceptual framework for teaching and learning the medical interview using a patient centered approach. Medical Education 2003; 37: 1145-53

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Early detection of Prostate Cancer Bangladesh Perspective

MA SALAM

(J Banagladesh Coll Phys Surg 2014; 32: 89-93)

Introduction:

Generally the prostate cancer is a slow growing cancer and early detection may allow an effective protocol of treatment to achieve a long life. The goal of screening for prostate cancer is to find it early, in the hope that it can be treated more effectively. Screening refers to testing to find a disease such as cancer in people who do not have symptoms of that disease. Screening can help find cancers at an early stage, when they are more easily cured. The goal of screening is to help people live healthier, longer lives.

What is the rationale for screening?

Cancer screening has become part of modern medicine. Today the screening for breast, cervical and colorectal cancer is already normal practice in some countries, and will probably become routine in other countries in the future. Screening for prostate, melanoma and lung cancer are subject to ongoing studies[1-3]. Logic behind screening is simple: to detect cancers at an early stage, when they are still curable.

The screening is currently performed using one of the three methods: mass screening (i.e. large scale screening of an entire population), selective screening (i.e. screening of high-risk populations) or opportunistic screening (e.g. incorporated as part of a medical consultation). Diagnostic testing differs from screening as it attempts to identify the disease in the presence of symptoms, while screening is offered to symptom-free individuals[2-5].

All screening procedure carries a risk of over diagnosis and overtreatment, which should be balanced against the benefits for those in which the cancers are diagnosed at a curative stage. The potential value of PSA for the early detection of prostate cancer was described in the early 1990s, both prostate cancer incidence and mortality rates have changed profoundly[6]. For example between 1989 and 2003, the age-standardized incidence rate of prostate cancer increased by 48.4% in The Netherlands (reaching an incidence of 93.2 cases per 100,000 men). Based on rates from 2001 to 2003, 17.1% of U.S. men born today will be diagnosed with cancer of the prostate at some time during their lifetime. It is now the most frequently diagnosed non-cutaneous cancer, with 225,000 new cases reported each year in Europe alone[7].

This increase of incidence suggests that this is due to the increased detection of cancers. This is supported by the reports on autopsy studies have revealed that histologic prostate cancer occurs in an even larger proportion of men compared to the screening incidence: up to 55% of men in their fifties, and 64% of men in their seventies have prostate cancer diagnosed at autopsy, while only 5–10% are detectable in a screening setting during life[8].

Most guidelines, still mention the traditional cut-off of 4 ng/ml as an indication for biopsy. An increase in the number of core samples per biopsy have been advocated, based on the observation that more cancers are diagnosed when more biopsies are taken. Cut-off values of PSA for biopsy have been reduced in some areas of the world due to the detection of significant cancers in the low PSA range[9]. The awareness of prostate cancer in the general population has been increased due to print and electronic media and also from the urologic profession[9]. If the current trends of transmission of information about prostate cancer continue, the number of living men diagnosed with prostate cancer will increase even further[11]. Prostate cancer can often be found early by testing prostate-specific antigen (PSA) in a man's blood digital rectal exam (DRE). If the results of either one of these tests are abnormal, further testing is needed to see if there is a cancer. If prostate cancer is found as a result of screening with the PSA test or DRE, it will probably be at an earlier, more treatable stage than if no screening were done

Routine screening for prostate cancer in US since 1990, the prostate cancer death rate has dropped. But it is not

Address of Correspondence: Professor MA Salam, Chairman, Urology and Transplantation Foundation of Bangladesh, Panthapath, Dhaka, Bangladesh, Former Chairman and Professor of Uro Oncology BSMMU.

Received: 1 January, 2013 **Accepted:** 15 March, 2014

clear that this reduced death rate is a direct result of screening. It could also be caused by other factors like improvements in treatment of prostate cancer.

This is clear that screening program may help to find many prostate cancers early, but there are problems connected to the prostate cancer screening tests used today. Both the PSA test and the DRE are not 100% accurate. These tests can sometimes have abnormal results even when a man does not have cancer (false-positive results). Normal results can also occur even when a man does have cancer (false-negative results). Equivocal and unclear test results can cause confusion and anxiety and require more expansive and invasive test. False-positive results can lead some men to have a prostate biopsy when they do not have cancer. The trans rectal US guided biopsy may be associated with small risks of pain, infection, and bleeding. A false-negative results can give some men a false sense of security even though they actually have prostate cancer.

More importantly even if screening detects a cancer, doctors often can't tell whether this cancer is truly dangerous or not. Detecting and treating all prostate cancers early might seem as if it would always be a good thing, but some prostate cancers grow so slowly that they would probably never cause problem, may be it would never have led to their death, or even caused any symptoms. These men may still be treated with either surgery or radiation, which may be associated with urinary, bowel, and or sexual side effects that may seriously affect a man's quality of life.

Some of the detected cases particularly elderly peoples are to be followed without being treated right away and enrolled in a watchful waiting or active surveillance program. Although this group of patients is not treated right away, they still need regular blood tests and prostate biopsies to determine the need for future treatment. The entire program is linked with risks of anxiety, pain, infection, and bleeding. A large number of studies are going on to see if prostate cancer screening is worthwhile to reduce the risk of death from prostate cancer. The most recent results from 2 large studies were not conclusive and conflicting, and didn't offer clear answers.

Results from one study done in the United States found that annual screening with PSA and DRE did detect more prostate cancers than in men not screened, but this screening did not lower the death rate from prostate cancer. A European study did find a lower risk of death from prostate cancer with PSA screening (done about once every 4 years), but the researchers estimated that about 1,050 men would need to be screened (and 37 treated) in order to prevent one death from prostate cancer. Neither of these studies has shown that PSA screening helps men live longer (lowers the overall death rate).

The American Cancer Society recommends that men thinking about prostate cancer screening should make informed decisions based on available information, discussion with their doctor, and their own views on the benefits and side effects of screening and treatment.

Screening men who are older or in poor health in order to find early prostate cancer is less likely to help them live longer. This is because most prostate cancers are slow growing, and men who are older or sicker are likely to die from other causes before their prostate cancer grows enough to cause problems. The young people particularly if the prostate cancer runs in their family and develop prostate cancer, it may shorten your life if it's not caught early.

Currently there is no unanimous opinion in the medical community regarding the benefits of prostate cancer screening. Those who advocate regular screening believe that finding and treating prostate cancer early offers men more treatment options with potentially fewer side effects. Who recommend against regular screening note that because most prostate cancers grow very slowly, the side effects of treatment would likely outweigh any benefit that might be derived from detecting the cancer at a stage when it is unlikely to cause problems.

In 2012, the U.S. Preventative Task Force recommended against the use of PSA screening for healthy men of all ages, stating that the harms of screening outweigh the benefits. In contrast, physician-led groups, such as the American Society of Clinical Oncology and the American Urological Association, maintain that PSA screening should be considered in the context of a man's life expectancy and other medical conditions. Most experts agree that there is no role for PSA screening for men expected to live less than 10 years. Ultimately, decisions about screening should be individualized based on a man's level of risk, overall health, and life

expectancy, as well as his desire for eventual treatment if he is diagnosed with prostate cancer.

The American Urological Association (AUA) Foundation changed and updated it's screening recommendations on May 3, 2013. The PSA test remains an important tool in the diagnostic process. Men over 40 should discuss PSA screening with their physicians to determine if and when PSA screening is right for them, based on health and family history factors, they differ from those of the U.S. Preventive Services Task Force (USPSTF).

Timing for screening of Prostate cancer

Appropriate age to start screening is generally based on individual risk, with age 40 being a reasonable time to start screening for those at highest risk for example genetic predispositions or strong family histories of prostate cancer at a young age. For otherwise healthy men at high risk (positive family history or African American men), starting at age 40-45 is reasonable. Most guidelines recommend an initial PSA and DRE at age 40, but others recommend starting at age 50. When to stop screening is also controversial. Some groups propose 75 as a reasonable cut-off age. Other groups suggest this is an individual decision based on life expectancy and overall current health.

Indication of Biopsy of prostate

A prostate cancer screening may reveal results that prompt a doctor to recommend a biopsy. There are many other supplementary tests and considerations that can help a man who is undergoing screening decide if a biopsy is necessary, including: Lower vs. higher free PSA test, PSA velocity (rate of rise over time), PSA density (PSA per volume of prostate), Family history, Ethnicity, Prior biopsy findings, Abnormal digital rectal exam results and Different forms of PSA (i.e. bPSA, pro-PSA). In general, a lower free PSA (percentage) indicates a higher risk of finding cancer at biopsy, as does a higher PSA velocity and PSA density.

Informed decision making, reduction of unnecessary prostate biopsies

Unbiased and correct information regarding this procedure and its consequences should be offered to every man considering prostate testing. Validated information, well understandable text in local language should delivers the information in all countries around the world. It may be noted that it has been seen that such information reduces the number of men who initially wanted to be screened.

Men should also ask themselves if they are at risk for having prostate cancer, and if they would like to follow the procedure screening involving PSA testing, urologic investigations, prostate biopsies, and potential treatment. It is possible to provide risk assessments for every step of the screening procedure. This willful participation might support patients and doctors in their decision to follow or refrain from further steps, dependent on their interpretation of the risk calculated. At the time of cancer diagnosis it should be explained about the presence of prostate cancer and possible treatment should be discussed.

Much interest has been given to the increase of specificity of the biopsy procedure in the general population with serum markers like PSA isoforms and kallekreins in order to diminish the number of false negative biopsies[12,13]. Enhancing specificity always resulted in a reduction of sensitivity of prostate cancers. Based on autopsy incidence, the number of potentially detectable tumors is manifold the number of currently diagnosed cancers. It is therefore not the absolute number, but the number of clinically relevant cancers that is of interest[14]. The PSA isoforms and kallekreins should therefore ideally be related to the characteristics of the cancers detected.

To reduce over diagnosis in a screening setting, markers are needed that reduce the risk on a positive prostate biopsy, increasing the specificity of this procedure. Men from the age of forty, as well as their advising doctors, need instruments to reduce their doubts and anxiety of the potential presence of a prostate cancer. This, together with balanced information about the benefits and risks of the individual outcome of screening procedures, might induce a more selective and step-wise screening action. Risk assessment, incorporating the main determinants known for the presence of prostate cancer from the age of 50, such as age, family history, and micturition complaints, should form the base of an individual screening approach. Objective values of serum markers might enhance the accuracy of such of risk predictors. prostate-cancer-specific mortality, levels of over diagnosis may remain unacceptable for population-based screening.

Bangladesh Perspective

Bangladesh is one of the small developing countries in south Asia. The prostate cancer prevalence is low compared to the developed world. But the prevalence of prostate cancer in Bangladeshi urban population is increasing as indicated by the increased number of patient are currently diagnosed with prostate cancer. In 1980s most of the prostate cancer was diagnosed as advanced cases and the treatment option was bilateral orchectomy. The scenario has been changed with time. Increased number of early and advanced cases of prostate cancer cased are detected in clinical practices. There may be various reasons for such a higher prevalence. The life expectancy of Bangladeshi population has been increased significantly; more peoples are health conscious and involved in health check program. As Serum PSA is included in the health check program they are referred to the urology OPD or Urologist consultation where they may be diagnosed with a prostate cancer. Urologist population is also increased in Bangladesh there are about 200 Urologist working in different hospitals throughout the country. An indirect screen is always taken place while elderly men presented to the general out patient department with lower urinary tract symptom, they are evaluated for their LUTS and a PSA test is included as a mandatory in their evaluation. Most urban dweller knows some information about Prostate cancer and its implication and it was observed that many times elderly men discuss their doctors about testing his PSA. Currently most of the district city general hospitals provide PSA Test. In hospital practice the early detection become possible through the PSA test and Digital rectal examination.

Conclusions:

It is still too early to say whether population-based prostate cancer screening is a useful tool with regard to cancer mortality. We must wait until the results of ongoing prostate cancer screening trials are available. Until then, routine population screening may not be encouraged. Those men who do want a PSA test should participate in carefully designed, balanced program. Various efforts are performed to find new markers in the proteome and genome of blood and urine. Based on large and longitudinal serum collections of men diagnosed with prostate cancer in screening settings, the EC-sponsored P-MARK consortium evaluates candidate markers as prognostic tools[15].

Until alternative screening tools are found, PSA will continue to be used, and over diagnosis will remain an unavoidable drawback of prostate cancer screening. The current challenge is to ensure that, over diagnosis should not result in overtreatment. To this end, research efforts presently focus on clarifying which cancers can be managed through active surveillance.

References:

1. de Koning HJ, Auvinen A, Berenguer Sanchez A et al (2002) Large-scale randomized prostate cancer screening trials: program performances in the European randomized screening for prostate cancer trial and the prostate, lung, colorectal and ovary cancer trial. Int J Cancer 97(2):237–244.
2. Lowe JB, Ball J, Lynch BM et al (2004) Acceptability and feasibility of a community-based screening programme for melanoma in Australia. Health Promot Int 19(4):437–444.
3. Van Iersel CA, de Koning HJ, Draisma G et al (2006) Risk-based selection from the general population in a screening trial: selection criteria, recruitment and power for the Dutch-Belgian randomised lung cancer multi-slice CT screening trial (NELSON). Int J Cancer (in press).
4. Henschke CI, Yankelevitz DF, Libby DM, Pasmantier MW, Smith JP, Miettinen OS (2006). Survival of patients with stage I lung cancer detected on CT screening. N Engl J Med 355(17):1763–1771.
5. O'Brien B, Nichaman L, Browne JE, Levin DL, Prorok PC, Gohagan JK (2000) Prostate, lung, colorectal and ovarian cancer screening trial project team. coordination and management of a large multicenter screening trial: the prostate, lung, colorectal and ovarian (PLCO) cancer screening trial. Control Clin Trials 21(Suppl 6):310S–328S.
6. Catalona WJ, Smith DS, Ratliff TL et al (1991) Measurement of prostate-specific antigen in serum as a screening test for prostate cancer. N Engl J Med 324(17):1156–1161. [PubMed]
7. Parkin DM, Bray F, Ferlay J, Pisani P (2005) Global cancer statistics, 2002. CA Cancer J Clin 55(2):74–108.
8. Sakr WA, Grignon DJ, Crissman JD et al (1994) High grade prostatic intraepithelial neoplasia (HGPIN) and prostatic adenocarcinoma between the ages of 20–69: an autopsy study of 249 cases. In Vivo 8(3):439–443.
9. Nadler RB, Loeb S, Roehl KA, Antenor JA, Eggener S, Catalona WJ (2005) Use of 2.6 ng/ml prostate specific antigen

prompt for biopsy in men older than 60 years. J Urol 174(6):2154–2157. [PubMed]

10. Aus G, Abbou CC, Bolla M et al (2005) EAU guidelines on prostate cancer. Eur Urol 48(4):546–551.

11. Welch HG, Schwartz LM, Woloshin S (2005) Prostate-specific antigen levels in the United States: implications of various definitions for abnormal. J Natl Cancer Inst 97(15):1132–1137.

