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MH Samorita Medical College Journal

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MH SAMORITA HOSPITAL & MEDICAL COLLEGE, DHAKA
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Web: www.mhsamorita.edu.bd Email: mhsmc@yahoo.com

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Contents

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INFORMATION FOR AUTHORS

Manuscript Preparation and Submission

Guide to Authors

MH Samorita Medical College Journal provides rapid publication (twice in a year) of articles in all areas of different subjects. The Journal welcomes the submission of manuscripts that meet the general criteria of significance and scientific excellence.

The manuscripts should be submitted addressing Editor-in-Chief.

The Journal of MH Samorita Medical College only accepts manuscripts submitted as triplicate hard copy with a soft copy.

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 (**International or National)** or any other publisher.

The submitting (Corresponding) author is responsible for ensuring that the submitting article has been signed by all the co-authors. 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 board officially establishes the date of receipt. Further correspondence and proofs are 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 papers should be addressed to Editor-in-Chief (MH Samorita Med Coll J)

The cover letter

Cover letter is expected to be submitted along with manuscript. Use the cover letter to explain why the paper should be published in the Journal of MH Samorita Medical College. The cover letter should include the corresponding author's full address, telephone/ fax numbers and e-mail address.

Ethical aspects

- Ethical aspect of the study is considered very carefully 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 would be blackened out.

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 MH Samorita Medical College Journal (MHSMCJ) 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.

Article Types

Four types of manuscripts may be submitted.

Editorials: It should preferably cover a single topic of common 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 and its volume should **not exceed 5000 words** or equivalent space including title, summary/abstract, main body, references, table(s) and figure(s).

Review Articles: Submissions of reviews 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) and should **not exceed 5000 words**. It should be focused and must be up to date.

Case Reports: This should cover uncommon and/or interesting cases and should **not exceed 1000** words or equivalent space.

Review Process

All manuscripts are initially screened by editor and sent to selective reviewers. Reviewers are requested to return comments to editor within 3 weeks. On the basis of reviewers' comments the editorial board decides whether the articles are accepted or send for re-review the manuscripts. The MH Samorita Med Coll J editorial board tries to publish the manuscript as early as possible fulfilling all the rigorous standard journal needs.

I. Preparing a Manuscript for Submission to MH Samorita Med Coll J

Editors and reviewers spend many hours reading and working on manuscripts, and therefore appreciate receiving manuscripts that are easy to read and edit. The following information provides guidance in preparing manuscripts for the journal.

I A. 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 :

- Title page
- Abstract
- Main body/Text: Introduction, Materials and Methods, Results, Discussion and conclusion (For an original article/ Systematic review)
- Acknowledgement
- References
- 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. General Principles

- The text of observational and experimental articles is usually (but not necessarily) divided into the following sections: Introduction, Materials and Methods, Results, and Discussion(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.
- Authors need to work closely with editors in developing or using the publication formats and should submit supplementary electronic material for peer review.
- Double-spacing all portions of the manuscript— including the title page, abstract, text, acknowledg- ments, 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. 2. Title Page

The title page should have the following information:

- The title should be brief, relevant and self explanatory. It should reflect the content of the article and should include all information that will make electronic retrieval of the article easy. Subtitles should not be used unless they are essential.
- Title should not be phrased as questions.
- The names of the authors should appear below the title that should include full names of all authors **(no initial).**

Example: Md MA Hamid **(correct form);** Hamid MA **(incorrect).**

The affiliations and full addresses of all authors should be mentioned in the title page.

- 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.
- 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.
- Source(s) of support in the form of grants, equipment, drugs, or all of these.

I A. 3. Abstract

Original Article: Structured abstracts are essential for original research. Structured abstract includes introduction, objective(s), materials and methods, results and conclusion. Should be limited to 250 words. The abstract should provide the introduction of the study and blinded state and should mention the study's purpose, basic procedures including selection of study subjects or laboratory animals, main findings (giving specific effect sizes and their statistical significance, if possible) and the principal conclusion. Because abstracts are the only substantive portion of the article indexed in many electronic databases, and the only portion that many readers read, it should accurately reflect the content of the article; so, authors need to be careful about that.

Review Article: is expected to contain background, objective(s), main information and conclusion in brief form. Without any subheading the content should be described in a single paragraph.

Case Study: needs to have background, case summary and conclusion. The content should be described in a single paragraph.

Do not put references in the abstract.

I A. 4. Main body

I A. 4 a) Original article

The body of the text should be divided into the following sections: i) Introduction, ii) Materials and methods, iii) Results, iii) Discussion and iv) Conclusion.

i) Introduction

Should not exceed **500 words**. This section includes background of the problem (that is, the nature of the problem and its significance). It should be very specific, identify the specific knowledge in the aspect, reasoning and what the study aim to answer. Only pertinent primary references should be provided and no data or conclusions should be included from the work to be reported. **Justificatio**n of the study and its **objective(s)** should be mentioned at the end of this section. All information given in this section must have references that to be listed in the reference section.

ii) Materials and methods

The Methods section should be written in such way that another researcher can replicate the study. The type of study (study design), study period, sampling technique, sample size, study population, data collection technique and tool as well as data handling, processing and data analysis should be briefly mentioned in this section.

ii a) Selection and Description of Participants

Describe selection of the observational or experimental participants (patients or laboratory animals, including controls) clearly, including eligibility (inclusion) 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 etc. 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.

ii b) Technical Information

- Describe methods, apparatus (give the manufacturer's name and address in parentheses), and procedures in sufficient detail to allow others to reproduce the results.
- Cite references to established methods, including statistical methods. 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.
- For a systematic review article include a section describing the methods used for locating, selecting, extracting, and synthesizing data. These methods should also be summarized in the abstract.

ii c) Statistics

- Describe statistical methods with enough detail to enable a know- ledgeable 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).
- Cite references for the design of the study and statistical methods (standard for the work) when possible.
- Define statistical terms, abbreviations, and most symbols.
- Specify the computer software used.

iii) Results

Results should be described in past tense.

- Present results in logical sequence in the text, tables, figures and illustrations, giving the main or most important findings first. Maintain the sequence of results with the specific objectives selected earlier.
- Do not repeat all the data in the tables or illustrations in the text; emphasize or summarize only the most important observations.
- When data are summarized in the result 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 (relevant to objectives) and to assess supporting data. Use graphs as an alternative to tables with many entries; do not duplicate data in figures (graphs/ charts) and tables. **Example:** Age range of the studied respondents should be appeared **either in table or in figure**.
- Avoid nontechnical uses of technical terms in statistics, such as "random" (which implies a randomizing device), "normal," "significant," "correlations," and "sample."

iv) Discussion

The discussion must be described in **past tense.** This section should reflect the author's comments on the results.

- Emphasize the new and important aspects of the study and the conclusions that follow 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 those findings.
- Compare and contrast the results with other relevant studies and potential argument for discrepancy and consistency should be given here.
- 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, 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.

v) Conclusion

It should be described in **present tense**. Conclusion should be the main message and the authors' impression from the results of the study. The article should be concluded briefly (**not more than 100 words**). Recommendation(s) can also be included in this section which should not exceed 30 words.

I A. 4 b) Review article

For a systematic review or meta-analysis the body of text should be divided into the following sections (Like an original article): i) Introduction, ii). Materials and methods, iii) Findings/Results, iii a) Main information about the topic, iv) Discussion and v) Conclusion. For a general review article section No. ii (Materials and methods) and iii (Findings/Results) iv) (Discussion) are not relevant. So, for a general review article section No. i). Introduction, iii a). Main Information about the Topic and v). Conclusion are required.

i) **Introduction**: should not exceed **500 words**. This section will include background of the topic. At the end of the review, why the author want to publish the topic on the article ie., the objective should be mentioned.

ii) **Material and methods**: How the review was done, what sorts of articles were searched, how they were searched, the total number of articles reviewed should be mentioned here. This section is not required for a general review article.

iii) **Results/findings**: The findings on the topic after reviewing the articles should be compiled, analysed and described here like an original research article. This section is not required for a general review article.

iii a) **Main Information about the Topic**: The main information about the topic should be described and discussed elaborately with the help of published literatures in this section but the subtitles should be relevant to the topic(Title) for a general review article. This section may not be required for a systematic review or meta-analysis.

iv) **Conclusion:** The article should be concluded briefly (**not more than 100 words**).

I A. 4 c) Case Report

The body of the text should be divided into the following sections: i) Introduction, ii) Case Report (Description of the case), iii) Discussion and iv) Conclusion.

i) **Introduction**: A brief description should be given on the topic of the case with the help of published literatures.

ii) **Case Report**

- The findings (history, clinical examination and investigations) should be described here.
- Management (if any) can also be given.

iii) **Discussion**

- The discussion should be started by briefly summarizing the main findings of the case reported, then possible explanations for those findings should be explored.
- The findings of the case should be compared with other relevant studies and potential argument for discrepancy and consistency should be given here.

iv) **Conclusion**

- The article should be concluded briefly (**not more than 100 words**).
- The main findings of the reported case should be emphasized which the readers can consider as a clue to suspect a diagnosis for a rare case in future.

I A. 5. Acknowledgement

Acknowledge advisor(s) and/or any one who helped the researcher(s)

- Technically
- Intellectually
- Financially

I A. 6. References

I A. 6 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.
- Abstracts should not be used 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 be cited in the text as "unpublished observations" with written permission from the source.
- Citing a "personal communication" should be avoided 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. 6 b) Reference Style and Format

➢ **Reference Style**

Author should follow **Vancouver style**.

- R**eference list** should appear at the end of the article and should be numbered consecutively in the order as they are cited in the text, which is done by **superscript** (single press of 'ctrl shift +') in numerical form **(citation number).**
- When **multiple references** are cited at a given place in the text, use a **hyphen** to join the first and last numbers that are **inclusive**. Use **commas** (without spaces) to separate **non-inclusive** numbers in a multiple citation.

  Example: 2,3,4,5,7,10,12 are abbreviated to **(2-5,7,10,12**).
- **Do not** use a hyphen if there is no citation numbers in between 2 numbers that support your statement.

  Example: 1-2 (**in correct form).** 1,2**(correct form)**
- As a general rule, citation numbers in the text should be placed **outside full stops and commas**, inside colons and semicolons (applicable for any part of the document).

  Example: Masud Alam,1 Selim Khan[2]

  Example: Over the past decades public health relevance of mental health condition 'in children and adolescents has been of growing concern'.[1-3,5,6]
- 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.

➢ **Reference Format**

1. Citing a Book

The essential details required are (in order):

1.1 Name/s of author/s, editor/s, compiler/s or the institution responsible.

- Where there are **6 or less authors** you must list **all authors**.
- Where there are **7 or more author**s, only the **first 6 are listed** and **add "et al"** (after a **comma**).
- Put a comma and 1 space between each name. The last author must have a full-stop after their initial(s).

  Format: surname (**1 space**) initial/s (**no spaces or punctuation between initials**) (**full-stop OR if further names comma, 1 space**)

  Example: Smith AK, Jones BC, Bloggs TC, Ashe PT, Fauci AS, Wilson JD, et al.
- **When author/s is/are editor/s :**Follow the same methods used with authors but use the word **"editor"** or **"editors"** in full after the name/s. The word editor or editors must be in small letter. (**Do NOT** confuse with "ed."used for edition.)

  Example: Millares M, editor. Applied drug information: strategies for information management. Vancouver (WA): Applied Therapeutics Inc;1998.

Sponsored by institution, corporation or other organization (including PAMPHLET)

E**xample**: Australian Pharmaceutical Advisory Council. Integrated best practice model for medication management in residential aged care facilities. Canberra: Australian Government Publishing Service; 1997.

1.2. Title of publication and subtitle if any

- Italics or underlining should be avoided.
- Only the first word of the titles (and words that normally begin with a capital letter) should be started with capital letter (except proper noun).

Format: title (**full-stop, 1 space**)

Example: Harrison's principles of internal medicine.

Example: Physical pharmacy: physical chemical principles in the pharmaceutical sciences.

Example: Pharmacy in Australia: the national experience.

1.3. Edition (other than the first)

Number of edition **other than first one** should be mentioned as **2nd, 3rd,10th ed.**

Example: Blenkinsopp A, Paxton P. Symptoms in the pharmacy: a guide to the management of common illness. 3rd ed. Oxford: Blackwell Science; 1998.

1.4. Place of publication (if there is more than one place listed, use the first one)

- The place name should be written in full.
- If the place **name is not well known**, add a comma, 1 space and the state or the country for clarification. For places in the USA, add after the place names the 2 letter postal code for the state. This must be in upper case. eg. Hartford (CN): (where CN=Connecticut).

Format: place of publication (**colon, 1 space**)

Example: Hartford (CN):

Example: Texas (NSW):

Example: Kyoto (Japan):

1.5. Publisher

The publisher's name should be spelled out in full.