12. Bangma CH, Rietbergen JB, Kranse R, Blijenberg BG, Petterson K, Schroder FH (1997) The free-to-total prostate specific antigen ratio improves the specificity of prostate specific antigen in screening for prostate cancer in the general population. J Urol 157(6):2191–2196.

13. Raaijmakers R, de Vries SH, Blijenberg BG, Wildhagen MF, Postma R, Bangma CH, Darte C, Schröder FH (2007) HK2 and free PSA, a powerful prognostic combination in predicting minimal prostate cancer in screen-detected men within the PSA range 4–10 ng/mL. Eur Urol (submitted).

14. Yurdakul G, Bangma CH, Blijenberg BG, van Zelst BD, Wildhagen MF, van der Kwast TH, Schroder FH (2002) Different PSA assays lead to detection of prostate cancers with identical histological features. Eur Urol 42(2):154–158.

15. Van Gils MPMQ, Stenman UH, Schalken JA, Schröder FH, Luider TM, Lilja H, Bjartell A, Hemdy FC, Petterson KSI, Bischoff R, Takalo H, Nilsson O, Mulders PFA, Bangma CH (2005) Innovations in serum and urine markers in prostate cancer.Current European research in the P-Mark project. Eur Urol 48:1031–1041.

Superficial Temporal Artery-Middle Cerebral Artery Bypass (STA-MCA) for Middle Cerebral Artery Dissecting Aneurysm in a Child

FH CHOWDHURY[a], MR HAQUE[b], MS ISLAM[c], AC SARKER[d], SM ALAM[e]

Abstract

Intracranial arterial dissection or dissecting aneurysm is relatively uncommon and usually involve the vertebro-basilar system. Here we report a pediatric case of middle cerebral artery dissection/dissecting aneurysm with sub arachnoid hemorrhage and Sylvian fissure hematoma on dominant side. After initial evacuation of hematoma, we went for trapping and excision of dissection/dissecting aneurysm followed by STA-MCA bypass. Patient recovered fully from her neurological deficit. Probably this is the first reported case of STA-MCA bypass in Bangladesh.

Key Words: STA-MCA bypass, Middle cerebral artery dissecting aneurysm, Intracranial fusiform aneurysm.

(J Banagladesh Coll Phys Surg 2014; 32: 94-98)

Introduction:

Intracranial aneurysms may be saccular and fusiform types. Fusiform aneurysms are nonsaccular dilatations involving the entire vessel wall for a short distance[1,2,3] usually caused by dissection or atherosclerosis[4]. The underlying pathologies are disorders of collagen and elastin metabolism, infections and-very rarely due to neoplastic invasion of the arterial wall [5]. Fusiform aneurysms have different underlying pathologies, hemodynamics, anatomical distributions, natural histories and treatments than do the saccular variety[4]. Clinical presentation is due to occlusion, arterial rupture, or a pressure effect.

Intracranial fusiform aneurysms are rare and represents only 3%-13% of all intracranial aneurysms[1] and are usually located in the vertebro-basilar system[3,6]. Fusiform aneurysms in the anterior circulation remain rare and occur mostly in the middle cerebral artery and internal carotid artery[4]. There are some sporadic case reports about the treatment of fusiform aneurysms[1,4,5] Here we report a pediatric case of middle cerebral artery dissecting aneurysm on dominant side. After initial evacuation of hematoma, we went for trapping and excision of dissection/dissecting aneurysm followed by STA-MCA bypass.

Case Report:

A thirteen years old right handed girl presented with history of headache, vomiting, alteration of level of consciousness and right sided paralysis. On admission her GCS score was E2V2M5=09 with right sided hemiplegia (MRC grade-0). Emergency CT scan showed left Sylvian fissure, insula and external capsular zone hematoma with mass effect (Figure1A,B&C). Through left temporo-parietal craniotomy hematoma was evacuated. After operation her GCS improved a little (E4V2M5=11) and remained obtunded. Her hemiplegia remained unchanged. We did CT angiogram of brain that showed a left sided M3 fusiform aneurysm (Figure2A,B&C). Cerebral DSA failed to show the aneurysm and distal part of involved M3 and M4.But involved M3 and M4 was visualized lately by retrograde flow from cortical anastomosis (Figure2D).

a. Dr. Forhad Hossain Chowdhury, Neurosurgeon, Dept. of Neurosurgery, Dhaka Medical College Hospital, Dhaka.

b. Dr. Md. Raziul Haque, Associate Professor, Dept. of Neurosurgery, Dhaka Medical College Hospital, Dhaka.

c. Dr. Md. Shafiqul Islam, Neurosurgeon, Dept. of Neurosurgery, Dhaka Medical College Hospital, Dhaka.

d. Dr Asit Chandra Sarker, Associate Professor, Dept. of Neurosurgery, Dhaka Medical College Hospital, Dhaka.

e. Dr. Sarwar Morshed Alam, Dept. of Neurosurgery, Dhaka Medical College Hospital, Dhaka.

Address of Correspondence: Dr. Forhad Hossain Chowdhury, Flat No-C6, Rupayan Angel,152 Shantinagar, Dhaka, Bangladesh. Phone +8801711949570, e-mail- forhadchowdhury74@yahoo.com

Received: 1 January, 2013 **Accepted:** 15 January, 2014

We re-operated on her 10 days after hematoma evacuation through the previously done craniotomy site. After dural opening posterior ramus of Sylvian fissure was exposed by arachnoid dissection. Insula, M3 and M4 portions of MCA with aneurysm were identified. Thrombosed fusiform aneurysm of M3 was found in posterior part of Sylvian fissure. We trapped and excised the aneurysm (there was some retrograde flow at the distal part of the aneurysm) (Fig. 3A). Then by using frontal branch of STA (which was already dissected out and fashioned for microvascular anastomosis) a STA-MCA bypass was made with in the involved distal M3 segment of MCA (Fig. 3B) using high magnification under operating microscope. Dura was loosely closed around the STA. Wound was closed in layers without any drain.

On the first POD she became fully conscious with GCS score-15. From 3rd post operative day (POD) her hemiplegia began to improve (Fig. 4A). At the end of two week she could walk with support and at the end of three week she could walk without support. At the time of discharge her higher psychic functions including speech were normal and there was no nominal dysphasia. At the end of three month after operation she returned to her school. Histopathology report of excised aneurysm wall failed to give any specific vessel wall disorder. Postoperative MRA of brain (six month after operation) showed patent STA-MCA bypass (Fig. 4B)

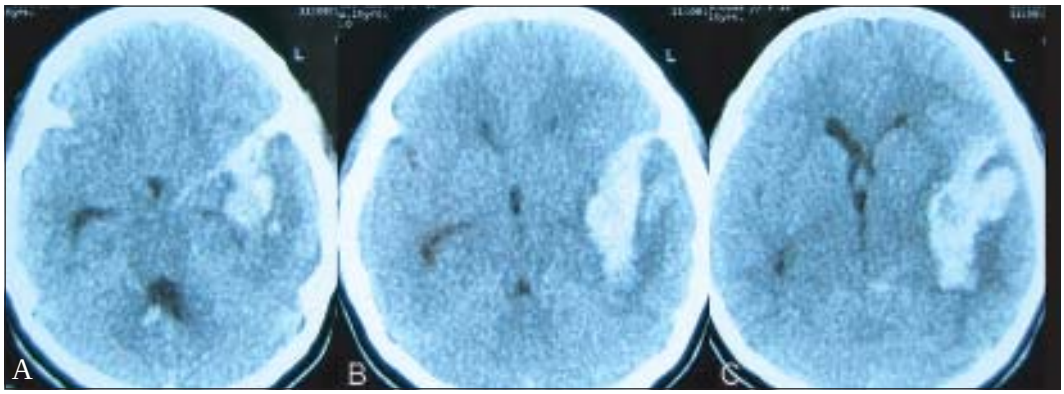


Fig.-1: *Emergency CT scan of brain, serial axial sections (A,B&C) showing left Sylvian fissure, insula and external capsular zone hematoma with mass effect.*

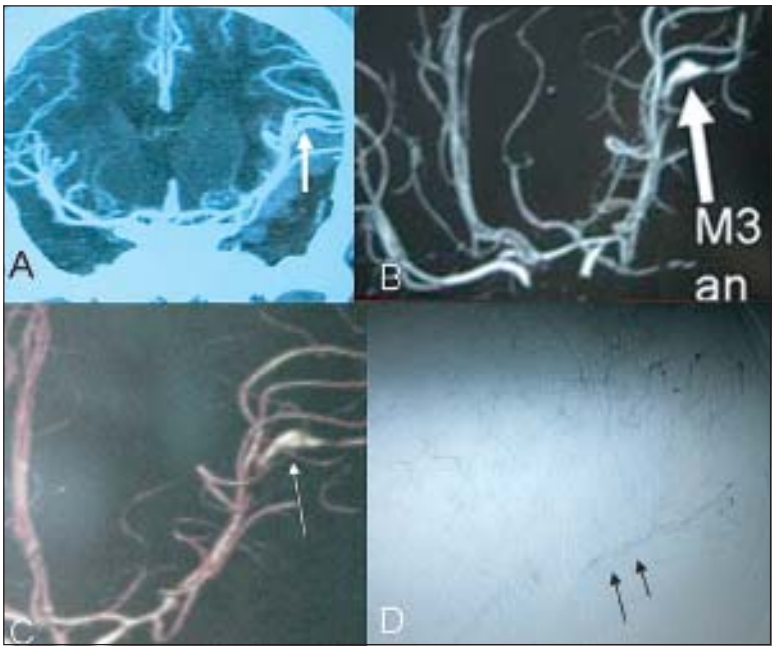


Fig.-2 (A, B & C): *CT angiogram of brain showing left sided M3 dissecting aneurysm(arrow marked).* **(D)** *Cerebral DSA showing slow and late retrograde filling of involved left distal M3 and M4 (arrows marked) form cortical anastomosis.*

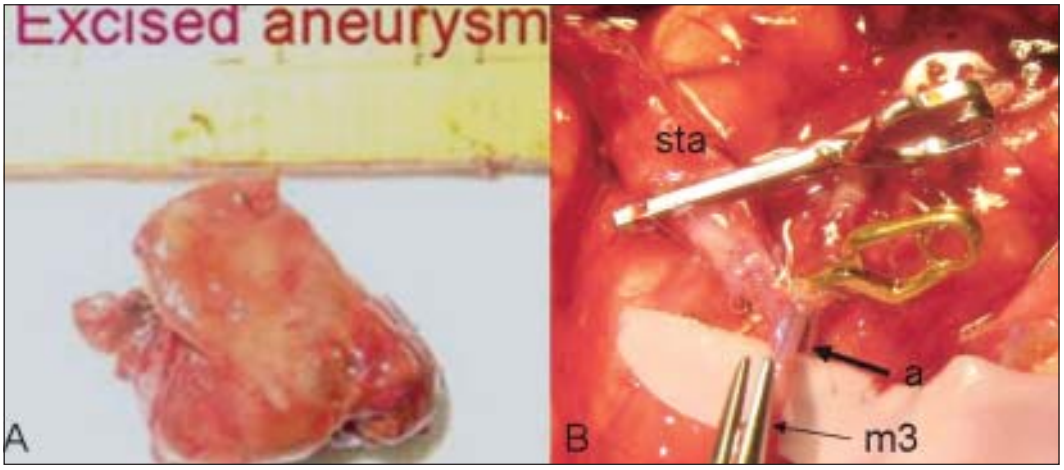


Fig.-3: *Peroperative pictures. A-excised dissecting aneurysm, B-STA-MCA (involved distal M3) bypass.*

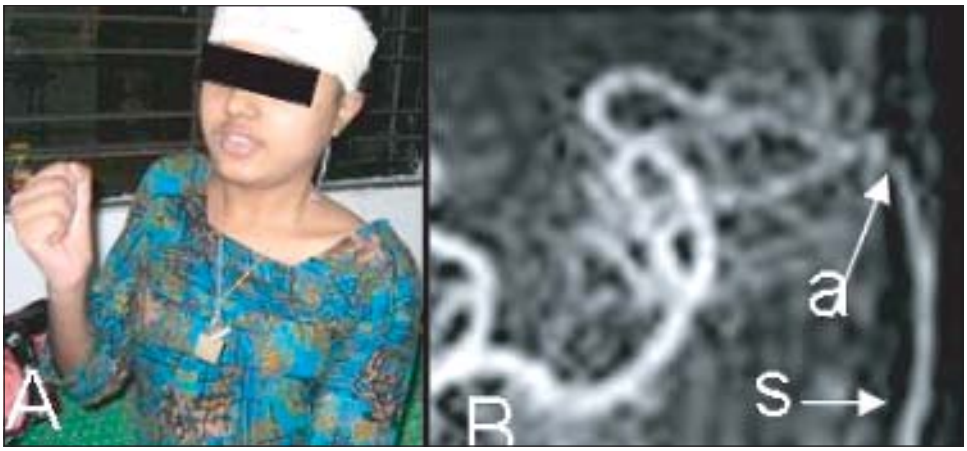


Fig.-4:*A-postoperative picture of patient in recovering stage of right sided hemiplegia. B-MRA of brain(6 months after operation) patient left STA-MCA bypass(a-anastomosis& STA)*

Discussion:

All fusiform aneurysms are spindle shape when viewed from outside[4,5]. Conceptually, there is still confusion as to the etiological, clinical and radiological features of fusiform aneurysms. However, the classic dissecting aneurysm also has a fusiform appearance[5] and several authors have recently reported the presence of fusiform aneurysms caused by dissection[1-4,7-10]. Spontaneous fusiform aneurysms are more often found in younger patients [1,2,3,4,10,11] and are more frequent in men [4,11].

Dissecting aneurysm can progress from a small focal dilatation or vessel narrowing, to a relatively thick-walled, tortuous dilatation and elongation of the artery. Hemorrhage is the most common presentation in patients with small lesions with focal dilatation, whereas ischemic symptoms were the most common presentation of patients with stenosis or vascular occlussion[5]. In our case initial symptoms were due to hemorrhage but later it could be assumed that symptoms were also due to ischaemia from thrombotic occlusion of the involved artery. Here during operation we found retrograde flow from involved artery distal to aneurysm. That means supplying area of that artery was just getting adequate arterial flow[1]‘ for survival but not adequate for functioning through cortical anastomosis. STA-MCA bypass reinforced the arterial supply of the brain to recover its functions.

Dissection has been proposed as the main underlying cause of fusiform aneurysms and most commonly involves the posterior circulation, especially vertebral and basilar arteries[3,4,7,8,10,11] Dissecting aneurysms can originate in any regions of the anterior circulation, such as the ICA[5], MCA[1,2,4,8,10] the ACA[7,8,9,11] and rarely in the anterior choroidal artery[5]. The MCA is the most common[4,12]. Various etiological factors for fusiform aneurysms have been proposed, including atherosclerosis, vessel dissection and association with other diseases such as von Recklinghausen's disease, fibromuscular dysplasia, systemic lupus erythematosus and various collagen-associated vascular diseases[2,4,12]. An intramural thrombus that ruptures into the lumen will cause a distal embolization and further expansion of the intramural clot will lead to vessel occlusion[4,5]. After occluding vessel by intramural clot, it can be recanalized and enlarged the dissection both laterally and longitudinally. Serpentine channel forms as disease extends longitudinally, combined with varying degrees of intraluminal thrombosis. Treatment of fusiform aneurysms should be based on the presence and type of symptoms, the lesion size and location and the risk of any accompanying intervention. Day et al.[4] have suggested guidelines for the treatment of patients with dissecting aneurysms of the MCA. They recommend that most small and some large focal dilatations, especially those that are asymptomatic, should be treated conservatively unless serial neuroimaging assessment indicates significant enlargement over time. However, the appearance of symptoms requires aggressive intervention. Lanzino et al.[9] and Nikawa et al.[10] also recommended conservative treatment in patients with dissecting aneurysms without neurological deterioration or recurrent SAH because of the possibility of spontaneous evolution of a dissecting aneurysm.

Day et al.[4] and several other authors [6,7,9,11] recommend that patients with stenotic or occlusive lesions presenting with acute ischemic symptoms should be treated conservatively. However, Kurino et al.[13] reported a patient with a dissecting aneurysm in the MCA who presented with ischemic symptoms and who showed a poor outcome after conservative treatment. They recommend surgical revascularization distal to the compromised artery. Consideration of aggressive treatment with endovascular or surgical methods for focal dilating fusiform aneurysms is recommended.[5] Several authors [4,10] recommended a proximal occlusion or trapping with or without resection combined with end-to-end anastomosis or EC-IC bypass [2,3,4,7,8,9,11,12,14]. But some can be treated using encircled aneurysm clips, such as the Sundt clip, by partial clipping followed by wrapping if the aneurysm is not ruptured case, and by occlusion of the aneurysm and parent vessel with packing of the coils by an endovascular method. Some authors [14] have reported that fusiform or dissecting aneurysms can be treated using endovascular methods.

References:

1. Al-Yamany M, Ross IB : Giant fusiform aneurysm of the middle cerebral artery : successful Hunterian ligation without distal bypass. Br J Neurosurg 12 : 572-575, 1998
2. Ceylan S, Karakus S, Duru S, Baykal S, Ilbay K : Reconstruction of the middle cerebral artery after excision of a giant fusiform aneurysm. Neurosurg Rev 21 : 189-193, 1998
3. Findlay JM, Hao C, Emery D : Non-atherosclerotic fusiform cerebral aneurysms. Can J Neurol Sci 29 : 41-48, 2002
4. Day AL, Gaposchkin CG, Yu CJ, Rivet DJ, Dacey RG Jr : Spontaneous fusiform middle cerebral artery aneurysms : characteristics and a proposed mechanism of formation. J Neurosurg 99 : 228-240, 2003
5. Park SH, MD; Yim MB, MD, PhD; Lee CY, MD; Kim E, MD; and Son EI,MD Intracranial Fusiform Aneurysms : It¡¯s Pathogenesis, Clinical Characteristics and Managements. JKNS 44(3):116-123,2008.
6. Echiverri HC, Rubino FA, Gupta SR, Gujrati M : Fusiform aneurysm of the vertebrobasilar arterial system. Stroke 20 : 1741-1747, 1989
7. Amagasaki K, Yagishita T, Yagi S, Kuroda K, Nishigaya K, Nukui H : Serial angiography and endovascular treatment of dissecting aneurysms of the anterior cerebral and vertebral arteries. J Neurosurg 91 : 682-686, 1999
8. Hashimoto H, Iida J, Shin Y, Hironaka Y, Sakaki T : Subarachnoid hemorrhage from intracranial dissecting aneurysms of the anterior circulation. Two case reports. Neurol Med Chir (Tokyo) 39 : 442-446, 1999
9. Lanzino G, Kaptain G, Kallmes DF, Dix JE, Kassell NF : Intracranial dissecting aneurysm causing subarachnoid hemorrhage : The role of computerized tomographic angiography and magnetic resonance angiography. Surg Neurol 48 : 477-481, 1997

10. Nikawa S, Yamada J, Sumi Y, Yamakawa H : Dissecting aneurysm of the middle cerebral artery manifesting as subarachnoid hemorrhage and hemorrhagic infarctions. Case report. Neurol Med Chir (Tokyo) 42 : 62-66. 2002

11. Wakabayashi Y, Nakano T, Isono M, Shimomura T, Hori S : Dissecting aneurysm of the anterior cerebral artery requiring surgical treatment. Case report. Neurol Med Chir (Tokyo) 40 : 624-627, 2000

12. Chuang MJ, Lu CH, Cheng MH.Management of middle cerebral artery aneurysm. Asian J Surg 35(1):42-48,2012

13. Kurino M, Yoshioka S, Ushio Y : Spontaneous dissecting aneurysms of anterior and middle cerebral artery associated with brain infarction. A case report and review of the literature. Surg Neurol 57 : 428-437, 2002

14. Hoh BL, Putman CM, Budzik RF, Carter BS, Ogilvy CS : Combined surgical and endovascular techniques of flow alteration to treat fusiform and complex wide-necked intracranial aneurysms that are unsuitable for clipping or coil embolization. J Neurosurg 95 : 24-35, 2001

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Adult Ileo- Ileal Intussusceptions- A Rare Variety in Intestinal Obstruction

SF KABIR[a], R AKTER[b]

Summary :

Intussusceptions of bowel is more frequent in children but in adult it is less. Generally adult intussusceptions carry the risk of malignancy. So treatment option is not similar with pediatric variety. A female of 40 years was admitted with severe intractable pain, vomiting but no malena. Ultrasonographic findings and CT scan of abdomen suggested that the lesion could be of bowel origin. After resuscitation the patient was managed surgically and with good outcome.

Key word: Ileum, Intussusceptions, Invagination

(J Banagladesh Coll Phys Surg 2014; 32: 99-101)

Introduction:

Adult Intussusceptions is rare but Ileo-Ileal variety is rarer in clinical practice. Although it is common in children. The etiology, clinical presentation of this condition is nonspecific and variable, may cause in delay for specific treatment. Preoperative diagnosis is usually difficult due to nature of presentations. Adult intussusceptions may be recurrent or chronic. The condition is distinct from pediatric intussusceptions in various aspects. In children it is primay and benign, pneumatic or hydrostatic reduction is sufficient in maximum cases. In contrast, adult intussusceptions are secondary to a pathologic condition such as carcinoma, polyp, stricture etc which usually discovered intraoperatively. We present a case report of bizarre presentation without any confirmation before Laparotomy.

Case report:

A 40 years old female mother of 4 child in Dhaka city, Bangladesh was hospitalized with intractable pain in the abdomen alongwith nausea and vomiting. She had low grade abdominal cramp for about a month. She had no history of bleeding per rectum or mucus in the stool. Past medical history included one caesarian section and bilateral tubal ligation. Patient was treated conservatively in the past as outdoor basis. But due to severity of pain and associated symptoms she got admitted.

a. Dr. Sheikh Firoj Kabir, Assoc. Prof. of Surgery, Tairunessa Memorial Medical College, Tongi, Dhaka, Bangladesh.

b. Dr. Mrs. Rabeya Akther, Senior Consultant, Gyane & Obs, Mitford Hospital, Dhaka, Bangladesh.

Address of Correspondence: Dr. Sheikh Firoj Kabir, Assoc. Prof. of Surgery, Safe Home: House # 287, Road # 7, Flat # B-5, Dhaka Cantt. Bangladesh. Cell : 01711-807407, E-mail: drfiroj@yahoo.com

Received: 7 March, 2013 **Accepted:** 10 September, 2013

Physical examinations showed a pale looking patient with abdominal pain which localized centrally and did not respond to analgesics and spasmodics. Patient was haemodynamically stable with a blood pressure of 130/80mm of Hg, pulse-100 beats/minute. She was afebrile. Rectal examinations found nothing abnormality.

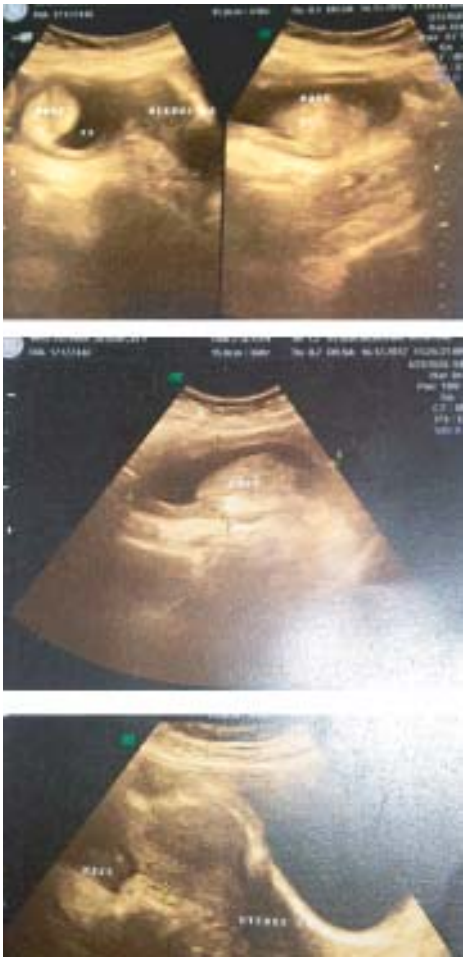


Fig.-1: *Ultra Sound Report*

Clinical examinations revealed periumbilical tenderness with hyper peristalsis with no sign of peritonism.

Laboratory test showed a CRP of 0.2 mg/dl, and white cell count of 14000/cumm of blood. Hemoglobin level was 10 gm/dl. Abdominal x-ray shows mesogastic sentinel loops.

Abdominal ultrasound revealed a 5 x 7cm ill defined bowel related mass in the umbilical region with collection in the pouch of doglus (figure-1). CT scan demonstrated an atypical soft tissue mass in the Ileo-caecal region with possibility of bowel loops. As the patient had a missed period with abdominal pain and collection in the pouch of doglus a possibility of ectopic pregnancy could not be ruled out.

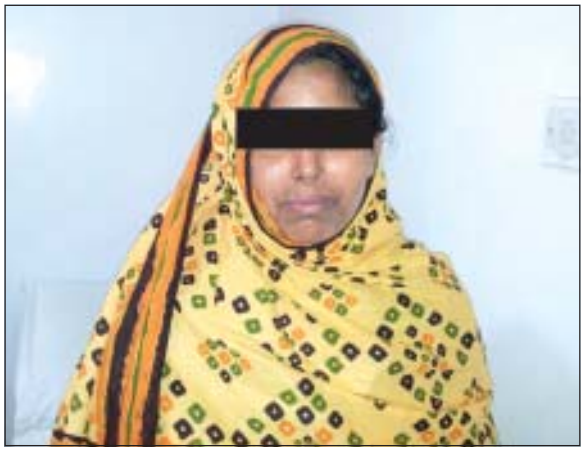


Fig.-2: *Photograph of the Patient With Permission*

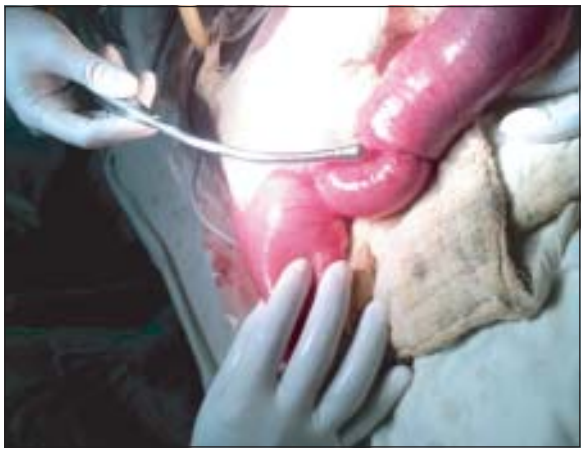


Fig.-3: *Ileo- Ileal Intussusceptions*

Based on observation laparotomy preformed. It revealed Ileo-Ileal intussusceptions. Partial resection of Ilium done with end to end anastomosis. The specimen was opened and it was an ulcerated lipoma, which protrudes into the Ileum.

Histopathological analysis showed this to be a benign submucosal lipoma. The post operative course was uneventful and the patient was discharged after one week.

Discussion:

Intussusceptions is an uncommon cause of Intestinal obstruction in adult and more than 95% cases in the pediatric age group [1]. It is expected to be found in 1/30,000 of all hospital admissions, 1/1300 of all abdominal operations[2]. The aetiology, presentation and management of intussusceptions in adults are different from children. In children intussusceptions is usually idiopathic or secondary to a viral illness. However in adults in more than 90% of cases a cause can be identified causing the intussusceptions [3,4]. This is usually a polyp or a tumour and in majority of cases the colonic tumours are malignant [5,6].

Intussusceptions leads to the development of venous and lymphatic congestion, resulting in intestinal oedema. If not treated immediately, the arterial blood supply to the bowel will be jeopardized. This can lead to ischaemia, perforation and peritonitis, resulting in a potentially lethal condition[7].

The clinical presentation is very non-specific which makes this a difficult condition to diagnose. Abdominal pain, nausea, diarrhea and bleeding per rectum are the common symptoms. Rarely this can present with acute intestinal obstruction. The classical triad of abdominal pain, sausage shaped palpable mass and passage of red current jelly stools seen in children is rarely observed in adults [6,7]. The use of investigations including a barium enema, ultrasound scan, and computed tomography can be helpful to establish the diagnosis [7-9]. CT scan has been reported to have a diagnostic accuracy of around 80% [9]. The classical finding on a CT scan is a target lesion or target sign which represents the outer intussuscepiens and the inner intussusceptum. The dense intussuscepted mass comprising of swollen bowel and mesentery within the lumen of the bowel is responsible for the characteristic target lesion seen on the CT scan[9]. Ultrasound scan is less invasive and reproducible investigation.

In 90% of cases a predisposing lesion can be found. This is contrary to intussusceptions in the paediatric

population, an organic lesion is found in only 10% of the cases [11] .In adults, it is important to differentiate between small bowel and colonic intussusceptions in 63% of cases of small bowel intussusceptions a benign underlying lesion can be found, whereas in 58% of cases of large bowel intussusceptions a malignant aetiology has to be expected[12]. The commonest benign lesion is a lipoma in the colon. These are solitary submucosal lesions with 75% occurring in the right colon. Small lipomas are asymptomatic. Other benign lesions includes adenomatous polyps and Peutz-Jeghers polyps. However in more than two thirds of cases there is a malignant tumour in the colon or small bowel resulting in intussusception[12].