Format: publisher (**semi-colon, 1 space**)

Example: Australian Government Publishing Service;

Example: Raven Press;

Example: Williams & Wilkins;

1.6. Year of publication

Format: year (full-stop, add 1 space if page numbers follow).

Example: 1999.

Example: 2000. p. 12-5.

1.7. Page numbers (if applicable).

- Abbreviate the word "page" to "p.".

Note: do not repeat digits unnecessarily

Format: p (**full-stop, 1 space**) page numbers (**full-stop**).

Example: p. 122-9 (**correct**); p. 122-129 (**incorrect**).

Example: p. 1129-57 (**correct**); p. 1129-157 (**incorrect**).

Example of citing a book: Lodish H, Baltimore D, Berk A, Zipursky SL, Matsudaira P, Darnell J. Molecular cell biology. 3rd ed. New York: Scientific American; 1995.

(*Name/s. Title. Edition(other than first). Place of publication: Publisher; year of publication. p. Page no*)

2. Citing a Chapter in an Edited Book (to which a number of authors have contributed)

- Name/s of author of the chapter
- Title of chapter followed by, In:
- Editor
- Title of book
- Series title and number (if part of a series)
- Edition (if not the first edition)
- Place of publication (if there is more than one place listed, use the first named)
- Publisher
- Year of publication
- Page numbers

(*Title of Chapter. In: Editor(s). Title of book and number. Edition (other than first). Place of publication: Publisher; year of publication. p. Page no*)

Example of citing a chapter in an edited book:

Porter RJ, Meldrum BS. Antiepileptic drugs. In: Katzung BG, editor. Basic and clinical pharmacology. 6th ed. Norwalk (CN): Appleton and Lange; 1995. p. 361-80.

3. Citing a Journal Article from a Print source

The essential details required are (in order):

- **Name/s of author/s of the article.**
  See step 1 of "Citing a book" for full details.
- **Title of article.**
  See step 2 of "Citing a book" for full details.

Example: Validation of an immunoassay for measurement of plasma total homocysteine.

- **Name of journal (abbreviated).**

- Abbreviate the name of the journal according to the style used in Medline.
- A list of abbreviations can be found at: http://w**ww.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals**

  Note: No punctuation marks are used in the abbreviated journal name.

Format: journal title abbreviation (1 space)

Example: Bang J Psychiatry

- **Year of publication (month or day should be omitted).**

Format: year (**semi-colon, one space**)

Example: 1996; 12(5): 127-33.

- **Volume number (and issue/part)**

  Format: volume number (**colon, one space**)

Example: 1996; 12(5): 127-33. Or
1996; 18: 1237-8.

- **Page numbers**

Note: Do not repeat digits unnecessarily

Format: page numbers (full-stop)

Example: 5310-5.

Example of citing a journal: Russell FD, Coppell AL, Davenport AP. In vitro enzymatic processing of radiolabelled big ET-1 in human kidney as a food ingredient. Biochem Pharmacol 1998; 55(5): 697-701.

Name(s). Title. Name of the Journal Year of publication; Volume Number (Session/Issue Number): Page Number.

- **No author given in article**

  Example: Coffee drinking and cancer of the pancreas [editorial]. BMJ 1981; 283: 628.

- **Journals with parts and/or supplements**

Examples

- **Volume with supplement**

  Environ Health Perspect 1994; 102Suppl 1: 275-82.

- **Issue with supplement**

  SeminOncol 1996: 23(1 Suppl 2): 89-97.

- **Volume with part**

  Ann ClinBiochem 1995; 32(Pt 3): 303-6.

4. Citing a Journal Article from Internet and Other Electronic Sources

This includes software and internet sources such as web sites, electronic journals and databases.

The **basic form** of the citations **follow the principles listed for print sources** (see above).

In the case of sources that may be subject to alteration it is important to acknowledge the **Date The Information Was Cited.** This is particularly true for web sites that may disappear or permit changes to be made and for CD-ROMS that are updated during the year.

4.1. Citing a Journal Article from the Internet

Note: Follow the same procedure for citing print journals as for electronic journals regarding date, volume pages and journal title

Format: Author/s (full-stop after last author, 1 space) **Title of article** (full-stop, 1 space)

Abbreviated title of electronic journal (1 space) **[serial online]** (1 space) **Publication year** (1space) **month(s)** - if available (1 space) **[cited year month (abbreviated) day]** - in square brackets (semi colon, 1 space) **Volume number** (no space) **Issue number** if applicable in round brackets (colon) **Page numbers or number of screens** in square brackets (full-stop, 1 space) **Available from** (colon, 1 space) URL:**URL address underlined**

Examples:

- Morse SS. Factors in the emergence of infectious disease. Emerg Infect Dis [serial online] 1995 Jan-Mar [cited 1999 Dec 25]; 1(1):[24 screens]. Available from:URL: http://www/cdc/gov/ncidoc /EID/eid.htm
- Garfinkel PE, Lin E, Goering P. Should amenorrhoea be necessary for the diagnosis of anorexia nervosa? Br J Psych [serial online] 1996 [cited 1999 Aug 17]; 168(4):500-6. Available from: URL:http://biomed. niss.ac.uk

4.2. Citing a Journal Article from WWW site

(If the author is not documented, the title becomes the first element of the reference.)

Format: Author (full-stop after last author, 1 space) **Title** (full-stop, 1 space) **[Online]** (full stop, 1 space) **Publication Year** (1 space) **[cited year month (abbreviated) day]** (semi colon) **Number of screens in square brackets or pages** (full-stop, 1 space) **Available from** (colon, 1 space)

URL: (no space) **URL address underlined**

Note: The number of screens is not necessary. Put a semi colon and 1 space after the cited date if no pages or screen numbers are listed.

When the date is approximated, indicate that by following the date with a question mark and inserting the statement in square brackets. Eg. [2001?]

Examples: National Organization for Rare Diseases [Online]. 1999 Aug 16 [cited 1999 Aug 21]; Available from: URL:http://www.rare diseases.org/

Royal College of General Practitioners.The primary health care team.[Online]. 1998 [cited 1999 Aug 22];[10 screens]. Available from:URL: http:// ww. rcgp.org.uk/informat/publicat/rcf0021.htmZand J. The natural pharmacy: herbal medicine for depression [Online]. [1999?] [cited 2001 Aug 23];[15 screens]. Available from:

URL:http://www.healthy.net/asp/templates/Article.asp?PageType=Article&Id=920

Important Points For Reference List

- For **online material**, please cite the **URL**, together with the **date you accessed** the website
- **Online journal** articles can be cited using the Digital Object Identifier **(DOI)** number

Samples of Reference List

A list of references contains details of those works cited in the text.

The references are listed in the same numerical order as they appear in the body of the text

1. Getzen TE. Health economics: fundamentals and flow of funds. New York (NY): John Wiley & Sons; 1997.
2. Millares M, editor. Applied drug information: strategies for information management. Vancouver, WA: Applied Therapeutics, Inc.; 1998.
3. Australian Government Publishing Service. Style manual for authors, editors and printers. 5th ed. Canberra: Australian Government Publishing Service; 1994.
4. Australian Pharmaceutical Advisory Council. Integrated best practice model for medication management in residential aged care facilities. Canberra: Australian Government Publishing Service; 1997.
5. Bennett GL, Horuk R. Iodination of chemokines for use in receptor binding analysis. In: Horuk R, editor. Chemokine receptors. New York (NY): Academic Press; 1997. p. 134-48. (Methods in enzymology; vol 288).
6. Coffee drinking and cancer of the pancreas [editorial]. BMJ 1981;283:628.
7. Morse SS. Factors in the emergence of infectious disease. Emerg Infect Dis [serial online] 1995 Jan-Mar [cited 1996 Jue 5]; 1(1):[24 screens]. Available from: URL:http:// www. cdc.gov/ ncidoc/EID/eid.htm

I A. 7. Conflict of interest

All authors are requested to disclose any actual or potential conflict of interest including any financial, personal or other relationships with other people or organizations.

It is important to be consistent when you are referencing.

I A. 8. Tables and Illustrations (Figures)

I A. 8 a) Tables

- In tables, capture information concisely and display it efficiently.
- Use tables /fig that are relevant to the study.
- Try to limit the number of tables/figures.
- 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. 8 b) 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).

- Review the images of such files on a computer screen before submitting them to be sure that 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 colour, MH Samorita Med Coll J accept coloured illustration when it seems essential. This Journal publish illustrations in colour only if the author pays the additional cost. Authors should consult the editorial board of the journal about requirements for figures submitted in electronic formats.

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- **Material and Methods**
- Study Design
- Duration and place of study
- Ethical approval
- Patient consent
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- **Results**
- Clearly present the data
- Avoid data redundancy
- **Discussion**
- Avoid unnecessary explanation of someone else' work unless it is very relevant to the study
- Provide and discuss with the literatures to support the study with references.
- Mention about limitation of the study
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- Give your conclusion
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- Acknowledge any person or institution who have helped for the study
- **Reference**
- Abide by the Vancouver style
- Use reference at the end of the sentence after the full stop with superscript
- **Legends**
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- Figures

Editorial

Diabetes Mellitus: A Global Health Issue

Alim MA

Diabetes Mellitus (DM) is a major public health problem worldwide. This condition affects 415 million people and is set to escalate to 642 million by the year 2040.[1] It kills 5 million people annually worldwide and most of them prematurely.[1] Further 193 million people with diabetes remain undiagnosed due to often mild or asymptomatic nature of this condition especially in Type-2 DM.[1] It is one of the top 10 leading causes of death in USA.[2] In Bangladesh 41.2% are aware of their condition, 36.9% were treated and 14.2% controlled their condition.[3] Prevalence of Type-2 DM is 7.4%, higher in male and in urban area. Increasing trend of DM among urban and rural population is 0.05% and 0.06% respectively.[4] Eighty three percent of adult population aged 25 years or older never had their blood glucose checked and 3.9% of study population had documented diabetes in Bangladesh.[5]

Diabetes Mellitus is a metabolic disorder with profound consequences, both acute and chronic. Human cell cannot metabolize sugar properly due to defect in insulin action or secretion. So, body starts catabolic activities through breakdown of its own fat, protein and glycogen resulting in high blood sugar.[6] Important risk factors include- genetic predisposition, high rate of tobacco use, escalating rate of obesity, high prevalence of physical inactivity, first degree relative with DM, history of gestational diabetes mellitus (GDM), dyslipidaema, hypertension (HTN), cardiovascular disease, women with polycystic ovarian syndrome (PCOS) and who delivered baby weighing more than 9 pound and urban dwellers.[4,5] Rural people do not test for blood glucose level until it gets complicated. They are diagnosed incidentally.[4] Genetic and environmental factors play important role in development of this disease. In developing countries majorities of individual with DM are aged between 45-65 years, in developed countries more than 64 years. But it has no boundary. It may affect old and young, rich and poor, rural and urban dwellers.[4]

Usually, it presents with polyuria, polydypsia, polyphagia, weight loss, blurring of vision and features of complications.[4] It has biochemical diagnosis but symptoms have strong value to support the diagnosis. Fasting plasma glucose ≥7.0 mmol/L, random plasma glucose (with symptoms of diabetes) and 2 hours after 75 gm oral glucose load ≥11.1 mmol/L and Glycosylated haemoglobin (HbA_1C) ≥6.5% are diagnostic.[5]

For treatment of DM, diet and discipline have great value along with drugs. High risk people can reduce 58% risk of developing type 2 DM by losing weight through physical activity, low calorie and low fat diet.[1] Regular check up for early detection, need to know emergency medicine, regular follow up in pregnancy, good sugar control are essential to prevent long term complications. Patients have to take care of their feet to prevent ulcer.[4] Healthy diet and increased physical activity should be the first step. Decreased intake of animal fat, salt, refined food, alcohol and tobacco are important.[7] Intake of refined carbohydarte, sugar added to tea and coffee should be reduced and intake of presweetend mixes should be avoided. Consumption of cookies, cakes, sugary snacks, chocolates, candies, pastries, sweets (mishti), fizzy drinks and sweetend beverages including fruit juice should be avoided. Sugary snakcs should be replaced with fresh whole fruits and fresh vegetables. Increased intake of locally available high fibre diet, vegetables, fruits, safe water and plain milk are important. Eating variety of foods in short intervals and at least 3 main meals with 8 glasses of water per day should be practiced.