Operative treatment is required in all cases of adult Intussusception and unlike children conservative treatment does not work. This usually involves segmental resection. The optimal treatment for adult intussusceptions is slightly controversial. The type of procedure depends upon the location of intussusceptions, pre-operative diagnosis and condition of the intestine at the time of laparotomy. A few authors have described intra-operative reduction of intussusceptions before resection [13] .However most authors do not recommend this due to a higher incidence of malignancy in these cases and hence the risk of tumour embolisation and seedling[13].

In most cases of adult intussusceptions, primary resection without reduction should be preformed particularly in those more than 60 years of age due to a higher risk of malignancy. In cases of small bowel intussusceptions reduction before resection should be carried out only if there is a pre-operative diagnosis of benign etiology and the bowel is viable.

Conclusion:

Ileo-Ileal intussusceptions in adult is one of the rare cause of intestinal obstruction. Unlike children, the treatment option almost always surgical. Surgical resection of the intussusceptions without reduction is the preferred treatment in adult as colonic or enteric intussusceptions are associated with malignancy.

References:

1. Tan KY, Tan SM, Tan AG, Chen CY, Chng HS, Hoe MN: Adult intussusception: experience in Singapore. ANZJ Surg 2003, 73:1044-1047.
2. Dener C, Bozoklu S, Bozokiu A, Ozdemir A: Adult intussusception due to a malignant polyp: a case report. Am Surg 2001, 67(4):35l-3.
3. Van Heel DA, Panos MZ: Colonoscopic appearance and diagno- sis of intussusception due to large bowel lipoma. Endoscopy 1999, 3 1 (6): 508.
4. Erkan N, Haciyanll M, Yildirlm M, Sayhan H, Vardar E, Polat F: Intus- susception in adults: An unusual and challenging condition for surgeons. Intj Cobrectal D/s 2005. 20:452-56.
5. Dharia KM, Marino AW, Mancini HW: Enterocolic intussuscep- tion in adults. D/s Co/on Rectum 1972, 1 5(3): 194-200.
6. Azar T, Berger DL: Adult intussusception. Annals of Surg 1997, 226:134-8
7. Yalamarthi S, Smith R Adult intussusception: case reports and a review of literature. *Postgrad* Med J, 2005, 81: 174.177.
8. Eisen LK, Cunningham JD, Aufuses AH Jr: Intussusception in adults: institutional review. JAm Coll Surg 1999, 1 88(4):390-5.
9. Lorenzi M, Iroatulam AJ, Vernillo R, Banducci T, Mancini S, Tiribocchi A, Ferrari FS, Mancini S: Adult colonic intussusception caused by a malignant tumor of the transverse colon. Am Surg 1999, 65(1): 11-4.
10. Pinero A., Riso A., Castellanos G., M. Parrilla P . Intestinal invagination in the adult. Gastro-enterol Hepatol, 1988, 21: 398-400.
11. Begos D. G., Sandor A., Modlin I.M. The diagnosis and management of adult intussusception. Am J Surg, 1997, 173: 88-94.
12. Reinjen HA. Joosten HJ, de Boer HH: Diagnosis and treatment of adult intussusception. Am J Surg 1989, 158: 25-8.
13. Nagorney DM, Sarr MG, Mcilarth DC: Surgical management of intussusception in the adult. Ann Surg 1981, 193:230-6.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Percutaneous Pulmonary Valve Implantation (PPVI) with Melody®: First Ever Case Report in South Asia

NN FATEMA[a], MR HOSSAIN[b]

Summary:

An eleven years old boy was diagnosed as a case of Tetralogy of Fallot (TOF) with absent left pulmonary artery (LPA) since two years of age. He had history of total corrective surgery in 2007 from India. He developed severe right pulmonary artery

origin stenosis, severe tricuspid and pulmonary valve regurgitation after surgery and redo surgery was performed in 2009. During redo surgery an orthotropic conduit was placed between right ventricular (RV) out flow tract and pulmonary artery. He developed significant conduit dysfunction in the form of stenosis and regurgitation in 2011 and planned for Melody® trans-catheter pulmonary valve replacement. Finally in 25th December 2012, first ever case of pulmonary valve replacement in South Asia was performed in catheterization laboratory of CMH Dhaka with technical support from a Saudi cardiac team. Patient was discharged three days after the procedure with a fully functioning Melody® valve without any stenosis or regurgitation.

(J Banagladesh Coll Phys Surg 2014; 32: 102-106)

Introduction:

Trans-catheter pulmonary valve therapy is an alternative to surgery for children and adult who have a failing surgically placed conduit for congenital heart defect like Tetralogy of Fallot (TOF), Pulmonary Atresia, Transposition of Great Arteries (TGA), Double Outlet Right Ventricles (DORV) etc. Conduits are considered as not functioning when it has become stenotic (RVOT gradient greater more than or equal to 35 mm Hg) or regurgitant (moderate or more severe regurgitation)[1]. Percutaneous pulmonary valve implantation (PPVI) is a new treatment option in patients with RVOT conduit dysfunction. Conduit size of the individual must be equal to or greater then 16 mm in diameter to qualify for the procedure. Melody ® is a replacement pulmonary heart valve used for above purpose.

The objective of PPVI is to prolong the life span of right ventricle to pulmonary artery conduit thus postponing open heart surgery[2-4].

Early follow up result of the procedure showed significant reduction of RV pressure and RVOT gradient. The most common complication which may be encountered in follow up is stent fracture[5].

a. Col (Dr.) Nurun Nahar Fatema, Paediatric Cardiologist, Combined Military Hospital Dhaka

b. Major General (Prof) Md. Rabiul Hossain, Consultant Physician, Bangladesh Armed Forces

Address of Correspondence: Col. (Dr.) Nurun Nahar Fatema, CMH Dhaka, Phone-01819239021.

Received: 17 July, 2013 **Accepted:** 7 February, 2014

So PPVI has the potential to become the standard procedure in the treatment of dysfunctional conduit. We report the first case of PPVI in South Asia where a Melody ® with 20 mm ensemble was used in 11 years old boy successfully.

Case report:

J, an eleven years old boy was diagnosed as Tetralogy of Fallot (TOF) with absent left pulmonary artery at the age of two years. He had his first surgery on 27th July 2007 in Narayana Hridayalaya, Bengalore, India which was a trans-atrial repair.

During his follow up, some residual and new problems were observed in echocardiography, chest x-ray and electrocardiography. Patient was symptomatic also in the form of exert ional dyspnoea.

So cardiac catheterization was repeated on 3rd September 2008 and severe right pulmonary artery (RPA) origin stenosis, severe tricuspid regurgitation (TR) and severe pulmonary regurgitation (PR) was noticed.

He was referred again to same centre of India for Redo surgery.

This time an orthotropic conduit (23 mm aortic homograft) was placed between RV out flow tract (RVOT) to pulmonary artery (PA) on 22nd January 2009.

He was doing well in first one year of follow up.

Later he again developed easy fatigability. His echocardiography showed severe conduit stenosis (15th December 2011) of PPG 65 mm Hg with calcified area

of 10 mm, free PR and dilated RA RV , RVOT with RVOT diameter of 18 mm. It is a common complication of conduit placement.

He was planed for PPVI as parents were refusing further surgery. Communication for procurement of valve from Medtronic, USA started.

It took about a year to arrange things to have a launching program in Combined Military Hospital (CMH) Dhaka for the first ever case of PPVI not only in Bangladesh but also in South Asia.

J was taken into cath Lab on 25th December 2012 for the procedure.

Procedure:

Equipment required.

- Short introducer sheath 18F
- Pigtail catheter 7F
- Multi track catheter 7F
- High pressure balloon Neumed 22 mm X 4 cm
- Inflation device
- BIB balloon catheter 22 mm X 4 cm
- Dilator 18F
- CP stent 39 mm and 42 mm
- Melody valve
- Ensemble delivery system (20 mm)

Steps:

Right femoral vein and right femoral artery accessed. Patient was heparinized as per standard protocol. Pressure run was recorded in Aorta, LV, RV and pulmonary artery.

Aortogram was done to assess coronary artery arrangement.

RVOT angiogram was done to assess morphology of RVOT and conduit, and severity of stenosis. Pulmonary angiogram was done to look for severity of regurgitation.

A 14F Mullin sheath was guided over a landerquest wire into PA and dilator was removed. Balloon dilatation of conduit was done with 22 mm high pressure balloon.

A CP stent of 39 mm length was placed inside conduit to cover the calcified area.

Another 42 mm CP stent was again implanted to cover the whole length of conduit and BIB balloon of 20 mm was used for expansion of stent.

The Melody valve was washed in 3 saline baths for 5 minutes each to clear the gluteraldehyde preservative.

The Ensemble of 20 mm was flushed and the balloons were de aired.

The venous access was dilated with dilators upto 22 mm.

The valve was crimped on the balloon catheter of the delivery system carefully to orientate it in the direction of blood flow and finally covered with the outer sheath.

The valve over the ensemble was forwarded over the wire to the site of implantation

The valve was uncovered at the optimum site. Check angiography was performed through side port to confirm the site of implantation. Dilatation of inner balloon was followed by dilatation of outer balloon to deploy valved stent.

The delivery system was pulled out carefully keeping the guide wire in position.

Repeat hemodynamic assessment was performed and angiography was done at pulmonary artery.

No PR or PS was noticed. Haemostasis was achieved by applying a tie with silk. Patient was shifted to cardiac ICU after extubation.

Echocardiography on next morning showed well functioning Melody valve with negligible residual

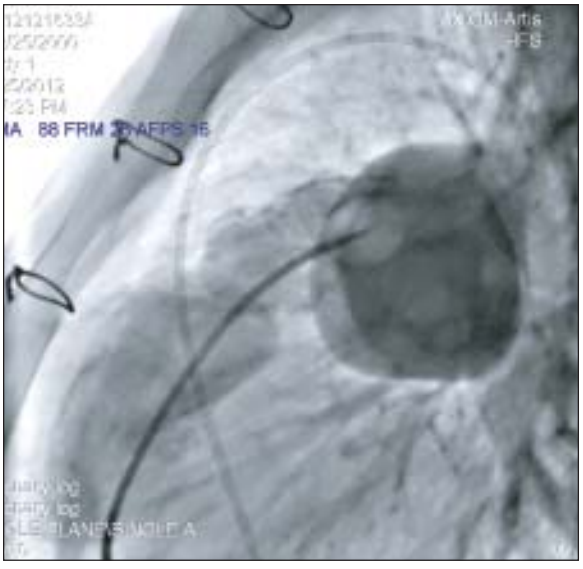


Fig.-1: *Pulmonary artery angiogram showing dysfunctional conduit.*

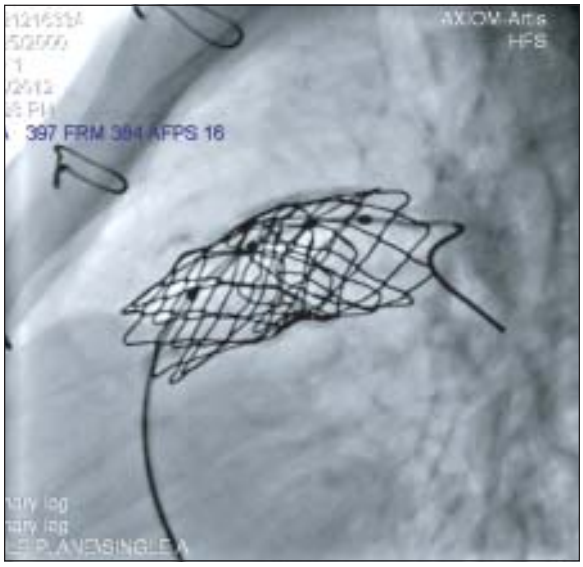


Fig.-2: *RVOT landing zone after placing two CP stent.*

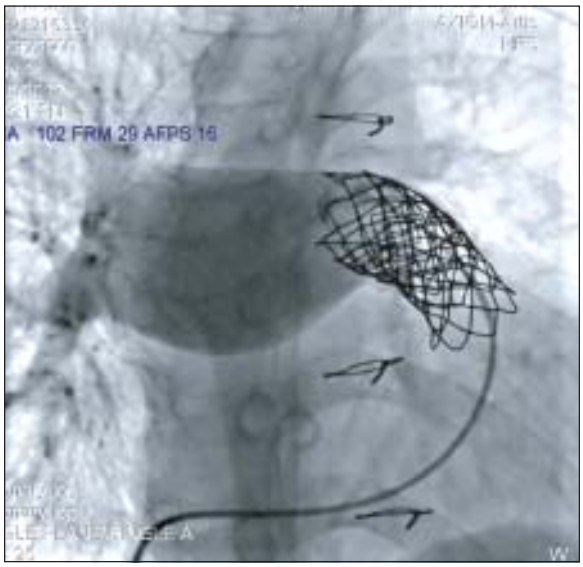


Fig.-3: *Melody® valved stent deployed after inflation of inner and outer balloon.*

pulmonary stenosis (PPG 15 mm Hg) and no PR. Patient was discharged after 03 days with an advice to continue Aspirin for 06 months and follow up after 04 weeks.

Discussion:

Congenital cardiac defect involving the right ventricular outflow tract (RVOT) require initial surgical interventions as early as the neonatal period. These defect may include severe pulmonic stenosis, pulmonary Atresia with or without ventricular septal defect, Tetralogy of Fallot, Transposition of the great arteries and Truncus arteriosus [6, 7, 8].

The surgical correction of above conditions often includes the creation of an artificial right ventricle (RV) to pulmonary artery (PA) connection by RV to PA conduit. After repair, over time, these conduits are prone to develop valve dysfunction leading to pulmonary regurgitation (PR), stenosis (PS), thrombosis, infections and calcifications [9, 10]. Chronic PR after relief of RV out flow tract obstruction can lead to RV[11] dilatation, biventricular dysfunction, heart failure symptoms, arrhythmia and sudden death[11]. There is a very close relationship between the degree of PR and RV volumes which was confirmed by many studies [11-15]. Tetralogy of Fallot is the most common from of cyanotic congenital heart disease with frequency of 06-08 per 1000 live birth [16]. Total surgical repair was available for last 50 years with favorable outcome in most cases. But surgical repair of tetralogy of Fallot (TOF) may results in many anatomic and functional abnormalities. In the majority of patients in follow up period most common problems are pulmonary regurgitation and conduit stenosis. Though RV volume overload due to severe PR tolerated over years, there is now evidence that the compensatory mechanisms of the right ventricular myocardium ultimately fail and that if the load is not eliminated or reduced, the dysfunction might be irreversible [17]. As a result early pulmonary valve replacement is recommended in many centers.

Pulmonary valve replacement can be performed electively with little risk and may improve symptoms of right ventricular failure and provide excellent midterm survival in above mentioned group of patient[18].

Our patient was a case of TOF with absent left pulmonary artery who had surgery on two occasions earlier, than pulmonary valve replacement was decided for conduit dysfunction.

Indications for PPVI are[19]

- Severe pulmonary regurgitation.
- Residual RVOT obstruction>30 mm Hg.
- Electrocardiographic evidence of QRS prolongation >180 milliseconds.

Candidates for PPVI must also fulfill the anatomic requirements necessary for safe anchoring of the percutaneous valve: So conduit provide such properties

for PPVI. In contrast native or patched out flow after TOF repair tends to be dilated and remain dynamic and therefore do not provide a secure implantation site[19].

In our case we have used Melody valve which was approved by Food and Drug Administration (FDA) in USA in January 2010.

Procedure related risks are stent fracture, embolization of valve, acute coronary compression etc. Coronary compression demands urgent surgical intervention.

Experience of PPVI in two centre over 100 patients showed excellent result regarding reduction of RVOT gradient and PR[20].

Outcome of pulmonary valve replacement in 170 patients with chronic PR after relief of ROVT obstruction was also found acceptable in another study[11].

Early result from the US clinical trial on Melody trans-catheter pulmonary valve also recommends the procedure as safe and effective one[21].

A multicentre survey was performed by Italian society of pediatric cardiology (SICP) to analyze the data of patients treated by Melody Medtronic valve[22]. This study result showed that the procedure is safe and successful. Major concerns are related to the occurrence of stent fracture and bacterial endocarditis.

Conclusion:

Percutaneous pulmonary valve implantation (PPVI) is a new treatment option in patient with RVOT conduit regurgitation and stenosis. Early results following PPVI have shown a significant reduction in right ventricular pressure and RVOT gradient. The most common complication of PPVI is stent fracture or PR in the context of endocarditis. We got excellent result in our first case. So PPVI has the potential to become the standard procedure in the treatment of dysfunctional conduit and will be easy to procure if price is considered for the developing countries.

References:

1. wwwp Medtronic.com > news room home > Media kits.
2. Oosterhof T, Hazekamp MG, Mulder BJ. Opportunities in pulmonary valve replacement. Expert Rev Cardiovasc Ther. 2009;7(9): 1117-1122.
3. Neyt M, Vinck I, Gewillig M, Van Brabandt H. Percutaneous Pulmonary and aortic valve insertion in Belgium: Going for conditional reimbursement or waiting of further evidence? Int J Technol Assess Health Care 2009;25(3):281-289.
4. Zahn EM, Hellenbrand WE, lock JE, McElhinney DB. Implantation of the melody transcatheter pulmonary valve in patient's valve in patients with a dysfunction right ventricular outflow tract conduit early from the U.S. Clinical trial. J Am Coll Cardiol. 2009; 54(18): 1722-1729.
5. Vezmar M, Chaturvedi R, Lee KJ et al. Percutaneous pulmonary valve implantation in the young 2-year follow-up. JACC Cardiovase Interv 2010; 3(4):439-448.
6. Weil Alkashkari, Qi-Ling Cao, Clifford J. Kavinsky, Ziyed M Hijaji. Percutaneous pulmonary valve implantation for RVOT Defect . Cardiac Interventions today 2010;10:1-12
7. Bove EL, Byrum CJ, Thomus FD. The influence of pulmonary insufficiency on ventricular function following repair of tetralogy of Fallot. Evaluation using radionuclide ventriculography. J Thoracic Cardiovasc Surg 1983; 85: 691-696.
8. De Ruijter FT, Weenink I, Hitch cock FJ. Right ventricular dysfunction and pulmonary valve replacement after correction of Tetralogy of Fallot. Ann Thorac Surg 2002;73:1794-1800
9. Therrien J, Siu SC, McLaughlin PR et al. Pulmoary valve replacement in adults late after repair of tetralogy of Fallot: are we operating too late? J Am coll cardiol. 2000;36: 1670-1675.
10. Gera T. Indications and timing of pulmonary valve replacement after tetralogy of Fallot repair. Semin Thorac cardiovase Surg pediets Surg arru 2006;11-22.
11. Cheuf Lee, Yaug Min Kim, Chang Ha lee, Jae Gun kwak, Chun Soo Park, Ju Young Song et al. Outcome of pulmonary valve replacement in 170 patients with chronic pulmonary regurgitation after relief of ventricular outflow tract obstruction. J Am coll cardiol 2013;60 (11):1005-1024.
12. Geva T., Sandweiss B.M, Gauvreau k., Lock J.W., Powell A.J. Factors associated with impraired clinical status in long-term survivors of tetralogy of Fallot repair evauated by magnetic resonance imaging, J Am Coll Cardiol 2004; 43: 1068-1074.
13. Bouzas B., Kiler P.J., Gatzoulis M.A., Pulmonary regurgitation: not a benign lesion, Eur Heart J 2005 26 () 433-439.
14. Cheung E.W., Wong W.H., Cheung Y.F... Meta-analysis of pulmonary valve replacement after operative repair of tetralogy of Fallot, Am J Cardiol 2010 106 () 552-557
15. Geva T., Gauvreau K., Powell A.J., et al. Randomized trail of pulomary valve replacement with and without right ventricular remodeling surgery, Circulation 2010 122 () s201-S208
16. Geva T. indications and timing of pulmonary valve replacement after Tetralogy of Fallot repair. Semin thorac cardiovase Surg pediatric Card Surg annu 2006;3447:11-22
17. Shachin Khambadkone, Philip Bonhoeffer. Complications of percutaneous pulmonary valve replacement in Complications

during percutaneous interventions in congenital and structural heart disease. Editors Ziyad M Hijaji, Ted Feldman, John P Cheatham, Horst Sievert. Informa Health care 2009 UK Ltd.

18. Emanuela R. Valsangiacomo Buceched, Hitendu H. Dave, Christian J. Kellenberger, Ali Dodqe-khatami, Rene Pretre, Felix Barger and Urs Bauersfeld. Remodelling of the right ventricle after early pulmonary valve replacement in children with repaired Tetralogy of Fallot: Assessment by cardiovascular magnetic resonance. Eur Heart J (December 2005) 26 (24) 2721-2727.

19. D Ruijter FT, Weenink I, Hitchcock FJ et al. Right ventricular dysfunction and pulmonary valve replacement after correction of Tetralogy of Fallot. Ann Thorac surg 2002; 73:1794-1800.

20. Andress Eicken, Peter Ewert, Alfred Hager, Bjoren peters, Sohrab Fratz, Titus Kuchne et al. Percutaneous pulmonary valve implantation: two centre experience with more then 100 patients. Euoropean Heart Jaurnal 2011; 32:1260-1265.

21. Evan M Zahn, William E, Hellenbrand, James E Lock, Dott B. McElhinney . Implatation of the Melody Transcatheter pulmonary valve in patients with a dysfunctional right ventricular outflow tract conduit. J Am Col Cardiol 2009;54 (18): 1722-1729

22. Gianfranco Butera, Ornella Milanesi, Isabella Spadoni, Luciane Piazza, Andrea Donti, Christian Ricci et al. Melody transcatheter pulmonary valve implantation, Results from the Registry of the Italian society of pediatric Cardiology. Cath Cardiovasc Interv 2013; 81: 310-316.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 32, No. 2, April 2014

Hyperreflexia in Guillain Barre Syndrome: A Case Report

AK SHAIKH[a], P SENGUPTA[b], KD MOHAMMAD[c], SM ALAM[d], R RABBANI[e], M HOSSAIN[f], MK DAS[g]

Abstract:

Guillain Barre Syndrome (GBS) is an immune mediated polyradiculoneuropathy classically characterized by acute ascending type of motor weakness of limbs with areflexia but in AMAN variant of GBS preserved or exaggerated reflex sometimes can occur. We report a 45 year old female patient who presented with acute flaccid quadriplegia, bilateral facial lower motor type nerve palsy and bulbar involvement, initial hyperreflexia of all four limbs and bilateral plantar extensor response 7 days following an attack of diarrhoea. Sensory and bowel bladder function was intact. She was treated with IV immunoglobulin and IV methylprednisolone. Nerve Conduction study (NCS) revealed AMAN variant of GBS. All reflexes disappeared on the 2nd day onward but returned on 21st day of illness when muscle power also improved. Follow up NCS confirmed regeneration of nerves of all four limbs. So in any patient presenting with acute quadriparesis GBS should be in the differential diagnosis even if there is preserved or exaggerated deep tendon reflexes.

Key words: Guillain Barre Syndrome, Hyperreflexia, AMAN variant GBS

(J Banagladesh Coll Phys Surg 2014; 32: 107-109)

Introduction:

Guillain Barre Syndrome (GBS) is an acute, frequently severe and fulminant immune mediated polyradiculopathy.[1] It is clinically characterized by acute symmetric muscle weakness and areflexia or hyporeflexia. Hyperreflexia has also been reported in different GBS variants most commonly with AMAN variant. So even though hyporeflexia or areflexia is necessary for the diagnosis of GBS, hyperreflexia does not exclude a GBS variant.

a. Dr Abdul Kader Shaikh, Associate Professor, Dept. of Neuromedicine, BSMMU, Part time Consultant, Square Hospital Ltd

b. Dr Poly Sengupta, MCPS Specialist, Square Hospital Ltd, Dhaka

c. Prof Dr Kazi Deen Mohammad, Professor and Principal, Dhaka Medical College and Hospital

d. Dr Sheikh Mahbub Alam, Assistant Professor, Dept. of Neuromedicine, BSMMU, part time Consultant, Square Hospital Ltd

e. Dr Raihan Rabbani, Assosiate consultant, Square Hospital, Dhaka

f. Dr Mosharof Hossain, Specialist, Square Hospital Ltd, Dhaka

g. Dr Mrinal Kanti Das, Assistant Registrar, Mymensingh Medical College Hospital.

Address of Correspondence: Dr Abdul Kader Shaikh, Associate Professor, Dept. of Neuromedicine, BSMMU, Part time Consultant, Square Hospital Ltd

Received: 4 June, 2013 **Accepted:** 14 March, 2014

We report a case of AMAN variant of GBS with brisk reflexes during the onset and recovery phase of GBS.

Case report

Mrs Shahnaj Parvin, a 45 year old female patient from Barisal was admitted in Square Hospital LTD on 19/03/2012 with the complains of sudden weakness of all four limbs and difficulty in swallowing of both solid and liquid diet for 1 day following an attack of diarrhea 7 days prior to admission. There was no associated fever, fatigability, headache, skin rash, diplopia, urinary incontinence, altered level of consciousness, convulsion. There was no previous history of similar weakness and no diurnal variation in weakness of limbs. She is a known case of hypertension and apart from history of hysterectomy 5 years ago she had no other significant past medical history.

On examination at admission she was fully conscious and oriented. She had bilateral lower motor neuron type facial nerve palsy and bulbar palsy with absent gag reflex. Muscle power was 3/5 in upper limbs and 1/5 in lower limbs. All deep tendon reflexes were exaggerated with bilateral extensor plantar response. She was transferred to ICU and was put on mechanical ventilation the 2nd day of admission. One day after admission all jerks became absent except biceps jerks and muscle power in both upper and lower limbs became 1/5. On the 3rd day all jerks were absent but plantar response continued to remain bilaterally extensor. Sensory and

bowel bladder function was intact all through. Investigations showed normal total and differential count of WBC, electrolytes and creatinine kinase. CSF study done on 2nd day of admission showed normal cell count with normal protein (40 mg/dl) and glucose (68 mg/dl), ADA, MTB PCR with gram stain, AFB stain and culture sensitivity were negative. MRI of brain and cervical spine were normal. NCS revealed AMAN variant of polyneuropathy. Anti Campylobacter Jejuni antibody, anti GM1 antibody was not done. She was treated with IV Immunoglobulin from Day 2 and continued for 5 days; IV methylprednisolone was given on Day 1 as cervical transverse myelitis with cranial polyneuropathy was considered as another possibility. Nine days later her muscle power started improving and 21 days after admission all jerks reappeared. At discharge she had muscle power of 2/5 in all four limbs, considerable improvement of facial nerve palsy and bulbar palsy. Follow up about 1 year later on 3/3/2013 revealed muscle power 3/5in proximal muscles of lower limb, 4/5 in distal group of muscles, 4/5 in both upper limbs. All jerks were lively, bilateral flexor plantar response. Repeat NCS revealed renervation of all four limbs which supports our diagnosis.

Discussion:

Guillain Barre Syndrome manifests as a rapidly evolving areflexic motor paralysis with or without sensory disturbance.[1] Although hypo/areflexia is mandatory for the diagnosis of GBS, retained tendon reflexes or brisk reflexes have been reported in some GBS variants most commonly in AMAN in Chineese, Japanese, and Europian and Indian population. [2, 3, 4]

Preceeding history of C. Jejuni infection is frequently found associated with AMAN variety of GBS with hyperreflexia.[5,6,7,8] Patients in such cases have history of abdominal pain and diarrhea.[1] Hyperreflexia/ preserved tendon reflexes are thought to be present in mild or less severe form.[2] Cranial, sensory, bulbar and respiratory involvement are also less frequent in such cases.[2] However acute flaccid quadriplegia with bilateral facial and bulbar involvement requiring artificial ventilation on the 2nd day of admission until 1 week before discharge in our patient indicated severe form of the disease.

Preserved tendon reflexes in GBS have been considered as an indicator of rapid clinical recovery.[3] Patients with axonal GBS however can show both rapid and slow recoveries.[5] It is proposed that patients with preserved tendon reflexes recover quickly because at least some of the motor units need to function to elicit visible reflexes. [5] Hyperreflexia have been found to be present both in acute progressive phase and in the early recovery phase in a study, [2] and also throughout the course of the illness.[9] In our case, the patient had hyperreflexia of all four limbs with bilateral plantar extensor on the day of admission, however on the 2nd day all jerks disappeared except both biceps jerks which disappeared on the 3rd day. And all the jerks became lively 21 days after admission. Muscle power started improving on the 9th day of admission.

Circumstantial evidence suggests that all GBS results from immune response to nonself antigens (infectious agents, vaccine) that misdirect to host nerve tissues through a resemblance of epitope (molecular mimcry) mechanism. [1] A proposed mechanism for hyperreflexia is that the primary immunological attack is directed against the motor axons and Anti GM1 antibody binds with neural structures in the spinal cord as a result of the local dysfunction of the blood – CNS barrier caused by the inflammation of the spinal nerve roots leading to the dysfunction of spinal inhibitory interneurons, or of the upper motor neurons causing the abnormal reflex spread to other segments. [2] Functional corticospinal tract involvement has also been proposed.[3] The presence of normal sensory nerve function rather than motor is required for tendon jerks because tendon jerks are dependant on synchronized volley of impulses, a purely axonal lesion would preserve tendon jerks better than a demyelinating lesion.[5]

CSF shows albuminocytological dissociation in most but not all cases of GBS presenting with hyperreflexia, [2,7] and may be absent if done within 1st week[1] . We performed CSF on the 2nd day of admission, which can explain the absence of raised protein level in our case.

Hyperreflexia is occasionally seen in chronic motor neuropathy associated with high titre anti GM1 antibody.[2] Anti GM1 antibodies , anti GD 1a Ig M antibodies, Anti GalNAc GD1a Ig G antibody are also associated with AMAN variant.[1] None of these could be done in our case because the tests are not available in our country.