Physical activities like walking should be 5 days in a week or at least 3 days in a week for 30-45 minutes per session. Modalities like house work, gardening, walking, climbing stairs (instead of lift), exercise like fast walking, jogging, cycling, swimming should be encouraged.[7]

Properly sized shoes must always be worn during exercise to avoid injury. Feet should be washed regularly and kept dry between toes. Walking bare footed, with open sandals and tight shoes should be avoided.[3]

If patient is symptomatic, age more than 40 years should be prescribed Statin and Aspirin to avoid the risk of cardiovascular diseases if there is no contraindication.[8] Lipid profile should be checked annually. Patients should be in regular follow up for complications of disease and drugs. Initially at baseline and then measurement of blood pressure, calculation of Body Mass Index (BMI), examination of lower limb for ulcer and sensation should be done regularly. Lab investigations include urine routine examination, fasting lipid profile annually and retinal examination every 2 years.[5]

If DM remains uncontrolled, complications like blindness, renal failure, and lower limb ulcer requiring amputation may develop. Sixty eight percent people with DM die from cardiovascular diseases, 16% die of stroke.[9] Median DM duration of 4 years has shown that 52% are treated for HTN, 40% for dyslipidaemia, 37% for microalbuminuria, 5% for diabetic peripheral neuropathy.[1,2] Healthcare expenditure for people with DM is on average 2 times higher than that of the people without DM.[1] It possesses a major socioeconomic challenge.

The main problem in low and middle - income countries is the ignorance of general mass and shortage of healthcare resources. Nonetheless, even with limited resources, action can be taken to prevent the development of DM, heart attacks and strokes if resources are used population wide, for cost effective interventions and raising the awareness among general mass.

(MH Samorita Med Coll J 2019; 2(1): 1-2)

Brig. Gen. (Retd) Prof. Dr. Md. Abdul Alim
Professor and Head, Department of Medicine
MH Samorita Hospital and Medical College, Dhaka

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Effect of Structured Diabetes Education Programme on Glycaemic Control in Children and Adolescents with Diabetes

Nahar J[1], Ahmed B[2], Tayyeb S[3], Islam N[4], Zabeen B[5]

Abstract

Introduction*: Type-1 diabetes mellitus(T1DM) is the most common form of diabetes comprising over 90% of cases of childhood and adolescent diabetes. Education is the keystone of management of diabetes in children and adolescents and structured education is the key to a successful outcome.*

Objectives: *This study was done to evaluate the impact of Structured Diabetes Education Programme on metabolic control of diabetes in children and adolescents.*

Materials & Methods*: This study was conducted at the CDiC (Changing Diabetes in Children), Paediatric Diabetic Clinic, BIRDEM from March to August 2012. Before and after study design was used. One hundred twenty four registered T1DM patients with their parents/caregivers were selected randomly during an 8 weeks recruitment period (January to February 2012). They attended a structured diabetes education program along with their parents regularly for 3 months. HbA1c levels were measured at base line and after 6 months. The glycaemic status of the patients were evaluated before and after the education programme. A total of 124 children were included in the study but 17 were lost to follow up. Most dropped out patients did not attend the follow-ups for personal reasons. So, 107 patients completed all the sessions and considered for data analysis.*

Results: *The total (107) study population comprised of 46 (43%) males and 61 (57%) females with a mean age of 11.2 ± 3.8 years and median duration of diabetes was 17.9 [IQR: 4.0-45.3 months].(Table 1). The pre- and post-intervention HbA1c were 9.3± 2.0 (95% CI 8.8-9.5) and 8.5 ± 1.6 (95% CI 8.3-8.9) percent respectively. A significant decrease in the HbA1c was observed in the study group. Mean difference in HbA1c before and after the intervention was 76 % (95% CI 0.439-1.08) ($p < 0.0001$) (Fig 1). Comparing the glycaemic control, post HbA1c was reduced significantly in girls than in boys. (p .03) (Fig 2).*

Conclusion: *Structured diabetes education programme as a part of multidisciplinary approach in treating diabetes has a positive impact on glycaemic control in children and adolescents with diabetes. However, further study with large sample size is required to find out the long term effects.*

(MH Samorita Med Coll J 2019; 2(1): 3-6)

1. Dr. Jebun Nahar, Associate Professor , Department of Paediatrics, Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorders (BIRDEM), Dhaka, Bangladesh.
2. Dr. Bulbul Ahmed, Senior medical Officer, Department of Paediatrics, Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorders (BIRDEM), Dhaka, Bangladesh.
3. Dr. Samin Tayyeb, Senior medical Officer, Department of Paediatrics and Changing Diabetes in Children Programme, Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorders (BIRDEM), Diabetic Association of Bangladesh, Dhaka, Bangladesh.
4. Dr. Nasreen Islam, Registrar, Department of Paediatrics, Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorders (BIRDEM), Dhaka, Bangladesh.
5. Dr. Bedowra Zabeen, Consultant and Coordinator, Department of Paediatrics & Changing Diabetes in Children Programme, Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorders (BIRDEM), Diabetic Association of Bangladesh, Dhaka, Bangladesh.

Address of correspondence: Dr. Bedowra Zabeen, Consultant Paediatric Endocrinologist, Coordinator, CDiC Paediatric Diabetes Center, Room 309, 1/A Shegunbagicha, BIRDEM 2, Dhaka-1000, Bangladesh. Email: bzabeen@hotmail.com, bzabeen@gmail.com Cell : +8801819259647

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Introduction

Type-1 diabetes mellitus (T1DM) is the commonest form of diabetes comprising more than 90% of cases of childhood and adolescent diabetes.[1] The chronic hyperglycaemia is associated with long term damage, dysfunction and failure of various organs, in particular the eyes, kidneys, nerves, heart and blood vessels.[2,3] The importance of optimal control in children and adolescents to prevent the long-term complications of diabetes is well recognized. The management of T1DM in children and adolescents has been a challenge for health care providers because it requires high adherence to complex tasks such as insulin injection, blood glucose monitoring, nutrition plans and physical activity.[4] Guidelines have recently been changed to encourage keeping HbA1c in children of less than 7%.[5] Specialized care is essential through multidisciplinary team which ensures the best long-term outcomes. [6] Education is the key stone of management of diabetes in children and adolescents. Improving diabetes care for children and young people requires educational programmes, to provide the knowledge and skills necessary to make informed choices, to facilitate self-directed behaviour changes and ultimately to reduce the risk of complications.[7] Intensive education and treatment may also prevent or delay the onset and progression of complication.[7] Diabetes self-management education (DSME) is the process of teaching individuals with diabetes in all aspects of the disease.[8] Studies carried out across the globe especially with short-term follow-up demonstrated positive effects of DSME programmes on knowledge, frequency and accuracy of self-monitoring of blood glucose; self-reported dietary habits and glycaemic control.[9-13] This study was undertaken to evaluate the impact of structured diabetes education programme on glycaemic control in children and adolescents with type-1 Diabetes.

Materials and Methods

This study was conducted at the CDiC (Changing Diabetes in Children), Paediatric Diabetic Clinic, BIRDEM from March to August 2012. One hundred twenty four registered T1DM patients with their parents/caregivers were selected randomly from the diabetic OPD during 8 weeks recruitment period (January to February 2012). A before and after study design was used. Based on international guidelines, specially designed educational modules according to the local needs were developed by the research team.[14,15] The studied patients attended a structured diabetes education programme along with their parents regularly for 3 months. Group and individual sessions were organized by multidisciplinary Paediatric diabetes team consisted of Paediatric Diabetologist, Diabetes Educator, Nutritionist and Psychologist. The education session included basic information of diabetes, acute complications, planning for special needs, nutritional advice, practical demonstration of insulin therapy and glucometer use. Audio-visual materials were used during the session. Books, posters in local language, video presentations were used for better understanding of the patients and their caregivers. Practical sessions were also organized individually to demonstrate how to inject insulin and how to use glucometer. Glycaemic status of the patients were evaluated before and after the education programme. HbA1c levels were measured at base line and after 6 months. Demographic and clinical data were collected including age at disease onset and during registration, sex, clinical and laboratory aspects of metabolic control. A total of 124 children were included in the study but 17 were lost to follow up. Most dropped out patients did not attend the follow-ups for personal reasons. So, one hundred and seven patients completed all the sessions and included in analysis. Data was analyzed by SPSS version 17. Qualitative data was expressed as numbers and percentages and quantitative data was expressed as mean and standard deviation. Paired sample t-test was used for comparison of pre and post HbA1C. The level of significance was defined as $p < 0.05$.

Results

The study population (107) comprised of 46 (43%) males and 61 (57%) females with a mean age of 11.2 ± 3.8 years and median duration of diabetes was 17.9 months [IQR:4.0-45.3] (Table 1). The pre and post intervention HbA1c were 9.3± 2.0 (95% CI 8.8-9.5) and 8.5 ± 1.6 (95% CI 8.3-8.9) percent respectively. A significant decrease in HbA1c was observed in the study group ($p < 0.0001$) (Fig. 1). Comparing the glycaemic control, post HbA1c was reduced significantly in girls than in boys. (p .03) (Fig 2).

Table 1. Demographic Characteristics of Studied Patients (n=107)

Mean age at Diagnosis	11.2 ± 3.8
Mean age at registration	13.5 ± 3.4
Family history of diabetes	66(45.5%)
Gender	
Male	46(43%)
Female	61(57%)
Mean duration of diabetes	17.9
	[IQR:4.0-45.3 months]

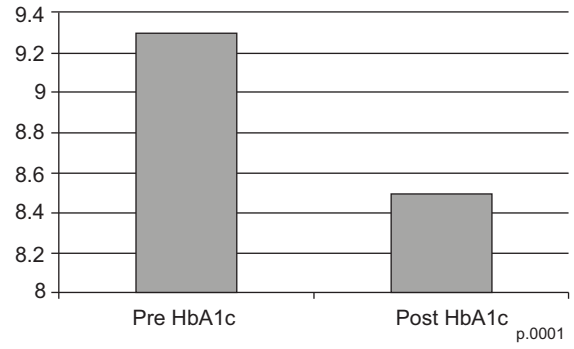

Fig. 1: ***Pre and post Education HbA1c in all studied Children and Adolescents (n=107)***

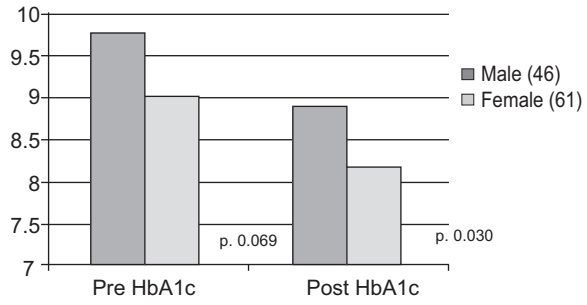

Fig. 2: ***Association of Pre and post Education HbA1c with Gender (n=107)***

Discussion

Diabetes self-management education programmes are seen as vital and fundamental tools for enabling patients to effectively manage their diabetes in high-income countries.[16] This study demonstrates that an intensive, structured and systematic group-based DSME programme can help patients manage their blood glucose levels more effectively than conventional, healthcare setting similar to the results of other research studies.[9-13] Interactive group programmes of patients' education help to stimulate interaction among participants, which enhances the efficacy of education and peer listening improves learning.[17,18] The interactive group sessions not only improve the relationship among the study population and medical personnel but also help in better understanding of patient's behaviour and thoughts. Self-monitoring blood glucose (SMBG) is an important part of glycaemic control and is recommended at least 3 or more times daily for T1DM patients.[19] Many similar studies in their programmes have laid special emphasis on regular SMBG and made special arrangements to ensure its practical application by the patients[7,9-11] and frequent SMBG had better glycaemic control. In this study, due to lack of awareness and low socioeconomic conditions most patients were not doing SMBG regularly. Free glucometers along with 3 months supply of strips for at least one test daily were provided to them along with log books to ensure record keeping. Similar type of study done in Pakistan with DSME but limited resource of strips had better glycemic control after 6 months.[20]According to current international paediatric guidelines the treatment and education of children and youth with diabetes need to be delivered by a certified multidisciplinary team consisting of a Paediatric diabetologist, a specialized diabetic nurse educator, a dietitian as well as an associated child psychologist, and a social worker.[15] The results of this study show that proper and well directed DSME programme delivered by trained personnel helps to improve glycaemic control without undue emphasis on the number of insulin injections and favourably altering various long-term risk factors in TIDM but over a shorter period of time. In the present study, post HbA1c has been significantly improved in girls than in boys. Different finding was found in different studies, where mean glycosylated hemoglobin (HbA1c) was significantly higher in females.[21,22] Loveman et al[12] conducted a systematic review to assess the clinical effectiveness and cost effectiveness of educational interventions and included 24 studies. The results showed a long lasting improvement in metabolic control completed the study and reduction in complications in T1DM patients.[12] The results of this study showed that proper and well directed DSME programme delivered by trained personnel helps to improve glycaemic control.

Conclusion

Implementing a multidisciplinary approach with structured diabetes education programme during treating diabetes in children and adolescent results in improved glycemic control. However, further study with large sample size is required to authenticate the results of this study.