NCS is the most important supportive investigation in the diagnosis of GBS. Hyperreflexia is more often found

in patients with AMAN than AIDP.[2] In our case the initial NCS report done on the 12th day of admission revealed AMAN variant of GBS, follow up NCS with needle EMG report revealed regeneration in all four limbs.

On the day of admission we considered it to be a case of transverse myelitis (cervical) with cranial polyneuropathy on the background of hyperreflexia and bilateral plantar extensor with bilateral lower motor neuron type facial nerve palsy along with bulbar involvement and so IV methylprednisolone was started. However subsequently all the jerks started disappearing and this led us to consider GBS.

Another possibility could be Postinfectious Myeloradiculopolyneuropathy but the neurological involvement in her case was symmetrical which goes in favour of GBS. In case of Myeloradiculopolyneuropathy neurological involvement is expected to be asymmetrical along with sensory involvement and CSF pleocytosis. GBS with stroke was not considered to explain hyperreflexia as the patient's Glassgow Coma Scale (GCS) was intact and MRI of brain was normal. Normal CSF along with normal MRI of brain excluded brainstem encephalitis to explain clinical features in our case.

Conclusion:

In conclusion, preserved tendon reflexes and even hyperreflexia may be present although rarely, in some GBS variants. So GBS should be in the differential diagnosis when dealing with any patient presenting with acute quadriparesis with preserved or brisk reflexes.

References:

1. Longo DL, Fauci AS, Braunwald E, Kasper DL, Hauser SL, Jameson JL, et al. editors. Harrison's principles of internal medicine. 18th ed. New York: McGraw Hill; 2012.
2. Kuwabara S, Ogawara K, Koga M, Mori M, Hattori T ,Yuki N. Hyperreflexia in Guillain Barre Syndrome: Relaion with acute motor axonal neuropathy and ant GM1 antibody. J Neurol Neurosurg Psychiatry.1999; 67: 180-184.
3. Singhal V, Bhat KG. Guillain Barre Syndrome with hyperreflexia: A variant. J Paediatr Neurosci. 2011;6:144-5.
4. Baheti NN, Manuel D, Shindhe PD, Radhakrishnan A, Nair M. Hyperreflexic Guillain Barre syndrome. Ann Indian Acad Neurol. 2010; 13(4): 305-307.
5. Kuwabara S, Mori M, Ogawara K, Hattori T, Yuki N. Indicators of rapid clinical recovery in Guillain Barre Syndrome. J Neurol Neurosurg Psychiatry. 2001; 70:560-2.
6. Islam Z, Jacobs BC, Belcum V, Mohammad QD, Islam MB, Herbrink P et al. Axonal variant of Guillain Barre Syndrome associated with Campylobacter infection in Bangladesh. Neurology. 2010; 74: 581-587.
7. Dhadke SV, Dhadke VN, Bangar SS, Korade MB. Clinical profile of Guillain Barre Syndrome. J Asso Physicians India. 2013; 61:168-172.
8. Selimovic BM, Lavrinc D, Mori O, Ng LK, Price L, Suturkova L et al. Enteritis caused by Campylobacter Jejuni followed by acute motor axonal neuropathy: a case report. Journal of Medical Case Reports. 2010; 4:101.
9. Yuki N, Hirata K. Preserved tendon reflexes in Campylobacter neuropathy. *Ann Neurol.1998;* 43:546–547.

Oculr Imaging

MZ RAHMAN[a], J AHMED[b]

(J Banagladesh Coll Phys Surg 2014; 32: 110-111)

Case Report:

1 st case: A 45 years old lady,normotensive, non diabetic presented to us with complaints of recurrent attack of pain,redness both eyes for 6 months, that were improved with the use of topical steroid. She also complaints of dimness of vision in her both eyes for 2 months On examination there were anterior Uveitis, vitritis, bilateral optic disc swelling and choroidal nodule. This inflammation and nodule give us clues for granulomatous uveitis. Her ESR was-40 mm in 1St hour, MT was 2mm induration, X-ray chest showed bilateral enlarged hilar lymph node,CT scan of brain was unremarkable. Our clinical diagnosis was bilateral granulomatous panuveitis with involvement of optic disc due to sarcoidosis.

We have started topical steroid and with the consultation with neurologist and pulmonologist oral prednisolone was started. The disease process well responded with the treatment.

2nd case: A 29 years old gentleman presented with the bilateral decrease vision for 3 months. On examination there found lare granuloma involving optic nerve head that leads to disc swelling, macular edema,hard exudates, haemorrhages and vascular tortucity. His ESR was 35 mm in 15t hour, MT showed no induration's, X-ray chest, HRCT chest, CT scan of brain were un-remarkable, serum ACE was 120 IU/L. The clinical diagnosis of sarcoid granuloma involving optic nerve head was made. With consultation of neurologist oral prednisolone was started , the lesions healed completely.

1. Dr. Md. Zahedur Rahman, Consultant Ophthalmologist, Specialist in Uveal Diseases and Ocular Inflammation, Bangladesh Eye Hospital.

2. Prof. Jalal Ahmed, Director, Professor & Head, Department of Cataract, National Institiute of Ophthalmology, Consultant Phaco and LASIK,Bangladesh Eye Hospital.

Address of Correspondence: Dr. Md. Zahedur Rahman, Consultant Ophthalmologist, Specialist in Uveal Diseases and Ocular Inflammation, Bangladesh Eye Hospital.

The images:

The fundus photograph of right eye of 1st patient (1 A) shows mild optic disc swelling otherwise normal fundus.

The fundus photograph of left eye(Fig: 1 B) shows obvious disc swelling, hyperemic disc and small nodule in peripheral fundus.

The fundus fluorescin angiography of both eyes (Fig: l C) showing hyperflurescence of optic disc due to staining

The X- chest (Fig: l D) of same patient shows bilateral hilar lymphadenopathy.

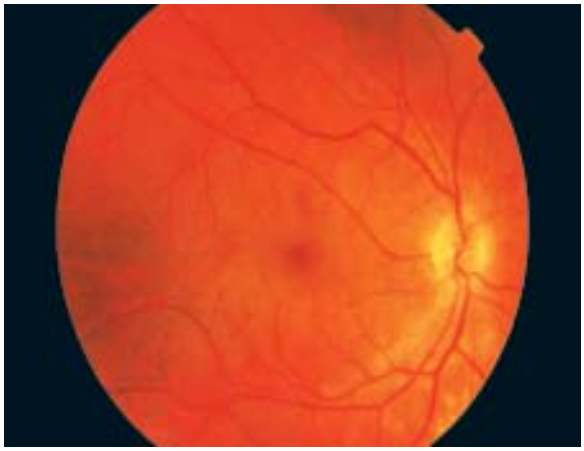


Fig.-1(A)

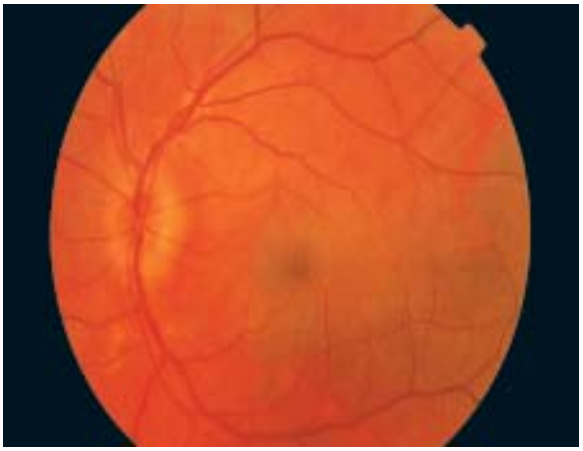


Fig.-1(B)

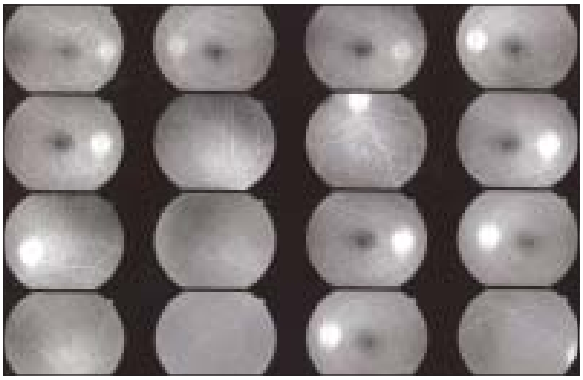


Fig.-1(C)

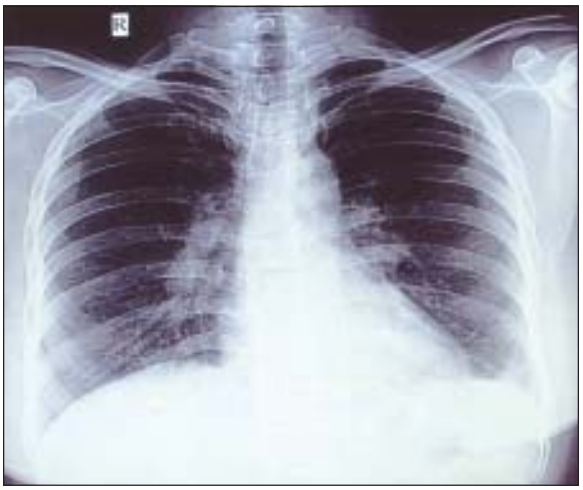


Fig.-1(D)

The fundus photograph of right eye (Fig:2A) of 2nd patient showing, large yellow white elevated lesion involving optic nerve head extending superiotemporally towards superotemporal vascular arcade and macula. There are also present disc swelling, overlying haemorrhage and vascular tortucity macular edema, hard exudates.

The fundus photograph of right eyes of the 2nd patient after treatment showing, the granuloma involving optic nerve head completely resolved. The macular edema, disc swelling, hard exudates, hemorrhages also resolved and the vascular tortucity decrease.

Differentials:

Bilateral optic disc swelling may be due to raised intracranial due to ICSOL, benign intracranial hypertension, intracranial vascular malignant hypertension, occlusion etc.

Granulomatous uveitis with optic disc swelling may be due to sarcoidosis, tuberculosisis, Vogt Koyanaga Harada disease.

Conclusion:

Various systemic diseases like sarcoidosis, tuberculosis, rheumatoid arthritis, SLE, seronegative arthopathy, Behcet,s diseae etc, ocular manifestation maybe the I^{s}' presentation.

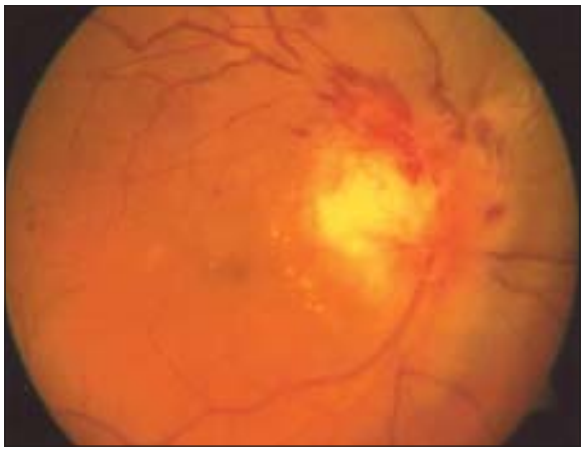


Fig.-2 (A)

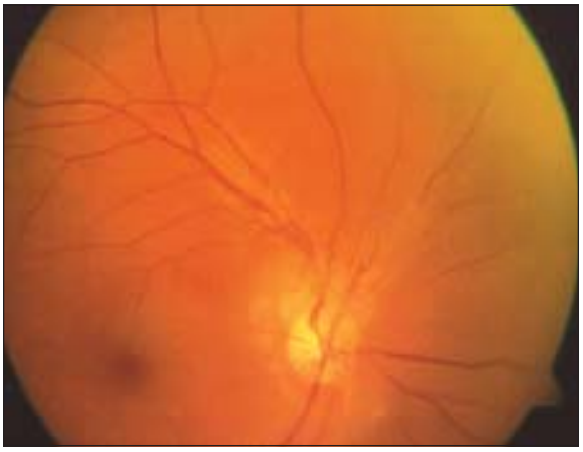


Fig.-2 (B)

LETTER TO THE EDITOR

(J Banagladesh Coll Phys Surg 2014; 32: 112-113)

To

The Editor-in-Chief

Journal of Bangladesh College of Physicians and Surgeons.

Dear Sir,

At the very outset, I would like to congratulate the editor for publishing this review article on "Breast Feeding versus Formula Feeding and Diarrheal Disease in Infants and Children-A Review". From a meticulous reading of the article, I have found the content is very informative and beneficial for the doctors in particular. Additionally, I would like to share some of my observations and comments.

Exclusive breastfeeding means that the infant receives only breast milk. No other liquids or solids are given, not even water with the exception of oral rehydration solution, or drops/syrups of vitamins, minerals or medicines. (WHO, 23 May 2014)

Appropriate feeding practices are essential for proper nutrition, growth, development and survival of infant and young children. These feeding practices which include both breast feeding and complementary feeding are collectively known as infant and young child feeding (IYCF).

It is estimated that sub-optimal breast feeding, especially non-exclusive breast feeding in the first 6 month of life, resulting in 1.4 million death and 10% of the disease burden in children younger than 5 years.[1,2]

Author of this review, indeed, described very nicely about the composition of breast milk and its anti-infective role, current recommendation on breast-feeding, benefits of breast feeding and hazards of formula feeding. Working hard, the author enriched the article by referring national and international publications.

I read the article with much interest especially on discussion about comparison of bottle-fed and breastfed children and association of various type of gasteroenterities. Author gave more emphasis to explain scientifically how human milk provides anti-infective benefits. It is to be mentioned that the protection from diarrhea through breastfeeding is mediated in two ways: directly, through specific and non-specific immune mechanism, and indirectly, as no extra water is needed in a breast feed infant[3] which may be contaminated and become the media of infection, particularly in a developing country. Antibody to Giardia lamblia also found in breast milk[4]. The least acknowledged of all the advantages of breastfeeding are the ecological benefits. Artificial milk is non-renewable products that create ecological damage at every stage of their production, distribution and use[5].

Despite evidences supporting the positive and cost-effective health impacts of exclusive breastfeeding on child survival, breast feeding practice in resource poor areas of the world is low. In Africa, Asia, Latin America and Caribean, only 47%-57% of infants less than two months and 25%-31% of infants 2-5 months are exclusively breast fed[6]. This article cited a reference which shows less than 40% of infants below 6 months are currently exclusively breastfed worldwide[7].

Apart from the information given in the article, I would like to supplement some more information:

- Breast fed infants achieve higher score in cognitive assessment than formula-fed[8].
- Breast-feeding has protective effect against obesity in children[9].
- The risk for presenting diarrhea is higher in formula fed (48.7%) and breast fed plus formula fed children (37.3%) when compared to exclusive breast feeding (32.5%)[10]. Because the acidic fraction of oligosaccharides of human milk prevents adhesion of pathogenic strains of enteropathogenic Escherichia coli serotype 0119, Vibrio cholera and Salmonella with the gut mucosa.
- A recent hospital based study reveals that prelacteal feed is given in 29.2%, colostrums is given in 79.2% and exclusive breast feeding up to six months is given in 24% babies[11].
- WHO recommends in 2010 for breastfeeding of babies by mothers known to be HIV- infected (and whose infants are HIV uninfected or of unknown HIV status) should exclusively breastfeed their infants for the first 6 months of life, introducing appropriate complementary foods thereafter, and continue breastfeeding for the first 12 months of life. Breastfeeding should then only stop once a nutritionally adequate and safe diet without breast milk can be provided[12].

- Bangladesh Government has re enacted and amended the Breast Milk Substitutes(Regulation of Marketing) Ordinance,1984 and passed The Breast milk Substitutes, Infant Foods manufactured commercially and the accessories related thereto (Regulation of Marketing) Act,2013 (Act XXXV of 2013) in the Parliament of Bangladesh on September,22,2013.
- Restricting breast milk substitute, infant foods etc. among other restrictions contained in the said act in particular section 2 clearly provides certain restriction mainly to give any financial or any other incentives to anybody related with health care in Bangladesh.

Finally, I thank the author again for highlighting such an important issue and writing the review article in details.

1. Prof. Abid Hossain Mollah
Professor of Paediatrics
Dhaka Medical College Hospital, Dhaka.

2. Dr. Zohora Jameela Khan
Assistant Professor
Paediatric Haematology – Oncology
Dhaka Medical College Hospital, Dhaka.

References:

1. Dadhch J P, Agarwal RK. Mainstreaming early and exclusive breast feeding for improving child survival, Indian Pediatrics 2009;48:11-17
2. Falar M, Jooste PL, Mqoqi NP, Benale AJ. Breast feeding and complementary feeding practices in Swaziland population.Arf J Health Sci 2000; 7:51-54.
3. Almorth S, Bidinger PD. No need for water supplementation for exclusively breastfed infants under hot and arid conditions.Tansactions of the Royal Society of Tropical Medicine and Hygiene1990; 84(4):602-604.
4. Waterspiel JN et al.Secretory anti-Giardia lamblia antibody in human milk: Protective effect against diarrhea. Pediatrics 1994; 93:28-31.
5. WABA, Breastfeeding Nature's way, World breastfeeding week Brochure (1997).
6. Lamberti LM , Walker CLF,Noiman A, Victoria C, Black RE. BMC Public Health 2011 ; (supp13):S15.
7. World Health Organization, World Breastfeeding week August 7, 2011, Retrieved August 8, 2011.
8. Anderson JW, Johnstone BM, Remley DT. Breastfeeding and cognitive development: a Meta analysis. Am J Clin Nutr 1990; 70:525-35.
9. Arenz S, Ruckeri R, Koletzko B, Kries RV. Breast-feeding and Childhood obesity-a systematic review. International Journal of obesity2004; 28:1247-1256.
10. Bener A, Ehlayel MS, Abdulrahman HM. Excusive breast feeding and prevention of diarrheal disease. A study in Qatar.Rev.Bras.Saude Matern.Infant.Recife 2011, 11(1):83-87.
11. Begum T, Hoque SA, Islam MR, Khatoon S, Shah AR. Infant Feeding Practice of Mother attending Pediatric out Patients Department in A Tertiary Care Center.Bangladesh J Child Health 2013; Vol 37(3)138-141.
12. WHO Guidelines on HIV and infant feeding, 2010; recommendation 2.

Author's Reply

To

The Editor-in-Chief

Journal of Bangladesh College of Physicians and Surgeons.

Dear Sir,

At the very beginning I express my gratitude and heartiest thanks to the editor for publishing this review article on "Breast Feeding versus Formula Feeding and Diarrheal Disease in Infants and Children-A Review". It is my pleasure, and I feel proud that, the review article focusing such an important issue is published in a prestigious, popular and widely read academic journal of Bangladesh. I have tried to disseminate already published and studied information pertaining breast milk, formula milk, breastfeeding, and consequences of formula feeding in infants and children.

I thank all reader of ***Journal of Bangladesh College of Physicians and Surgeons,*** *specially***Prof. Abid Hossain Mollah,** Professor of Paediatrics, Dhaka Medical College Hospital, who has gone through in depth of the article and expressed valuable comments and important new and additional information on the issue. Sir, thank you very much to enrich our knowledge.

I also thank **Dr. Zohora Jameela Khan,** Assistant Professor, Paediatric Haematology – Oncology, Dhaka Medical College Hospital for putting comments and enriching update on the issue.

I want to share another thing that, a study on the same issue is under way in the northern district of Bangladesh, of which data will be reached in my hand soon. I hope the gathered information of the forth coming study will give us lots of unknown findings and observations.

Finally, I again forward my regards and gratitude to BCPS journal authority and proficient readers of the journal, thank you all.

Dr. Most. Umme Habiba Begum
Assistant Prof.
Paediatric Dept.
Northern Private Medical College, Rangpur

COLLEGE NEWS

(J Banagladesh Coll Phys Surg 2014; 32: 114-119)

College news Examinations news: Results of FCPS Part-I, Part-II and MCPS examination held in January are given bellow:

4429 candidates appeared in FCPS Part-I, examinatin held in January, 2014 of which 739 candidates came out successful.

Subject wise results are as follows:

Result of FCPS Part-I Examination (January, 2014)

SL. No.	Subject	January-14		
		Total Candidate	Total Passed	Percentage %
1.	Anaesthesiology	75	23	30.67
2.	Biochemistry	1	0	0.00
3.	Dental Surgery	160	12	7.50
4.	Dermatology & Venereology	63	10	15.87
5.	Family Medicine	2	0	0.00
6.	Haematology	15	3	20.00
7.	Histopathology	19	4	21.05
8.	Medicine	1652	448	27.12
9.	Microbiology	16	2	12.50
10.	Obst. & Gynae	884	30	3.39
11.	Ophthalmology	102	28	27.45
12.	Otolaryngology	130	16	12.31
13.	Paediatrics	453	62	13.69
14.	Physical Medicine & Rehabilitation	29	8	27.59
15.	Psychiatry	8	0	0.00
16.	Radiology & Imaging	44	3	6.82
17.	Radiotherapy	35	8	22.86
18.	Surgery	741	82	11.07
19.	Transfusion Medicine			
	Total	4429	739	16.69

The following candidates satisfied the Board of Examiners and are declared to have passed the FCPS - II Examinations held in January, 2014 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons

Roll No.	Name	From where graduated	Subject
023-8907	Azizun Nessa	Dhaka Medical College, Dhaka	Nephrology
023-8912	Sk. Nishat Abdullah	Rangpur Medical College, Rangpur	Plastic and Reconstructive Surgery
023-8914	Harun-Or-Rashid	Comilla Medical College, Comilla	Urology
084-7006	Md. Golam Ferdous Alam	Rangpur Medical College, Rangpur	Anaesthesiology
084-7007	Md. Zahedul Islam	Sir Salimullah Medical College, Dhaka	Anaesthesiology
084-7010	Mohammad Omar Faruq	MAG Osmani Medical College, Sylhet	Anaesthesiology
084-7012	Mohammad Obaidullah	Sher-E-Bangla Medical College, Barisal	Anaesthesiology
084-7016	Tushar Kanti Sikdar	Chittagong Medical College, Chittagong	Dermatology and Venereology

Roll No.	Name	From where graduated	Subject
084-7021	Mohammad Salah Uddin	Rajshahi Medical College, Rajshahi	Dermatology and Venereology
084-7026	Md. Tawhidul Islam	MAG Osmani Medical College, Sylhet	Dermatology and Venereology
084-7032	Mohammad Nasir Uddin	Sir Salimullah Medical College, Dhaka	Dermatology and Venereology
084-7035	Salma Begum	Sir Salimullah Medical College, Dhaka	Dermatology and Venereology
084-7039	Humayra Sufian	Dhaka Medical College, Dhaka	Dermatology and Venereology
084-7044	Mst. Romena Alam	Rangpur Medical College, Rangpur	Haematology
084-7045	Monwar Tarek	Sir Salimullah Medical College, Dhaka	Haematology
084-7047	Mohammad Ali	Sher-E-Bangla Medical College, Barisal	Haematology
084-7048	Humayra Nazneen	Dhaka Medical College, Dhaka	Haematology
084-7049	Mafruha Akter	Dhaka Medical College, Dhaka	Haematology
084-7051	Shahanaz Jahan	Mymensing Medical College, Mymensing	Histopathology
084-7063	Md. Safiul Islam	Dhaka Medical College, Dhaka	Medicine
084-7092	Mohammad Golam Rob Mahmud	Jalalabad Ragib-Rabeya Medical College, Sylhet	Medicine
084-7202	Mostaque Ahammad	Chittagong Medical College, Chittagong	Medicine
084-7223	Nahida Zafrin	Dhaka Medical College, Dhaka	Medicine
084-7234	A.H.M. Anisuzzaman	Rajshahi Medical College, Rajshahi	Medicine
084-7269	Md. Sakhawat Hossain	Sher-E-Bangla Medical College, Barisal	Medicine
084-7274	Md. Atiqul Islam	Sir Salimullah Medical College, Dhaka	Medicine
084-7297	Md. Ashraf Uddin Ahmed	Bangladesh Medical College, Dhaka	Medicine
084-7334	Naser Ahmed	Mymensing Medical College, Mymensing	Medicine
084-7368	Debashis Biswas	Faridpur Medical College, Faridpur	Medicine
084-7373	Mohammad Mahfuzul Hoque	Dhaka Medical College, Dhaka	Medicine
084-7412	Md. Shamim Ahasan	Dhaka Medical College, Dhaka	Medicine
084-7448	Md. Monjur-E-Elahi	Dhaka Medical College, Dhaka	Medicine
084-7511	Mohammad Ali	Dhaka Medical College, Dhaka	Medicine
084-7530	Mohammad Nadim Hasan	Rangpur Medical College, Rangpur	Medicine
084-7574	Fatema Mahbooba Akter	Mymensing Medical College, Mymensing	Obst and Gynae
084-7578	Tahamina Khanum	Rajshahi Medical College, Rajshahi	Obst and Gynae
084-7608	Mst. Rawshon Ara Begum	Rangpur Medical College, Rangpur	Obst and Gynae
084-7609	Latifa Akhter	Chittagong Medical College, Chittagong	Obst and Gynae
084-7611	Jesmin Hyder	Rajshahi Medical College, Rajshahi	Obst and Gynae
084-7612	Farhana Islam	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
084-7615	Bidisha Chakma	Chittagong Medical College, Chittagong	Obst and Gynae
084-7619	Sultana Begum	Mymensing Medical College, Mymensing	Obst and Gynae
084-7633	Sonia Jesmin	Rangpur Medical College, Rangpur	Obst and Gynae
084-7641	Mst. Rebeka Khanam	Rajshahi Medical College, Rajshahi	Obst and Gynae
084-7653	Shohela Perveen	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7663	Shereen Yousuf	Bangladesh Medical College, Dhaka	Obst and Gynae
084-7680	Nasima Begum	Faridpur Medical College, Faridpur	Obst and Gynae
084-7699	Nasima Rahman	Rajshahi Medical College, Rajshahi	Obst and Gynae
084-7703	Sabera Sultana Biswas	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7705	Banika Biswas	MAG Osmani Medical College, Sylhet	Obst and Gynae
084-7712	Dilara Parveen	Comilla Medical College, Comilla	Obst and Gynae
084-7713	Shamima Akhter	Chittagong Medical College, Chittagong	Obst and Gynae
084-7716	Kohinoor Ahmed	Dhaka Medical College, Dhaka	Obst and Gynae
084-7717	Mahmuda Afrin Tinni	Rajshahi Medical College, Rajshahi	Obst and Gynae
084-7722	Sanjida Ahmed	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7728	Farzana Islam	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
084-7730	Tanjila Karim	Institute of Applied Health Science, under USTC, Chittagong	Obst and Gynae

Roll No.	Name	From where graduated	Subject
084-7750	Sufia Khatun	Dinajpur Medical College, Dinajpur	Obst and Gynae
084-7753	Rawshan Ara Sultana	Dhaka Medical College, Dhaka	Obst and Gynae
084-7757	Most. Salma Akhtar Zahan	Rangpur Medical College, Rangpur	Obst and Gynae
084-7758	Bilkis Ferdous	Faridpur Medical College, Faridpur	Obst and Gynae
084-7779	Shamima Yasmin	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7792	Uma Dev	Chittagong Medical College, Chittagong	Obst and Gynae
084-7803	Soneya Chowdhury	Chittagong Medical College, Chittagong	Obst and Gynae
084-7809	Mahbuba Akhter	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
084-7845	Mst. Tahmina Parvin	Rangpur Medical College, Rangpur	Obst and Gynae
084-7874	Nazia Sultana	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7884	Serajoom Munira	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7887	Khurshid Jahan	Dhaka Medical College, Dhaka	Obst and Gynae
084-7894	Marlina Roy	Mymensing Medical College, Mymensing	Obst and Gynae
084-7900	Amena Begum	Dhaka National Medical College, Dhaka	Obst and Gynae
084-7917	Chandana Saha	Mymensing Medical College, Mymensing	Obst and Gynae
084-7924	Tabenda Akter	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7926	Nazmun Nahar	MAG Osmani Medical College, Sylhet	Obst and Gynae
084-7931	Rokeia Akter	Shaheed Ziaur Rahman Medical College, Bogra	Obst and Gynae
084-7933	Amena Begum	Sir Salimullah Medical College, Dhaka	Obst and Gynae
084-7941	Meherin Majeed	Dhaka Medical College, Dhaka	Obst and Gynae
084-7956	Syeda Yeasmin Akter	Khulna Medical College, Khulna	Obst and Gynae
084-7958	Mst. Dilraj Banu	Rangpur Medical College, Rangpur	Obst and Gynae
084-7965	Nazma Mazumder	MAG Osmani Medical College, Sylhet	Obst and Gynae
084-7983	Homayra Akter	Rajshahi Medical College, Rajshahi	Obst and Gynae
084-7990	Bipul Biswas	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
084-7995	Rummana Zafrin	Chittagong Medical College, Chittagong	Obst and Gynae
084-8006	Rebeka Sultana	Dhaka Medical College, Dhaka	Obst and Gynae
084-8013	Halima Akter	Mymensing Medical College, Mymensing	Obst and Gynae
084-8021	Saiqa Rehnuma	Mymensing Medical College, Mymensing	Obst and Gynae
084-8030	Rezwana Mirza	Comilla Medical College, Comilla	Obst and Gynae
084-8040	Zakia Sultana	Rangpur Medical College, Rangpur	Obst and Gynae
084-8063	Lipi Paul	Dhaka Medical College, Dhaka	Obst and Gynae
084-8070	Ummul Sifat Rizwana Rahman	Armed Forces Medical College, Dhaka	Obst and Gynae
084-8074	Nilufa Sultana	Mymensing Medical College, Mymensing	Obst and Gynae
084-8086	Zabun Nesa	Dhaka Medical College, Dhaka	Ophthalmology
084-8089	Taslima Mazid	Mymensing Medical College, Mymensing	Ophthalmology
084-8094	Mohammad Farid Hossain	Sher-E-Bangla Medical College, Barisal	Ophthalmology
084-8097	Al-Mamun	Dhaka Medical College, Dhaka	Ophthalmology
084-8103	Israt Jahan	Rajshahi Medical College, Rajshahi	Ophthalmology
084-8110	Sadeq Ahmed	Dhaka Medical College, Dhaka	Ophthalmology
084-8111	Farzana Afzal	Bangladesh Medical College, Dhaka	Ophthalmology
084-8112	Mahziba Rahman Chowdhury	Bangladesh Medical College, Dhaka	Ophthalmology
084-8114	Md. Hasanuzzaman	Dhaka Medical College, Dhaka	Ophthalmology
084-8116	Mohammad Masudul Hasan	Rajshahi Medical College, Rajshahi	Ophthalmology
084-8117	Rahima Bhuiya	Sher-E-Bangla Medical College, Barisal	Ophthalmology
084-8118	A.K.M. Mamunur Rahman	Sir Salimullah Medical College, Dhaka	Ophthalmology
084-8121	Mst Mousumi Akhtar	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
084-8123	Md. Asaduzzaman	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
084-8124	Binay Kumar Das	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
084-8126	Mohammad Kamrujjaman	Chittagong Medical College, Chittagong	Oral and Maxillofacial Surgery