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Formative Assessment of Interns in Paediatrics and its Feedback: Views of Interns and Faculties of Paediatrics in a Medical College Hospital of Bangladesh

Afroza S[1], Rouf MA[2], Basir HB[3]

Abstract

Introduction: *Assessment is the key component of a curriculum in medical education. Formative assessment has a central role in the evolving learning process because it influences learning. Feedback in medical education is an integral and important constituent of teaching as it encourages and enhances the learners' knowledge, skills and professional performance. The objective of the present study was to find out the views of Interns and faculties of Paediatrics regarding formative assessment of Interns and its feedback during their attachment in the department of Paediatrics of a Medical College Hospital.*

Materials and Methods*: This cross sectional study was conducted among the 66 interns who were placed in the department of Paediatrics, Shaheed Suhrawardy Medical College Hospital from January to October' 2015 and 12 faculties (All inclusive) of the same department. The respondents were enrolled by purposive sampling. Data were collected from 1st to 25th Oct'2015 using two separate semi-structured questionnaires with 5-point-Likert scale (one each) for both Interns and Faculties. Data were analysed manually.*

Results*: According to 85% studied interns formative assessment is very important and 81% opined that provision of formative assessment helped them to become more attentive in the ward. Formative assessment also helped them to become more punctual and to achieve better knowledge and skills comprising 74% and 78.5% respectively. It also helped them (comprising 53%) to become more close to the patients and more confident in patient management (76.7%). About 82% agreed that feedback on formative assessment is very important for them and for other interns. Correct responses on OSPE stations and guideline for delivering the answer were provided during feedback session (stated by 92.5% and 88% studied interns respectively). Majority (90.5%) agreed that feedback on formative assessment was helpful to find out their learning gaps during clinical attachment in paediatrics. According to 80% of studied Interns, feedback session was helpful to become more confident in patient management and their approach to teachers became easier.*

All the faculties expressed that formative assessment is very important for Interns and most of them (91.7%) stated that it is motivational to them (Interns) for patient management. All of them agreed that formative assessment increases the confidence and improves psychomotor skills of Interns in patient management. Majority of them (91.7%) stated that formative assessment helps to improve doctor-patient relationship of Interns, to attend ward regularly and to be more attentive to their (Interns) tasks given in the ward.

All the faculties opined that feedback session following formative assessment is very important where correct responses on OSPE stations are being discussed and guideline for delivering the answers are provided (91.7%). All the faculties stated that feedback helps the Interns to find out their learning gaps and to be more confident for patient management. All the faculties mentioned that presence of supervising doctors is very important during Intern feedback session, which helps the supervising doctors to identify their gaps during supervising Interns, it also helps to improve their confidence on supervision of Interns and patient management. Finally, the faculties opined that feedback session helps to improve professional relationship among teachers and trainee doctors.

Conclusion: *It can be concluded that formative assessment in a way of OSPE along with feedback is very important to develop knowledgeable, skilled and confident future doctors for standard patient care.*

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1. Prof. Dr. Syeda Afroza, Professor and Head, Department of Paediatrics, MHSHMC, Dhaka.
2. Dr. MA Rouf, Assistant Professor, Department of Paediatrics, ShSMC, Dhaka
3. Dr. Habiba Binte Bashir, Registrar, Department of Paediatrics, MHSHMC, Dhaka.

Address of correspondence: Prof. Dr. Syeda Afroza, Professor & Head, Dept. of Paediatrics, MH Samorita Hospital & Medical College, Dhaka, Bangladesh. Email: s_afroza@yahoo.com. Cell no:+8801915326302.

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Introduction

Assessment is one of the most important component of a curriculum in Medical Education. Learning is the act of acquiring new or modifying and reinforcing existing knowledge, behaviors, skills, values, or performances and may involve synthesizing different types of information.[1] Learning sessions are usually judged by taking different assessments at different level. These assessment systems are

organized in different fashions and feedback fulfill the inert importance of assessments.[1] Formative assessment is a process through which assessment elicited evidence of student learning is gathered and instruction is modified in response to feedback.[2]

Feedback is defined, as information given to a student describing his or her performance, the information intended to be used to guide future performance. [3] Feedback in medical education is an integral and important constituent of teaching as it encourages and enhances the learners' knowledge, skills and professional performance. Feedback has to be delivered in an appropriate settings. It should focus on the performance and not on the individual; should be clear and specific; delivered in non-judgmental language; should emphasize positive aspects; be descriptive rather than evaluative; and should suggest measures for improvement.[4] Alam KK[5] found in his study that 89% teachers agreed that if written answers are given back to the students after the examination the students will find out their weakness. When a student receives clear and descriptive feedback on their work, they have the opportunity to analyze what they have done well and which part(s) need improvement. Assessment and feedback given is also a source of information to the teacher about how much and what the students have learned.[6] When formative assessments are used in conjunction with summative assessment, the potential exists to improve outcomes for all students.[7]

The objective of the present study was to find out the views of Interns and Faculties of Paediatrics regarding formative assessment of Interns and its feedback during their attachment in the Department of Paediatrics of a Medical College Hospital.

Materials and Methods

This cross sectional study was conducted in the department of Paediatrics, Shaheed Suhrawardy Medical College Hospital, Dhaka from January to October' 2015. Study populations were Interns placed in the Dept. of Paediatrics from January to October' 2015 and faculties (All inclusive) of the same department. Sample Size was 66 Interns and 12 Faculties (All inclusive) of the Department of Paediatrics. The respondents were enrolled by purposive sampling. Data were collected from 1st to 25th Oct' 2015 using two separate semi-structured questionnaires with 5-point-Likert scale for both Interns and Faculties. Information were gathered on views of Interns and faculties of Paediatrics regarding formative assessment of Interns (conducted by OSPE) and its feedback during their attachment in the department of Paediatrics. Data were analysed manually and presented in tables.

Results

Views of Interns on formative assessment at the end of clinical attachment in1s Paediatrics are shown in Table 1. According to 85% studied Interns formative assessment is very important and 81% opined that provision of formative assessment

Table 1. Views of Interns regarding Formative Assessment in Paediatrics (N=66)

Views (On Formative Assessment)	Response No.(%)				
	SDA	DA	UC	A	SA
Formative assessment is very important for Interns at the end of clinical attachment in Paediatrics.	1(1.5)	2 (3)	5 (7.5)	35 (53)	21(32)
The provision of formative assessment is helpful for attention in the ward.	2(3)	5(7.5)	5(7.5)	28(42)	26(39)
Help interns to be more punctual in the ward	2(3)	8(12)	7(10.5)	24 (36)	25 (37.8)
Helps Interns to attend the ward regularly for achieving better knowledge and Skills	2(3)	7(10.5)	5(7.5)	29 (43.8)	23 (34.7)
Helps Interns to attend the ward round regularly with good attention	3 (4.5)	5(7.5)	6(9)	22 (33)	29 (43.8)
Helps Interns to become more close to the patients at bedside for better learning	2(3)	7 (10.5)	11(16.6)	15(22.6)	20(30.5)
Helps Interns to become more confident in patient management	2(3)	6(9)	5(7.5)	28 (42)	23 (34.7)

(SDA: Strongly disagree, DA: Disagree, UC: Uncertain, A: Agree, SA: Strongly agree).

helped them to become more attentive in the ward. Formative assessment also helped them to become more punctual and to attend the ward regularly for achieving better knowledge and skills comprising 74% and 78.5% respectively. More than half (53%) stated that formative assessment helped them to become more close to the patients at bedside for better learning and to become more confident in patient management (76.7%). Table 2 reveals the views of faculties regarding formative assessment of Interns. All the faculties expressed that formative assessment is very important for Interns. Most of them (91.7%) opined that it is motivational to Interns. All the faculties agreed that formative assessment increases the confidence of Interns in patient management, at the same time it is helpful to improve psychomotor skills of Interns regarding patient care. In addition, most of them (91.7%) stated that formative assessment helps to improve doctor-patient relationship, 91.3% faculties opined that formative assessment helps the interns to attend ward regularly as well as to become more attentive to their tasks given in the ward. Table 3 expressed the views of Interns regarding feedback on formative assessment. About 82% of studied Interns agreed that feedback on formative assessment is very important for them and for other Interns. Most of them (92.5%) agreed that discussion

Table 2. Views of Faculties (Teachers) regarding Formative Assessment in Paediatrics (N=12)

Views (On Formative Assessment)	Response No.(%)				
	SDA	DA	UC	A	SA
Formative assessment is very important for Interns at the end of clinical attachment in Paediatrics	0(0)	0(0)	0(0)	2 (16.6)	10 (83.3)
Formative assessment is motivational to Interns for patient management	0(0)	0(0)	1 (8.3)	2(16.7)	9(75)
Helps interns to increase confidence for diagnosis and treatment	0(0)	0(0)	0(0)	2 (16.6)	10 (83.3)
Helpful for intents to improve psychomotor skills in patient care	0(0)	0(0)	0(0)	2(16.6)	10 (83.3)
Helps Interns to improve doctor-patient relationship	0(0)	1 (8.3)	0(0)	2(16.7)	9(75)
Helps the Interns to attend the Ward regularly throughout the clinical attachment	0(0)	0(0)	1 (8.3)	6 (49.8)	5 (41.5)
Helps the Intents to be more attentive to their given tasks in the Ward	0(0)	0(0)	1 (8.3)	5(41.5)	6 (49.8)

(SDA: Strongly disagree, DA: Disagree, UC: Uncertain, A: Agree, SA: Strongly agree).

Table 3. Views of Interns regarding Feedback on Formative Assessment in Paediatrics (N=66)

Views (On Feedback)	Response No.(%)				
	SDA	DA	UC	A	SA
Feedback on formative assessment is important for Interns.	1 (1.5)	3 (4.5)	8 (12)	33 (49.8)	21 (32)
Discussion on Correct responses of OSPE stations have been done	1(1.5)	0(0)	4(6)	41 (62)	20(30.5)
Guidelines on delivering the answer have been provided by the teachers at the session	1(1.5)	1 (1.5)	6(9)	37 (55.8)	21 (32)
Feedback is helpful to find out the learning gaps during clinical attachment in Paediatrics	2(3)	0(0)	4(6.1)	37 (56.1)	23 (34.8)
Feedback session is helpful to be more confident in patient management	2(3)	1(1.5)	10 (15.2)	31 (47)	22 (33.3)
Approach of interns to the teachers became easier after feedback session	3(4.5)	2(3)	8(12.1)	28 (42.4)	25 (37.9)

(SDA: Strongly disagree, DA: Disagree, UC: Uncertain, A: Agree, SA: Strongly agree).

on correct responses on OSPE stations has been done in their feedback session. Majority (88%) stated that guideline for delivering the answer were provided during feedback session. Most of the studied Interns (90.5%) stated that feedback on formative assessment is helpful to find out their learning gaps during clinical attachment in Paediatrics. More than 80% opined that feedback session helped them to become more confident in patient management and at the same time their approach to teachers became easier following feedback session. Views of faculties regarding feedback on formative assessment in Paediatrics are shown in Table 4. All the faculties opined that feedback session following formative assessment is very important for Interns, where correct responses on OSPE stations are being discussed by the teachers. Most of the faculties (91.7%) agreed that guideline for delivering the answer are being provided during feedback session. All the faculties stated that feedback on formative assessment helps the Interns to find out their learning gaps during clinical attachment and helps to increase their confidence on patient management. Moreover, all the faculties mentioned that presence of supervising doctors ie., registrars and Asstt. registrars is very important during intern feedback session, which helps them to identify their gaps during supervising Interns. Majority of the faculties (91.7%) agreed that feedback also helps to improve the confidence of supervising doctors on supervision of Interns, also to improve their confidence on patient management. Finally, all the faculties opined that feedback session helps to improve professional relationship among teachers and trainee doctors.

Table 4. Views of Faculties regarding Feedback on Formative Assessment in Paediatrics (N=12)

Views (On Feedback)	Response No. (%)				
	SDA	DA	UC	A	SA
The faculties provide feedback on formative assessment of Interns regularly at the end of their clinical attachment	0(0)	0(0)	0(0)	2 (16.6)	10 (83.3)
Feedback on formative assessment for Interns is very important	0(0)	0(0)	0(0)	2 (16.6)	10 (83.3)
Correct responses on OSPE stations are being discussed during feedback session	0(0)	0(0)	0(0)	2 (16.6)	10 (83.3)
Guidelines on delivering the answer have been provided by the teachers at the session	0(0)	1(8.3)	0(0)	5 (41.7)	6(50)
Feedback is helpful for Interns to find out the learning gaps during clinical attachment in paediatrics	0(0)	0(0)	0(0)	0(0)	12 (100)
Feedback session is helpful for Interns to be more confident in patient management	0(0)	0(0)	0(0)	2(16.7)	10 (83.3)
Presence of supervising doctors of Interns (AR Reg) at Feedback session is very important	0(0)	0(0)	0(0)	1(8.3)	11 (91.7)
Feedback session helps the supervising doctors to identify their gaps during supervision of Interns	0(0)	0(0)	0(0)	2(16.7)	10 (83.3)
Feedback session helps to improve confidence level of supervising doctors in supervision of Interns about patient care	0(0)	0(0)	1(8.3)	3(25)	8 (66.7)
Feedback session helps the supervising doctors to improve their confidence on patient management	0(0)	0(0)	1 (8.3)	6(50)	5 (41.7)
Feedback session helps to improve the professional relationship among teachers and trainee doctors	0(0)	0(0)	0(0)	3(25)	9(75)

(SDA: Strongly disagree, DA: Disagree, UC: Uncertain, A: Agree, SA: Strongly agree).