Roll No.	Name	From where graduated	Subject
084-8133	Md. Nazmul Hasan Khandker	Dhaka Dental College, Dhaka	Oral and Maxillofacial Surgery
084-8145	Md. Abdul Bari Mollick	Dhaka Dental College, Dhaka	Orthodontics and Dentofacial Orthopaedics
084-8158	Mohammad Shaharior Arafat	MAG Osmani Medical College, Sylhet	Otolaryngology
084-8162	Timir Kumar Debnath	Dinajpur Medical College, Dinajpur	Otolaryngology
084-8163	Nurul Karim Chowdhury	Sher-E-Bangla Medical College, Barisal	Otolaryngology
084-8166	Mohammad Mostafizur Rahman	Sir Salimullah Medical College, Dhaka	Otolaryngology
084-8169	Md. Shariful Islam	Sher-E-Bangla Medical College, Barisal	Otolaryngology
084-8171	Ashim Kumar Biswas	Dinajpur Medical College, Dinajpur	Otolaryngology
084-8172	Muddassir Mahmud	MAG Osmani Medical College, Sylhet	Otolaryngology
084-8173	Md. Monsur Alam	Comilla Medical College, Comilla	Otolaryngology
084-8174	Ashik Ikbal	Sir Salimullah Medical College, Dhaka	Otolaryngology
084-8176	Mohammad Hanif	Sir Salimullah Medical College, Dhaka	Otolaryngology
084-8180	Muhammad Rafiqul Islam	Mymensing Medical College, Mymensing	Otolaryngology
084-8182	Md. Mashiur Rahman	Faridpur Medical College, Faridpur	Otolaryngology
084-8187	Md. Shah Jamal Mullick	Mymensing Medical College, Mymensing	Otolaryngology
084-8203	Sushama Roy	Dhaka Medical College, Dhaka	Paediatrics
084-8215	Md. Mojibur Rahman	Faridpur Medical College, Faridpur	Paediatrics
084-8217	Md. Shafiul Alam	Sher-E-Bangla Medical College, Barisal	Paediatrics
084-8221	Fahmuda Akhter	Bangladesh Medical College, Dhaka	Paediatrics
084-8233	Fauzia Nasreen	Mymensing Medical College, Mymensing	Paediatrics
084-8239	Sajani Islam	Mymensing Medical College, Mymensing	Paediatrics
084-8243	Most. Samsun Nahar Sumi	Rangpur Medical College, Rangpur	Paediatrics
084-8247	Shyamal Sarker	Chittagong Medical College, Chittagong	Paediatrics
084-8262	Maher Akther	MAG Osmani Medical College, Sylhet	Paediatrics
084-8267	Mohammad Rafiqul Islam	Chittagong Medical College, Chittagong	Paediatrics
084-8314	Urmi Rahman	Dhaka Medical College, Dhaka	Paediatrics
084-8319	Abu Zafar Muhammad Khairul Anam	Dhaka Medical College, Dhaka	Paediatrics
084-8322	Lazina Sharmin	Mymensing Medical College, Mymensing	Paediatrics
084-8334	Abdullah Al-Amin	Dhaka Medical College, Dhaka	Paediatrics
084-8341	Soma Halder	Sher-E-Bangla Medical College, Barisal	Paediatrics
084-8342	Smrity Roy	Mymensing Medical College, Mymensing	Paediatrics
084-8345	Nayeema Sadia	Chittagong Medical College, Chittagong	Paediatrics
084-8352	Subir Nandy	Mymensing Medical College, Mymensing	Paediatrics
084-8370	Sanjita Biswas	MAG Osmani Medical College, Sylhet	Paediatrics
084-8386	Tania Islam	Rangpur Medical College, Rangpur	Paediatrics
084-8387	Ashith Chandra Das	Chittagong Medical College, Chittagong	Paediatrics
084-8388	Nure Ishrat Nazme	Sir Salimullah Medical College, Dhaka	Paediatrics
084-8393	Muhammad Firoze Hasan	Rangpur Medical College, Rangpur	Physical Medicine & Rehabilitation
084-8394	Mohammad Amanul Hoque	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
084-8397	Md. Nuruzzaman Khandaker	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
084-8398	Monzur Ahmed	Dhaka Medical College, Dhaka	Physical Medicine & Rehabilitation
084-8402	Fahmida Ahmed	Sir Salimullah Medical College, Dhaka	Psychiatry
084-8404	Umme Salma Talukder	Medical College for Women and Hospital, Dhaka	Psychiatry
084-8405	Jesmin Akhter	Dhaka Medical College, Dhaka	Psychiatry
084-8406	Mohammad Nazrul Islam	MAG Osmani Medical College, Sylhet	Radiology & Imaging
084-8411	A K M Sharifur Rahman	Sir Salimullah Medical College, Dhaka	Radiology & Imaging
084-8415	Sura Jukrup Momtahena	MAG Osmani Medical College, Sylhet	Radiotherapy
084-8417	Arman Reza Chowdhury	Rangpur Medical College, Rangpur	Radiotherapy
084-8418	Arunangshu Das	Mymensing Medical College, Mymensing	Radiotherapy

Roll No.	Name	From where graduated	Subject
084-8451	Mohammad Jamil Hossain	Rajshahi Medical College, Rajshahi	Surgery
084-8455	Mohammad Abu Hanif	Sir Salimullah Medical College, Dhaka	Surgery
084-8456	Md. Ershad-Ul-Quadir	Mymensing Medical College, Mymensing	Surgery
084-8458	Md. Omar Faruk	Rajshahi Medical College, Rajshahi	Surgery
084-8484	Mohammad Rajibul Haque Talukder	Sir Salimullah Medical College, Dhaka	Surgery
084-8497	Md. Shohorab Hossain	Rangpur Medical College, Rangpur	Surgery
084-8520	Khadija Rahman	Dhaka Medical College, Dhaka	Surgery
084-8522	Md Rashidul Hoq	Institute of Applied Health Science, under USTC, Chittagong	Surgery
084-8554	Md. Rezaul Kabir	Chittagong Medical College, Chittagong	Surgery
084-8555	Mosammat Mira Pervin	Chittagong Medical College, Chittagong	Surgery
084-8564	Md Rafiqul Islam	Rangpur Medical College, Rangpur	Surgery
084-8566	Abul Bashar Shahriar Ahmed	Bangladesh Medical College, Dhaka	Surgery
084-8575	Mohammad Mahbub Elahi	Dhaka Medical College, Dhaka	Surgery
084-8586	Mitu Debnath	Sher-E-Bangla Medical College, Barisal	Surgery
084-8594	Biplab Biswas	Khulna Medical College, Khulna	Surgery
084-8608	Mohammed Aminul Islam	Rangpur Medical College, Rangpur	Surgery
084-8610	Sharif Mushfaqur Rahman	Dhaka Medical College, Dhaka	Surgery
084-8624	Mohammad Sanuar Rahman	Comilla Medical College, Comilla	Surgery
084-8640	Hasan Ul Banna	Comilla Medical College, Comilla	Surgery
084-8659	Md. Abul Kalam Azad	Sir Salimullah Medical College, Dhaka	Surgery
084-8662	Najma Mahboob	Chittagong Medical College, Chittagong	Surgery
084-8666	Md. Najmul Haque	Sir Salimullah Medical College, Dhaka	Surgery
084-8667	Md. Shariful Alam	Comilla Medical College, Comilla	Surgery
084-8675	Muhammed Alam	Mymensing Medical College, Mymensing	Surgery
084-8681	Gazi Muhammad Salahuddin	Dhaka Medical College, Dhaka	Surgery
084-8708	Tanny Tarafder	Sir Salimullah Medical College, Dhaka	Surgery
084-8713	Md. Nurul Amin Bhuiyan	Chittagong Medical College, Chittagong	Surgery
084-8722	Kazi Shafiqul Alam	Chittagong Medical College, Chittagong	Surgery
084-8729	S. M. Ishtiaque Ali	Sir Salimullah Medical College, Dhaka	Surgery

The following candidates satisfied the Board of Examiners and are declared to have passed the MCPS Examinations held in January, 2014 subject to confirmation by the council of Bangladesh College of Physicians and Surgeons

Roll No.	Name	From where graduated	Subject
084-9009	Amena Akhtar	Rangpur Medical College, Rangpur	Clinical Pathology
084-9010	Paramananda Roy	Mymensing Medical College, Mymensing	Clinical Pathology
084-9011	Azmery Hossain Moly	Medical College for Women and Hospital, Dhaka	Clinical Pathology
084-9017	Md. Wahiduzzaman	Rangpur Dental College, Rangpur	Dental Surgery
084-9018	Mohammad Tofazzal Hossan	Dhaka Dental College, Dhaka	Dental Surgery
084-9031	Dilruba Aktar	Jalalabad Ragib-Rabeya Medical College, Sylhet	Dermatology and Venereology
084-9046	Rozina Afroz	Sir Salimullah Medical College, Dhaka	Dermatology and Venereology
084-9047	Tahmina Sultana	Comilla Medical College, Comilla	Dermatology and Venereology
084-9052	Md. Harun-Ur- Rashid	Mymensing Medical College, Mymensing	Forensic Medicine
084-9054	Nashid Tabassum Khan	Dhaka Medical College, Dhaka	Forensic Medicine
084-9056	Mohammad Saifule Islam	Rangpur Medical College, Rangpur	Forensic Medicine
084-9061	Monoj Sinha	Chittagong Medical College, Chittagong	Medicine
084-9095	Mohammad Mizanur Rahman	Dhaka Medical College, Dhaka	Medicine
084-9120	A. K. M. Shafiqul Islam	Dinajpur Medical College, Dinajpur	Medicine
084-9152	Nur Mohammad	Chittagong Medical College, Chittagong	Medicine

Roll No.	Name	From where graduated	Subject
084-9162	S. M. Abdul Munim	Sher-E-Bangla Medical College, Barisal	Medicine
084-9168	Kamalesh Chandra Basu	Sir Salimullah Medical College, Dhaka	Medicine
084-9174	Sukanta Chandra Das	Dhaka Medical College, Dhaka	Medicine
084-9190	A. K. M. Kamruzzaman	Chittagong Medical College, Chittagong	Medicine
084-9231	Ashfaque Ahmed Siddique	Dhaka Medical College, Dhaka	Medicine
084-9254	Tanzina Akhter	Sir Salimullah Medical College, Dhaka	Medicine
084-9342	Mohsina Haider	Dhaka Medical College, Dhaka	Obst and Gynae
084-9493	Sharmin Abbasi	Ibrahim Medical College, Dhaka	Obst and Gynae
084-9523	Mossammat Shoheli Nasrin	Mymensing Medical College, Mymensing	Ophthalmology
084-9531	Abdullah-Al-Mamun	Institute of Applied Health Science, under USTC, Chittagong	Otolaryngology
084-9579	Mohammad Ala Uddin	MAG Osmani Medical College, Sylhet	Paediatrics
084-9587	Fahmida Islam	Chittagong Medical College, Chittagong	Paediatrics
084-9597	Tarannum Khondaker	Z.H. Sikder Women's Medical College, Dhaka	Paediatrics
084-9608	Shayla Imam Kanta	Rangpur Medical College, Rangpur	Paediatrics
084-9613	Kazi Farzana Abedin	MAG Osmani Medical College, Sylhet	Paediatrics
084-9617	Nasim Jahan	Z.H. Sikder Women's Medical College, Dhaka	Psychiatry
084-9618	A. F. M. Riaz Rony	Dhaka Medical College, Dhaka	Psychiatry
084-9619	Shahriar Faruque	Mymensing Medical College, Mymensing	Psychiatry
084-9625	Md. Mamun Or Rashid	Chittagong Medical College, Chittagong	Radiology & Imaging
084-9626	Md. Tanshed Arafat	Mymensing Medical College, Mymensing	Radiology & Imaging
084-9627	Shafayat Bin Mollah Mosharraf	Sir Salimullah Medical College, Dhaka	Radiology & Imaging
084-9628	Muhammad Shoyab	Sir Salimullah Medical College, Dhaka	Radiology & Imaging
084-9629	Mariyam Sultana	Mymensing Medical College, Mymensing	Radiology & Imaging
084-9631	Mostofa Zahid Kamal	Mymensing Medical College, Mymensing	Surgery
084-9640	Most Bilkis Fatema	Rajshahi Medical College, Rajshahi	Surgery

FROM THE DESK OF EDITOR in CHIEF

(J Banagladesh Coll Phys Surg 2014; 32: 120)

Dear fellows

The Journal of BCPS team would like to start by thanking you for your relentless support. Just as we have promised the journal website is now fully functional and available for your viewing. Your frequent visit to the site will increase the number of hits and help us in attaining our goal to be indexed in the MEDLINE. Alongside we would also require original articles of international standard to reach that target. For your convenience we have made online submission of articles through our website www.journalbcps.org this will help speed up the whole process.

Professor H.A.M. Nazmul Ahasan
Editor-in-Chief
Journal of Bangladesh College of Physicians
and Surgeons

Obituary

(J Banagladesh Coll Phys Surg 2014; 32: 121)

The following Fellows who died 2014

Professor M.A. Hai

Professor M.A. Hai died on 5th February, 2014. He own fellowship without examination in Physiology, 1993from Bangladesh College of Physicians and Surgeons (BCPS)**.**

Professor A.H.M. Ahsanullah

Professor A.H.M. Ahsanullah died on 11th February, 2014. He passed fellowship in Neuro-Surgery in January, 1969 from Bangladesh College of Physicians and Surgeons (BCPS). He was the past president of BCPS.