Discussion

Majority of studied interns (85%) stated that formative assessment is very important and 81% opined that provision of formative assessment helped them to become more attentive in the ward. Formative assessment also helped the studied interns to become more punctual and to attend the ward regularly to achieve better knowledge and skills comprising 74% and 78.5% respectively. More than half (53%) stated that formative assessment helped them to become more close to the patients at bedside for better learning and to become more confident in patient management (76.7%). All the faculties expressed that formative assessment is very important for interns. Most of them opined that it is motivational to interns. All the faculties agreed that formative assessment increases the confidence of interns in patient management, at the same time it is helpful for interns to improve psychomotor skills regarding patient care. In addition, most of them (91.7%) stated that formative assessment helps to improve doctor-patient relationship of interns, attending ward regularly and to become more attentive to their tasks given in the ward. All these findings related to formative assessment are consistent with the results of some other studies.[5,8] Many research in the past identified that feedback is one of the most important issues in formative assessment, in other words without feedback formative assessments are useless.[9]

About 82% of studied intern agreed that feedback on formative assessment is very important for them and for other interns. Most of them (92.5%) agreed that discussion on correct responses on OSPE stations has been done in their feedback session. Majority (88%) stated that guideline for delivering the answers were provided during feedback session which is nearly consistent with another study result.[8] Among the studied Interns, 90.5% stated that feedback on formative assessment is helpful to find out their learning gaps during clinical attachment in Paediatrics. In a study by Khaleda[8], most of the Intern doctors (64% strongly agreed and 31.3% agreed) agreed that proper feedback after formative assessments can help to find out their learning gaps and weaknesses. [8] Some authors said that dialogic feedback brings higher quality learning outcome, feedback reduces the student's uncertainty about his or her performance and progress and it gives better outcome when properly delivered. [10] Eighty percent of studied interns opined that feedback session helped them to become more confident in patient management and their approach to teachers became easier following feedback session. In another study 40% Intern doctors opined that proper feedback can improve relationship between teachers and students. [11]

In the present study, all the Faculties opined that feedback session following formative assessment is very important for Interns, which is provided regularly at the end of clinical attachment and correct responses on OSPE stations are being discussed by the teachers. Most of the Faculties (91.7%) agreed that guideline for delivering the correct answers are being provided during feedback session. At the same time all the Faculties stated that feedback helps the Interns to find out their learning gaps during clinical attachment and helps to increase their confidence level on patient management. Alam KK[5] found in his study that 89% teachers agreed that if written answers are given back to the students after the examination the students would find out their weaknesses. In a national study[8] 76% teachers strongly agreed and 24% agreed that proper feedback after formative assessment can help to find out the learning gaps and weaknesses of learners. Some author mentioned that, constructive feedback on bed side teaching is more effective in developing clinical skills, communication skill and professional bed side manners. [4] In Khaleda's study[8] most of the teachers (78%) and Intern doctors (62%) strongly agreed that further improvement is possible in subsequent assessment following proper feedback after formative assessment.

Formative assessment followed by feedback improves students' achievement and it is one of the most effective instructional tool that influences the students' performance.

Conclusion

It can be concluded that formative assessment with feedback for Interns is very important to develop knowledgeable, skilled and confident future doctors for standard patient care in Paediatrics.

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Risk Assessment Score for Developing Type 2 Diabetes among Population Attending a Tertiary Care Hospital in Bangladesh

Akter N[1], Khan S[2]

Abstract

Introduction*: Bangladesh has nearly 8.4 million of diabetic patients which is expected to reach 16.8 million by 2030 according to international diabetes federation (IDF)[1]. As the prevalence of diabetes mellitus (DM) among Bangladeshi adults raises prediction of new cases of type 2 diabetes (T2DM) in Bangladesh requires early identification and screening for mitigating early preventive and health pro-motion measures for the local populations at high risk for developing T2DM. ADA (American diabetes association) diabetes risk score tool is a cost effective and simple method for identifying undiagnosed diabetic subject at community level.*

Objectives: *To find out the distribution of ADA risk score for developing type 2 diabetes among the study population and to determine the association of ADA risk score with modifiable and non-modifiable risk factors of type 2 diabetes.*

Materials and Methods: *This cross-sectional observational study was conducted among randomly sampled 410 adult Bangladeshi subjects. The study population consisted of adult non-diabetic patients visiting the outpatient department (OPD) of Medicine, MARKS Medical College and Hospital, Dhaka, Bangladesh from January to October 2018. Female with previous history of gestational diabetes mellitus (GDM) were also included. With written informed consent, the ADA (American Diabetes Association) risk assessment tool for type 2 diabetes mellitus (T2DM) questionnaire was used to collect the data including demographic characteristics and different risk factors and to calculate total risk score for predicting the risk of developing type2 diabetes.*

Results: *Out of 410 a significant number of studied subjects (32.4%) had ADA risk scores of ≥ 5. Among them, 23.9% were male and the rest were female. The Mean (±SD) age of the studied subjects was 45.01±8.41 years. In this study, 50-59 years age group (15.4%) had significant association with increased risk score; [p<0.001]. Subjects with high BMI (over weight and obese) had significant association with increased ADA risk score for developing diabetes; [p< 0.001]. There was significant association in between increased risk score (≥5) and physical inactivity, positive history of gestational diabetes mellitus (GDM), history of hypertension and family history of diabetes; [p<0.05].*

Conclusion: *From the present study it can be concluded that by using simplified ADA diabetes risk assessing tool a quite good number of adults could be identified to have risk to develop Type 2 diabetes in future. High BMI, physical inactivity, history of GDM, hypertension and family history of diabetes also contribute as risk factors for developing diabetes. But it needs follow up and further studies to establish these facts.*

Keywords: *Type 2 Diabetes Mellitus, Risk Factors for Diabetes, ADA Diabetes Risk Score.*

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Introduction

Diabetes mellitus (DM) is one of the common non-communicable diseases (NCDs) globally. It is one of the most important leading causes of death in most high-income countries. There is substantial evidence[1,2] that it is epidemic in many economically developing and newly industrialized countries. With rapid economic development, life-style of human beings has been changed dramatically globally. The prevalence and incidence of type 2 diabetes mellitus

1. Dr. Nazma Akter, Assistant Professor (Endocrinology & Metabolism), Department of Medicine, MARKS Medical College & Hospital, Dhaka, Bangladesh.
2. Dr. Saira Khan, Associate Professor (CC), Department of Paediatrics, MH Samorita Medical College & Hospital, Dhaka, Bangladesh.

Address of correspondence: Dr. Nazma Akter, Assistant Professor (Endocrinology & Metabolism), Department of Medicine, MARKS Medical College & Hospital, Dhaka, Bangladesh. Email: nazma_aktar_endo@yahoo.com Mobile: +88 01714743850

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(T2DM) are increasing rapidly in the world. IDF estimated that the situation is much worsened as the burden would increase from 366 million (2011) to 552 million (2030).[1] The projection data may vary from different organization but the problem remains the same as the burden of diabetes is increasing day by day. Issued in 2015, the IDF estimated that the number of global diabetes individuals aged 20–79 was 415 million and will increase to 642 million by the year 2040.[3]

In Bangladesh, most of the patients are type 2 diabetics and the risk of developing type 2 diabetes mellitus (DM) is determined by some modifiable risk factors related to rapid urban growth and changing lifestyle (i.e. obesity, sedentary lifestyle, diet, smoking, physical and emotional stress) and non-modifiable risk factors (i.e. family history of diabetes, age, race/ethnicity).[4,5] The rising prevalence of type 2 diabetes in Bangladesh is primarily attributed to rapid urbanization and associated changes in lifestyle, such as sedentary lifestyle, higher calorie food intake, and stressful life. However, evidence suggests that lifestyle related interventions targeting modifiable risk factors can either prevent or delay the onset of type 2 diabetes.[5] The IDF estimates Bangladesh has nearly 8.4 million of diabetes patients which is expected to reach to 16.8 million by the year 2030.[1]

Prevention of diabetes is important because its treatment is costly.[1] Identification of indi-viduals at risk for T2DM can allow the risk factors, many of which are modifiable, to be addressed reducing the likelihood of developing T2DM.[6] Major risk factors identified for DM can be used to guide screening strategies, preventive measures and health pol-icy development. The benefits of early detection and inter-vention to improve health outcomes and reduce morbidity of T2DM have been shown in many studies.[7] Awareness of risk factors is a prerequisite to prevent diabetes among general population. If people are aware of the risk factors developing diabetes, the rate of its occurrence could be minimized. Evidence eventually reported that people who perceive themselves to be at risk of a disease are more likely to engage in and comply with efforts to reduce their risk of developing the problem.[8] Thus, considerable efforts are needed to make people aware about severity and probability of ill effects of diabetes as well as the risk factors of diabetes to avoid harm.[9]

Personalized intervention is helpful to prevent or delay T2DM by life-style change and pharmaceutical interference.[10] Fasting plasma glucose (FPG), oral glucose tolerance test (OGTT) and HBA1c are commonly used for T2DM determination in clinical and epidemiological studies.[11] However, their application has some limitations, which cannot succinctly identify high risk individuals and screen large population on the spot. There are certain risk factors associated with diabetes which can be used to recognize high-risk individuals for early intervention.[12] Risk scores based on some risk factors without laboratory tests have been demonstrated as an effective, low cost and noninvasive tool for identifying the high-risk individuals for developing T2DM.[13] Studies also support the utilization of risk-assessment scor-ing systems in quantifying individual's risk for developing T2DM.[14]

As the prevalence of DM among Bangladeshi adults raises prediction of new cases of T2DM in Bangladesh requires early identification and screening for mitigating early preventive and health pro-motion measures or actions for the local populations at high risk for developing T2DM. There are few researches examining the risk-assessment score[15] for developing T2DM in Bangladesh. Thus, a simple risk-assessment scoring system for early screening of T2DM among Bangladeshi adults will be beneficial to identify the high-risk adults and thus taking adequate preventive measures in combating DM.

Materials and Methods

This cross-sectional observational study was conducted among randomly selected 410 adult Bangladeshi male and female subjects. The study population consisted of adult non-diabetic subjects visiting the outpatient department (OPD) of Medicine, MARKS Medical College and Hospital in Dhaka, Bangladesh from January to October 2018. Female with previous history of gestational diabetes mellitus (GDM) were also included. Individuals with a diagno-sis of endocrine illness, metabolic syndrome, T1DM, T2DM, apparent communicative, cognitive impair-ment or physical disability were excluded from the study. With written informed consent, The ADA (American Diabetes Association) risk for type 2 diabetes (T2DM) questionnaire was used to collect the data including demographic characteristics and different risk factors and to

calculate total risk score to predict the risk of developing type2 diabetes mellitus.

ADA (American Diabetes Association) Diabetes Risk tool

Various risk factors of T2DM were reviewed from the litera-ture like sex, age, ethnicity, family history of diabetes, diet, exercise, hypertension, body mass index (BMI), weight, waist circumference (WC) and gestational diabetes[16] etc. Various risk-assessment scoring systems were reviewed like American Diabetes Association (ADA),[17] Rotterdam,[18] Cambridge,[7] Finnish etc.[19-21] Study showed that ADA scores performed well and somewhat better than other scoring system.[22] The ADA risk test showed[23] good accuracy (area under the receiver operating curve 0.725) in screening for diabetes with an optimal cut-off score of five. The ADA developing type 2 diabetes risk tool was well developed and tested for validity and reliability on different population in different coun-tries.[24] After review of literature regarding risk factors of developing DM in Bangladesh, the ADA type 2 diabetes risk score was found to be relevant and useful for the Bangladeshi adults.

Method of Calculating ADA Diabetes Risk Score

To calculate risk for developing type 2 diabetes in ADA diabetes risk tool; height, weight, BMI and blood pressure were measured. Height and weight were measured by a reliable height scale and weighing scale, respectively. BMI: weight in kilograms/square of height in meters (kg/m^2) was categorized as underweight (d"18.5 kg/ m^2), normal (BMI: <25 kg/m^2), overweight (BMI: 25–<30 kg/m^2), obese (BMI: e"30 - <40 kg/m^2) and morbid obese (BMI: e"40 kg/m^2). Blood pressure was measured by a manual sphygmomanometer in standard conditions (measured 2 times after a 5-min rest between each measurement)[25] Hypertension was defined as a systolic blood pressure e"140 mmHg and/or dias-tolic blood pressure e"90 mmHg, or in case of use of anti-hypertensive medications.[17]

The ADA risk score system[24] has 7 risk factors correlating with the risk of developing T2DM and was used to detect a diabetes risk based on age (Less than 40 years: 0 point; 40–49 years: 1 point;50–59 years: 2 points; 60 years or older: 3 points), sex (Man:1 point; Woman: 0 points), body mass index (BMI) (Normal Weight: 0 point; Overweight: 1point; Obese: 2point; Morbid obese: 3), physical activity (Yes : 0 points; No:1 point), past history of gestational diabetes mellitus (GDM) (Yes:1 point; No:0 points), family history (Yes: 1 point; No: 0 points), and history of hypertension or high blood pressure (Yes: 1 point; No: 0 points). It demonstrates the probability of developing T2DM. If anyone score 5 or more; that person are at increased risk for having type 2diabetes. Only blood glucose test can confirm of having type 2 diabetes or prediabetes, a condition in which blood glucose levels are higher than normal but not yet high enough to be diagnosed as diabetes. Scores e"5 should be formally screened for diabetes, per ADA[24] guidelines.

Statistical Analysis

Data were analyzed with Statistical Package for Social Science (SPSS) software version 16. The means and standard deviations were used to describe continuous data. For categorical data, frequencies and percentages were estimated. Among the basic characteristics of the study subjects, the continuous variables were compared with each other using the Student's t-test. Categorical variables were compared with each other using the chi-square test. P value <0.05 was considered as significant.

Results

A total of 410 subjects were included in the study. Among them, male were 55.9% and female were 44.1%. The Mean (± SD) age of the study subjects was 45.01 ± 8.41 years. The mean height (m) and weight (kg) were higher in male than in female subjects; [p<0.001]. Average BMI (kg/m^2) was 25.12 ± 3.45 (± SD) in male and 24.62 ± 3.46 (± SD) in female subjects; [p=0.14]. Mean Blood Pressure (mm Hg) of the study subjects were SBP (Systolic Blood Pressure): 116.50 ±1.40 (±SD) and DBP (Diastolic Blood Pressure):77.24±8.44 (±SD) (Table 1) Among the studied subjects, the calculated mean (±SD) ADA risk score was 4.30 ± 1.54 and 3.39 ± 1.47 among male and female respectively. [p<0.001] (Table 1). A total 32.4% of the subjects had high dia-betes risk score (DRS ≥ 5). Among them, 23.9% were male and 8.5% were female; [p<0.001] (Table 2).

Table 1. Demographic, clinical characteristics & ADA risk assessment score for type 2 diabetes in studied subjects (n=410)

Variables	Male (Mean ± SD)	Female (Mean ± SD)	Total (Mean ± SD)	p value
Age (Yrs)	45.37 ± 9.06	44.57 ±7.51	45.01 ± 8.41	0.34
Height(m)	1.64 ± 6.99	1.56 ± 5.81	1.60 ±7.54	<0.001
Weight(kg)	68.27 ± 1.05	60.99 ± 9.47	65.06 ± 1.07	<0.001
Body mass index(kg/m^2)	25.12 ± 3.45	24.62 ± 3.46	24.90 ± 3.46	0.14
Systolic Blood Pressure (mm Hg)	117.34 ± 1.44	115.47 ± 1.35	116.5 ±1.40	0.18
Diastolic Blood Pressure (mm Hg)	78.25 ± 8.08	75.96 ± 8.74	77.24 ± 8.44	0.006
ADA Risk score for Type 2 Diabetes	4.30 ± 1.54	3.39 ± 1.47	3.90 ± 1.57	<0.001

ADA: American Diabetes Association; p<0.05=significant.

Table 2. Distribution of studied subjects according to ADA risk assessment score for type 2 Diabetes (n=410)

Variables		Male [N (%)]	Female [N (%)]	Total [N (%)]	λ Value	df	p value
ADA Risk Score for Type 2 Diabetes	Increased Risk (ADA risk Score ≥5)	98(23.9)	35(8.5)	133(32.4)	25.38	1	<0.001
	Low Risk (ADA risk Score <5)	131(32.9)	146(35.6)	277(67.6)			

ADA: American Diabetes Association; Pearson chi-square = » value; df: degree of freedom; p<0.05=significant.

Table 3 shows the distribution of risk factors for ADA Risk Assessment Score for type 2 diabetes among studied subjects. According to category of age, most of the male and female were from 40-49 years age group (male vs. female: 18.3% vs. 21.2%); [p<0.05]. More than half of the subjects (53.9%) had a positive family history (mother, father, sister or brother) of DM; [p<0.05]. A total of 47.3 % of the studied subjects had past history of hypertension or were on anti-hypertensive medications; [p=0.05]. Above half of the studied subjects (57.3%) did not do daily physical activity [male vs. female: 30.7 % vs.26.6%]; [p=0.29]. Some of the subjects had high BMI (35.9% were overweight and 9.5 % were obese); [p=0.20] (Table 3).

Table 3. Distribution of risk factors considered for ADA risk assessment score for type 2 diabetes among studied subjects (n=410).

Risk factors for ADA Risk Assessment Score for Type 2 Diabetes		Male [N (%)] 229(55.9)	Female [N (%)] 181(44.1)	Total [N (%)] 410(100.0)	p value
Age	<40 Years	72(17.6)	49(12.0)	121(29.5)	0.01
	40-49 Years	75(18.3)	87(21.2)	162(39.5)	
	50-59 Years	64(15.6)	37(9.0)	101(24.6)	
	≥60 Years	18(4.4)	8(2.0)	26(6.3)	
Family history of diabetes mellitus	Yes	108(26.3)	113(27.6)	221 (53.9)	0.002
	No	121(29.5)	68(16.6)	189(46.1)	
History of hypertension	Yes	118(28.8)	76(18.5)	194(47.3)	0.05
	No	111(27.1)	105(25.6)	216(52.7)	
Daily physical activity	Yes	103(25.1)	72(17.6)	175(42.7)	0.29
	No	126(30.7)	109(26.6)	235(57.3)	
Weight Category; (BMI: kg/m^2)	Normal Weight(< 25kg/m^2)	118(28.8)	102(24.9)	220(53.7)	0.02
	Overweight(25-<30 kg/m^2)	81(19.8)	66(16.1)	147(35.9)	
	Obese(≥30- <40 kg/m^2)	28(6.8)	11(2.7)	39(9.5)	
	Morbid Obese(≥40 kg/m^2)	2(0.5)	2(0.5)	4(1.0)	

ADA: American Diabetes Association; BMI: body mass index; GDM: Gestational Diabetes Mellitus. Pearson chi-square = » value; p<0.05=significant. Among 181 females 44 (24.3%) had history of GDM.

Table 4: Cross tabulation between different risk factors and ADA risk score for type2 diabetes in studied subjects (n = 410)

Risk Factors of ADA Risk Assessment for Type 2 Diabetes		Increased ADA Risk Assessment Scores for Type 2 Diabetes (score ≥5)		≥ Value	df	p value
		Yes[N (%)]	No[N (%)]			
Sex	Male	98 (23.9)	131 (32.0)	25.83	1	<0.001
	Female	35 (8.5)	146 (35.6)			
Age	< 40 Years	10 (2.4)	111(27.1)	1.17	3	<0.001
	40-49 Years	37 (9.0)	125 (30.5)			
	50-59 Years	63 (15.4)	38 (9.3)			
	≥60 Years	23(5.6)	3(0.7)			
History of GDM	Yes	14 (3.4)	21 (5.1)	46.76	1	0.002
	No	123 (30.0)	23 (5.6)			
Family history of	Yes	92(22.4)	129 (31.5)	18.47	1	<0.001
diabetes mellitus	No	41 (10.0)	148 (36.1)			
History of hypertension/	Yes	93 (22.7)	101(24.6)	40.36	1	<0.001
High blood pressure	No	40 (9.8)	176 (42.9)			
Physically active	Yes	32 (7.8)	143 (34.9)	27.90	1	<0.001
	No	101 (24.6)	134 (32.7)			
Weight category;	Normal Weight	44 (10.7)	176 (42.9)	42.18	3	<0.001
(BMI: kg/m^2)	Overweight	61 (14.9)	86 (21.0)			
	Obese	25(6.1)	14(3.4)			
	Morbid Obese	3 (0.7)	1(0.2)			

Pearson chi-square = χ value; df: degree of freedom; p<0.05=significant; BMI: Body Mass Index; GDM: Gestational Diabetes Mellitus.

The ADA predicted increased risk score of developing type 2 diabetes was more in male than in female (p<0.001). Subjects from 50-59 years age group, with high BMI (over weight and obese), having no physical activity, history of hypertension, positive family history for DM and female with previous history of GDM had significant association with increased ADA risk score for developing diabetes; [p< 0.05] (Table 4).

Discussion

The increasing prevalence of diabetes and its complications in Bangladesh would pose a real threat to existing health services. Assessment of the risk score for developing diabetes can assist in its early detection, prevention and reduce its incidence.[26] Early detection leads to a better quality of life, reduced morbidity, premature mortality and ultimately a reduction in associated health care and wider economic costs. Adults with elevated risk assessment score, regardless of their blood glucose status, are suitable candidates for lifestyle modification. The health-care professionals can empower the patients in the high-risk group to be self-motivated with life-style modifications like increasing physical activity, maintaining ideal body weight and periodic health checks including blood glucose and blood pressure.[27]

In this study, simplified ADA Diabetes Risk Score Tool has been used to identify high risk subjects for diabetes. ADA diabetes risk tool is a useful tool for identifying people with asymptomatic DM who might not seek early or regular evaluation, thus facilitating intervention early in the disease course. A high dietary fiber and increase in vegetable consumption, low-moderate total calorie, reduced fat, low glycaemic index foods and low polyunsaturated fat intake reduce the susceptibility

to DM.[28] At least 30 min of moderate physical activity with variable emphasis on high-intensity and resistance training exercise (e.g. brisk walking, swimming, cycling, dancing) on all or most days of the week are recommended.[28] Regular walking for at least 30 min per day reduces diabetes risk by 35%-40%.[29] This has been proven to effectively prevent and delay the development of DM among young adults.[30]

In this study, there were significant association between the ADA diabetes risk score and both non-modifiable and modifiable risk factors among subjects ($p<0.05$). The non-modifiable risk factors i.e. age, positive family history of diabetes, past history of hypertension or gestational diabetes were the significant independent risk factors for predicting T2DM ($p<0.05$).

In the present study, it was also found that people with high body mass index (BMI) had more chance of being in the higher risk group in the ADA risk score ($p<0.05$). However, physical inactivity was another significant modifiable risk factor.

In Bangladesh, prevalence of diabetes in adults is 6.9 %,[31] with significant predictors like age, hypertension, obesity/BMI and family history.[6] In this study among subjects, 32.4% had high diabetes risk score (DRS ≥5). This predicts that 32.4% of the adult people have high risk to develop T2DM if no primary preventive measures are taken to curb it. This study result can be utilized to increase awareness and motivate people about the importance of modifiable risk factors regarding T2DM.

Conclusion

From the present study it can be concluded that a quite good number of adults of our country are at risk of developing type 2 diabetes mellitus (T2DM). High BMI, advancing age, physical inactivity, history of GDM, hypertension and family history of diabetes also came out as risk factors for developing diabetes. But it needs follow up and further studies to establish this fact. People with high risk of DM should be referred for early detection or intervention and changes to a healthy lifestyle to prevent or delay the onset of T2DM.

Limitations

This study is limited by the cross-sectional design and is not causal or effect study or measure of temporal changes. Validation of the risk assessment with a large sample size in different populations would have enhanced generalization and authentication of the results.

Conflict of interest: None declared.

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Review Article

Principle of Management of Non-Alcoholic Fatty Liver Disease

Akter N[1], Ferdous HS[2]

Abstract

Non-alcoholic fatty liver disease (NAFLD) is the fastest growing cause of liver disease in the world, yet there is no approved pharmacotherapy. It includes a spectrum of conditions from simple steatosis to non-alcoholic steatohepatitis (NASH) and can lead to fibrosis, cirrhosis, liver failure, and/or hepatocellular carcinoma. NAFLD is also associated with other medical conditions such as obesity, diabetes mellitus (DM), metabolic syndrome, hypertension, insulin resistance, hyperlipidemia, and cardiovascular disease (CVD). In diabetes, chronic hyperglycemia contributes to the development of both macro and microvascular conditions through a variety of metabolic pathways. While lifestyle modifications remain the mainstay of treatment, only a proportion of individuals are able to make or sustain them. Rising demand and an improved understanding of NASH patho-physiology has led to a surge in development of new therapies. In this review the current knowledge of NAFLD epidemiology, risk factors, genetics, pathogenesis, and treatment are summarized in order to aid in further understanding and better management of NAFLD patients.

Search strategy: *Reviews on guidelines for management and original articles related to non-alcoholic fatty liver disease were searched for the present article e.g., MEDLINE, Journal of hepatology, Pub Med etc. After going through all, more recent and evidence based guidelines were selected. Clinical trials rather than observational studies or reviews have been selected to prepare the manuscript.*

Key words: *Non-alcoholic fatty liver disease; Metabolic syndrome; Steatohepatitis; Steatosis; Hepatocellular carcinoma.*

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is defined as accumulation of excess fat in the liver without alcohol consumption.[1] According to National Health and Nutrition Examination Survey, the rate of NAFLD in chronic liver disorder has been substantially increasing over the past few decades. The global incidence was 47% in 1988 to 1994, 63% in 1999 to 2004, and 75% in 2005 to 2008 and thus, it became a global public health issue.[2] In Bangladesh, prevalence of fatty liver is 34.34% which is much higher than the prevalence of Hepatitis B (4.9%) and Hepatitis C (0.2%).[3] Among the NAFLD patients, at least 10 to 20% would develop NASH, which is a serious condition of liver disorders. It is predicted to become the leading cause of liver transplantation over the next 10 years.[4] Metabolic disorders including obesity, type 2 diabetes, chronic kidney disease, hypertension, and colorectal malignant neoplasm also have been largely increasing in the patients with NAFLD, implying positive correlation between NAFLD and metabolic disorders.[4]

The spectrum of NAFLD is very wide, covering hepatic steatosis and non-alcoholic steatohepatitis (NASH) resulting eventually in hepatic cirrhosis and hepatocellu-lar carcinoma.[5] Non-alcoholic steatohepatitis is mediated by inflammatory cytokines, mitochondrial dysfunction secondary to nutrient excess and oxidative stress, resulting in hepatocyte inflammation, ballooning, apoptosis, and

1. Dr. Nazma Akter, Assistant Professor (Endocrinology & Metabolism), Department of Medicine, MARKS Medical College & Hospital, Dhaka, Bangladesh.
2. Dr. Hossain Shahid Ferdous, Professor of Endocrinology, Popular Medical College & Hospital, Dhaka, Bangladesh.

Address of correspondence: Dr. Nazma Akter, Assistant Professor (Endocrinology), Department of Medicine, MARKS Medical College & Hospital, Mirpur-14, Dhaka -1206. Email: nazma_aktar_endo@yahoo.com Mobile: +88 01714743850

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activation of hepatic stellate cells (HSC).[6,7] Indeed, cardiovascular disease is the leading cause of death in NAFLD[8] and so, emerging pharmacotherapy should ideally aim to reduce both liver-related and cardiovascular related mortality. Liver biopsy remains the gold standard for definitive diagnosis of NAFLD, but the development of noninvasive advanced imaging, biochemical and genetic tests will provide future clinicians with a great deal of information and opportunity for enhanced understanding of the pathogenesis and targeted treatment.[8]

Thus, it is required to understand the pathogenesis of NAFLD and to determine potential therapeutic target for effective pharmacological treatment for NAFLD. Weight loss, as part of lifestyle change, is the only recommended intervention, with a loss of >7% total body weight associated with clearance of histological non-alcoholic steatohepatitis (NASH) and a >10% loss associated with an improvement in fibrosis.[7] Medications are currently targeted to concomitant features of the metabolic syndrome such as hypertension, dyslipidaemia and diabetes.[9] Agents such as Vitamin E and pioglitazoneare are recommended.[9] In this review, the current knowledge of NAFLD genetics, epidemiology, risk factors, pathogenesis and treatment are summarized in order to aid in further understanding of this disease and better management of NAFLD patients.

Genetics

Data from numerous studies have given evidence for a heritable component to NAFLD and includes: Familial aggregation, twin studies, and inter-ethnic differences in susceptibility.[10] Whole exome sequencing studies performed on obese Caucasian participants with NAFLD have revealed deleterious mutations in Bardet-Biedl syndrome 1 gene as well as the Melanocortin 3 receptor gene. [11] In 2008, the first genome wide association study was published; it examined hepatic triacyl glycerol (HTAG) accumulation and identified association with increased HTAG and the PNPLA3 gene.[11] This single nucleotide polymorphism is a non synonymous cytosine to guanine nucleotide transversion mutation that results in anisoleucine to methionine amino acid change. Subsequent work has confirmed this variant (PNPLA3 rs738409) in Japanese, Indian, and Chinese NAFLD patients.[12] A separate meta-analysis, including 16 studies revealed that rs738409 GG genotype compared to the CC genotype was linked to a 73% greater liver fat content as well as a 3.24-fold increased risk of more pronounced necro-inflammatory scores and a 3.2-fold increased risk of developing fibrosis.[13] Xu et al[14] by way of meta-analysis totaling 23 case-control studies found that PNPLA3 rs738409 polymorphism have a significant association with a high cross-ethnicity risk for NAFLD as well as NASH. Using exome-wide association, Kozlitina et al[10] found, three variants to be associated with higher liver fat levels: two in the aforementioned PNPLA3 and one in the TM6SF2 gene, which is required for normal VLDL secretion.[11] In a study by Mahdessian et al [15] the TM6SF2 gene was found to be a regulator of liver fat metabolism, which influenced triglyceride secretion and hepatic lipid droplet content.

Risk Factors and Aetiology

Gender and Age

The prevalence of NAFLD increases with increasing age and the incidence of NASH and cirrhosis also increase in those patients who are 50 years or greater compared to younger age groups.[4] Several studies provide data to suggest a higher prevalence in males while others proposed the opposite.[10] However, according to Lonardo et al.[16] epidemiological review, NAFLD is more common in men and has been shown to increase in younger to middle aged people with a decline after the age of 50-60 years. It has been shown to rise in incidence after the age of 50 with a peak at 60-69 years in women.[16] Notably, it has been suggested that NAFLD begins in utero based on several studies, using magnetic resonance spectroscopy, showing steatosis in infants born to mothers with gestational diabetes (GD).[17] A study with 191 Italian children with biopsy confirmed NAFLD, showed hepatic steatosis, inflammation, hepatocyte ballooning, and fibrosis. Those pathologies were worse in those children who were non-breastfed compared to those who were breastfed.[18]

Ethnic differences

The rate at which NAFLD develops has been shown to be greatest in Hispanic patients.[19] Also, NAFLD in the Asian population has been increasing, and interestingly, can be seen in those who have a normal body mass index.[19] In a United States based study, the investigators found a lower degree of steatosis

in African Americans when compared to whites and also showed a higher grade of NAFLD findings in Asians and Hispanics.[20] The Hispanic population also has been shown to have a higher occurrence of steatohepatitis and cirrhosis, while African Americans have a decreased chance of developing liver failure.[20]

Diet, smoking and life style

Diets that model after a Westernized pattern, such as those high in red meat consumption, refined grains, pastries, and sugar laden beverages are associated with a greater likelihood for the development of metabolic syndrome and subsequent NAFLD.[20] It has been shown, through energy restriction and manipulation of dietary macronutrients, namely, restriction of carbohydrates, fat, or enrichment with mono unsaturated fatty acids, that dietary modifications can reduce metabolic syndrome.[21] Dietary supplementation with É-3 polyunsaturated fatty acids (n-3 PUFAs) has also been shown to decrease liver fat, so fish oil supplementation could be a simple therapeutic option but further studies are needed. The use of tobacco predisposes a person for the development of insulin resistance.[21] As to life style, associations have been shown between a person's fitness and sedentary behavior with the risk of developing NAFLD and NASH; the severity of NAFLD also intensifies with lower physical activity.[21]

Polycystic ovarian syndrome

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder in reproductive aged women and is typically characterized by obesity and insulin resistance.[22] Hence, women with PCOS are at a heightened risk of developing T2DM. In a study that evaluated 600 women with PCOS and 125 body mass index (BMI)-matched healthy control women, the prevalence of NAFLD was found to be higher in those with PCOS.[22] Women with PCOS are typically hyperandrogenemic and insulin resistance worsens the hyperandrogenemia by increasing ovarian androgen synthesis and decreasing liver SHBG (Sex Hormone Binding Globulin) production, resulting in elevated circulating free androgens. The subsequent hyperandrogenemia is associated with a more prominent insulin resistance in patients with PCOS, which endangers them for developing NAFLD.[23]

Metabolic syndrome and type 2 diabetes mellitus

Metabolic syndrome is a conglomerate of cardiovascular risk factors which predispose a person to develop type 2 diabetes and cardiovascular disease.[23] In fact, it has been stated that the incidence of NAFLD increases with increasing number of metabolic syndrome.[23] When compared to non-diabetic patients (matched for age, sex, and body weight), type 2 diabetes mellitus (T2DM) patients have liver fat contents 80% higher.[16] Interestingly, it has been shown that T2DM patient with NAFLD can have normal liver function tests, which may lead to believe people that the prevalence of NAFLD might be higher in T2DM patients than it is reported.[16]

Obstructive sleep apnea

Obstructive sleep apnea (OSA) is characterized by complete or partial airway obstruction caused by pharyngeal collapse during sleep.[17] A budding association of OSA with diabetes mellitus, metabolic syndrome, and cardiovascular disease has started to appear in last few years.[17] The pathogenic mechanisms that underpin this association is believed to be due to the alteration of gas exchange (repetitive hypoxemic and hypercapnic events), termed chronic intermittent hypoxia, which can lead to an increase in pro-inflammatory cytokines, endothelial dysfunction, oxidative stress, metabolic dysregulation, and finally insulin resistance.[16] Investigational evidence has suggested that chronic intermittent hypoxia may trigger liver injury, inflammation, and fibrogenesis with several studies showing the relationship between OSA and NASH.[17]

Pathogenesis of NAFLD to NASH

NAFLD is regarded as a hepatic symptom of metabolic syndrome (MetS) and is associated with visceral obesity, abnormalities in glucose and lipid metabolism, insulin resistance (IR), and hypertension.[18] In NAFLD patients, underlying metabolic conditions such as those described above result in worsening liver dysfunction and a higher incidence of liver fibrosis and may also lead to cardiovascular disease (CVD).[18] Though pathogenesis of NAFLD still remains unclear, "two-hit" theory has been proposed to explain the progression.[19] The **"first hit"** is hepatic steatosis that excessive lipid accumulation in hepatocytes is accompanied by elevated de novo lipogenesis and fatty acid uptake. Subsequently, oxidative stress and

hepatic inflammation are critical two factors of the **"second hit"** which cause remarkable hepatic cellular damage. Besides, multiple hits including genetic mutations and intestinal microbiome also account for the progression of NAFLD.[20]

Advanced glycation end-products (AGEs) might be involved in the mechanism that links NASH and diabetes mellitus (DM). Accumulating evidence indicates that in diabetic patient chronic hyperglycemia upregulates the production of AGEs (senescent macro protein derivatives) via non-enzymatic glycation.[21,22] It has been demonstrated that the binding of AGEs to their receptor (RAGE) induces oxidative stress followed by inflammatory and/or thrombogenic responses in a variety of cell types. Furthermore, in diabetes such binding is considered to be involved in the pathogenesis and worsening of angiopathic conditions.[21,22] In addition, there is a growing consensus that TAGE (Toxic advanced glycation end-products) -RAGE interactions affect gene expression, intracellular signaling, and the secretion of pro-inflammatory factors and induce reactive oxygen species (ROS) production in various cell types including hepatic stellate cells (HSC) and hepatocytes.[23] Therefore, it is suggested that TAGE signaling through RAGE and the subsequent ROS production play a role in the worsening of hepatic pathology observed in NASH.

Histopathology

Non-alcoholic fatty liver disease shows a wide range of histologic manifestations, which can range from a very mild steatosis (5% or more of hepatocytes involved), to more aggressive forms showing lobular and/or portal inflammation, ballooning hepatocytes, fibrosis, and ultimately cirrhosis.[24] The presence of less than 5% of steatosis is not regarded as clinically significant. In adult patients, steatosis typically affects the centrilobular hepatocytes first; whereas in children the periportal or panacinar patterns are more likely seen.[25] Steatosis comes in a few morphologic appearances, the macrovesicular terminology is used when large lipid droplets inhabit the cytoplasm and displace the nucleus.[24] However, macrovesicular steatosis also encompasses small lipid droplets, varying in size and keep their nuclear central location.[25] Finally, the terminology of microvesicular steatosis denotes the accumulation of innumerable lipid droplets with the hepatocyte nucleus remaining essentially in its original location.[26] Some mild NAFLD cases encountered in adults have shown a very mild degree of fibrosis, mainly centered on the portal area or occasionally zone 3.[27] It is still unclear whether the patients with NAFLD (i.e., not NASH) and a mild component of inflammation/ fibrosis have as benign of a course when compared with those who have steatosis alone.[26] In order to provide a consistent and reproducible assessment of NAFLD, the evaluation of morphological features should be semi quantified via an agreed upon scoring system to guide clinical decision making and for use in clinical trials.[28] Three histological scoring systems are currently in place: NASH clinical research network's NAFLD activity score (NASH CRN-NAS), steatosis activity, and fibrosis (SAF), and the Brunt staging system.[28]

Principles of Treatment of NAFLD

All patients with NAFLD require advice about lifestyle modification aimed at weight loss and increased physical activity, as well as treatment of any associated metabolic risk factors (diabetes, hypertension and dyslipidaemia). Management of patients with NAFLD depends largely on the stage of disease, emphasizing the importance of careful risk stratification.[29] There are five main areas to focus on when thinking about treatment strategies of NAFLD: life style modification, weight management, therapy targeting the components of the metabolic syndrome, liver-directed pharmacotherapy and treating the complications of cirrhosis.

1) Lifestyle Modification

Lifestyle modification aimed at weight loss and increased physical activity is vital in managing all patients with NAFLD irrespective of their underlying liver histology. Patients should be encouraged to avoid a sedentary lifestyle by increasing daily activities, undergoing regular exercise and eating healthy food. In one study, patients who received dietary advice and undertook 200 min moderate physical activity per week for 48 weeks had an overall 9.3% reduction in body weight (versus 0.2% in the control arm) and had improvement in hepatic steatosis and inflammation on liver biopsy.[30]

Diet

The optimum diet to treat NAFLD is not known. Until further evidence is available, a calorie restricted diet (600 Kcal less than a person needs to remain at the same weight) should be recommended aiming to lose 0.5–1 kg per week until the target weight is achieved.[31] Patients with NAFLD should be advised

to avoid saturated fats, simple carbohydrates and sweetened drinks.[32] A Mediterranean diet (high in monounsaturated fatty acids), as compared with a diet low in fat and high in carbohydrates, has been shown to reduce hepatic steatosis and improve insulin sensitivity in non-diabetic subjects with NAFLD.[31] Dietary supplementation with É-3 polyunsaturated fatty acids (n-3PUFAs) has also been shown to decrease liver fat.[22]

Exercise

Although the optimum exercise to treat NAFLD is not known, studies examining moderate intensity training, high intensity training and resistance exercise have shown improvement in liver enzymes and reduction in liver fat, independent of weight loss, but the effects on histology remain unknown.[33] Aerobic exercise increases skeletal muscle insulin sensitivity and as a result reverses one of the key patho-physiological mechanisms that cause NAFLD (insulin resistance).[34] Therefore, all patients with NAFLD should be advised to increase physical activity and undertake regular exercise. Until further evidence is available, one approach is to recommend 30 min of moderate exercise five times per week.[31]

2) Weight Management

Orlistat, a gut lipase inhibitor, reduces absorption of dietary fats, and is approved in obesity as an aid to weight loss in conjunction with a hypocaloric diet and lifestyle modification.[35] Several studies have investigated orlistat in NAFLD, with conflicting results; one study comparing 9 months of hypocaloric diet plus vitamin E with diet plus thrice daily 120 mg orlistat showed improved insulin sensitivity (P<.001), adiponectin (P=.03), steatosis (P=.005), ballooning (P=.04), inflammation (P=.045), and NAS histology score (P=.009) in those who lost d"9% body weight, but showed no correlation with the use of orlistat.[35] National Institute of Health and Care Excellence (NICE) guidelines[36] for obesity recommend that orlistat can be considered as an aid to weight loss in subjects who have not achieved their target weight by lifestyle modification and have a BMI>30 kg/m^2. So, it seems reasonable to consider this treatment in patients with obesity and NAFLD. Treatment duration should not be greater than 1 year as there is potential for fat soluble vitamin deficiency if continued for longer.[35]

Bariatric surgery

Bariatric surgery has an increasing role in the management of patients with obesity and the metabolic syndrome. Restrictive procedures such as the gastric band, gastric balloon and sleeve gastrectomy decrease stomach size leading to early satiety. Mal-absorptive procedures such as gastric bypass limit the absorption of food. Weight loss after bariatric surgery has beneficial effects on the components of the metabolic syndrome including improving insulin sensitivity, lipid profile as well as reducing long-term mortality.[37] It also has specific effects on liver histology including reduced steatosis, steatohepatitis and fibrosis.[38] The majority of the histological benefits occur within the first year post surgery.[39] As there is a lack of long-term outcome data about bariatric surgery as a specific treatment for NAFLD, bariatric surgery cannot be considered as a primary treatment for NASH.[40] However, NICE guidance suggests that surgery should be considered as a treatment for obesity for patients with BMI >40 kg/m^2 or between 35 and 40 kg/m^2 with other significant disease that could be improved with weight loss.[36] Bariatric surgery should be avoided in subject with advanced cirrhosis with portal hypertension as there is a risk of hepatic decompensation with rapid weigh loss.[41]

3) Liver-directed Pharmacotherapy

For patients with biopsy-proven NASH, where lifestyle modification has failed, liver-directed pharmacotherapy with pioglitazone or vitamin E can be considered.

Pioglitazone

Several studies have demonstrated benefit from the thiazolidinediones in patients with NASH both with and without diabetes. The optimum dose and duration of treatment are not known.[42] Aithal et al showed that 12 months of treatment with pioglitazone 30 mg/day reduced hepatocellular injury and fibrosis compared with placebo.[42] A recent meta-analysis has demonstrated that pioglitazone treatment in NASH significantly improves steatosis, inflammation and to a lesser degree, fibrosis.[43] Treatment with pioglitazone is associated with weight gain (4.7% in the PIVENS**;** Pioglitazone, Vitamin E, or Placebo for non-alcoholic Steatohepatitis study)[43] and there have been reports of increased risk of congestive cardiac failure,[44] bladder cancer and reduced bone density.[45] However, a meta-analysis of 16390 patients with type 2 diabetes mellitus (T2DM) treated with pioglitazone demonstrated an 18% reduction in death, myocardial infarction and stroke.[46] Therefore, after assessing risk and benefit, treatment with pioglitazone can be recommended cautiously for patients with more aggressive NASH who have failed lifestyle modification.[46]

Vitamin E

Vitamin E is an antioxidant that has recently been shown to have beneficial effects on histology in non-diabetic patients with NASH. In the large PIVENS trial, significantly more patients had improvement in steatohepatitis following 96 weeks of vitamin E800 IU/day compared with placebo (42% vs. 19%; p<0.001).[43] Although vitamin E looks like a promising treatment for NASH, there are concerns about the long-term effects of vitamin E with a meta-analysis showing a small overall increase in all-cause mortality at doses >400 IU/day.[47] It has also been reported that there might be an increased risk of hemorrhagic stroke and prostate cancer[48] with high dose Vit E treatment. Vitamin E has not been evaluated in patients with cirrhosis or diabetes and NASH. Therefore, it should currently be reserved for selected patients with more advanced pre-cirrhotic NASH who have failed lifestyle modification. The optimum dose and duration of treatment are not known.

4) Medications Currently Used in Patients of NAFLD with Co-morbidities

Hypertension

Approximately 70% of patients with NAFLD have hypertension[49] and so it is imperative to screen NAFLD for all patients having hypertension. If the blood pressure is >140/90 mm Hg, patients should be managed according to NICE hypertension guidelines.[50] ACE inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) are recommended as first-line for patients less than 55 years old, but calcium channel blockers are recommended for people over 55 years and those of Afro-Caribbean origin.[49] However, targeting the renin–angiotensin system (RAS) might be beneficial in all patients with NAFLD as the RAS plays a role in liver fibrogenesis and blocking the RAS reduces fibrosis in experimental models.[51] Further larger studies are needed to confirm these observations, but ACEI or ARBs seem a logical choice in the management of hypertension in subjects with NAFLD.

Dyslipidaemia

Dyslipidaemia is very common in patients with NAFLD and the metabolic syndrome. Dyslipidaemia should be managed according to evidence-based guidelines and should reduce cardiovascular morbidity and mortality in patients with NASH.[49] Statins are not only safe in NASH but are also associated with a reduced mortality.[52] Fibrates are synthetic agonists of peroxisome proliferator-activator receptors (PPAR) , but have not shown benefit in NAFLD other than the management of hypertriglyceridaemia.[53] NICE guidelines[52] recommend simvastatin treatment as primary prevention for cardiovascular disease in adults with a ≥20% 10-year risk of developing cardiovascular disease (using a risk calculator such as the Framingham risk calculator). More aggressive lipid control is recommended for secondary prevention of cardiovascular disease, aiming for a total cholesterol<4 mmol/L. In a large cardiovascular outcomes study, statins were shown to improve liver enzymes and cardiovascular outcomes in patients with raised liver function tests (LFTs) due to NAFLD.[54] Table 1 summarizes the management of hypertension and dyslipidemia in NAFLD.[55]

Table 1. Management of hypertension and dyslipidaemia in NAFLD[55]

Risk factor	Treatment/indication	Outcome
Hypertension	ACEI and ARBs first-line if BP >140/90 mm HgEscalate treatment according to NICE hypertensionguidelines.	Blocking RAS reduces hepatic fibrosisARBs improve transaminase levels and insulin sensitivity20% reduction in new onset T2DM with ACEI or ARBs
Dyslipidaemia	Primary prevention with statin if e"20% 10-year risk of developing cardiovascular disease. If secondary prevention, aim total cholesterol <4 mmol/L	Statins reduce 5-year incidence of all-cause mortality, major coronary events, coronary revascularisation and stroke by about 20% per mmol/L reduction in LDL cholesterol. May reduce incidence of HCC

ACEI, ACE inhibitor; ARB, angiotensin receptor blocker; BP, blood pressure; HCC, hepatocellular carcinoma; LDL, low-density lipoprotein; NAFLD, non-alcoholic fatty liver disease; NASH, non-alcoholic steatohepatitis; T2DM, type 2 diabetes mellitus; RAS, renin–angiotensin system.

Diabetes

Metformin

Metformin is the first line agent for type 2 diabetes mellitus (T2DM) and reduces the risk of all diabetes-related end-points including microvascular disease, myocardial infarction, large vessel disease, and cardiovascular mortality, in addition to aiding weight loss.[56] Although studies have not demonstrated any improvement in liver enzymes or liver histology,[57] there is epidemiological evidence to suggest it is associated with a reduced incidence of both liver and non-liver malignancies including hepatocellular carcinoma in those with NASH cirrhosis by 7%.[58]

Glucagon-like peptide-1

Glucagon-like peptide-1 (GLP-1) is a gut-derived hormone analogue that induces insulin secretion, reduces glucagon secretion, suppresses appetite and delays gastric emptying.[59] This class of drugs is licensed for the treatment of diabetes and obesity, improvement of both of which is desirable in patients with NASH.[59] GLP-1 analogues have been shown to improve liver histology in murine models of non-alcoholic steatohepatitis, reduce liver enzymes in patients with T2DM and in clamp technique,[60] lead to a reduction in de novo lipogenesis and an increase in fatty acid oxidation in the liver.

Liraglutide is a long-acting GLP-1 analogue which has been shown to induce improvements in peripheral, hepatic and adipose insulin resistance, and reductions in de novo lipogenesis in subjects with NASH.[61] But gastrointestinal side effects were common with liraglutide, which was the primary reason for discontinuation of treatment.[62]

DPP-4 inhibitors

Dipeptidyl peptidase-4 (DPP-4) rapidly degrades endogenous GLP-1, and a pilot study of sitagliptin, a selective DPP-4 inhibitor, demonstrated an improvement in liver histology in diabetic patients after 1 year of treatment.[63] But only 15 participants were enrolled and there was no control arm. Reduction in liver fat content using magnetic resonance spectroscopy (MRS) has also been demonstrated following 24 weeks of sitagliptin or vildagliptin therapy.[64] Sitagliptin 100 mg once daily for 1 year ameliorates NAS by improving steatosis and ballooning, irrespective of diabetes. Sitagliptin has stronger efficacy than that of weight reduction.[64] However, a number of studies have failed to show an effect of sitagliptin treatment on liver fat content, liver enzymes or liver stiffness[65] and currently its use is reserved for the management of diabetes.

Sodium glucose co-transporter 2 inhibitors

The sodium glucose co-transporter 2 (SGLT2) is primarily expressed in the renal proximal tubules, and reabsorbs 90% of glucose filtered at the renal glomeruli; inhibition therefore, facilitates urinary glucose excretion, and SGLT2 inhibitors are used in diabetes to improve plasma glucose levels and promote weight loss.[66] A meta-analysis of placebo controlled randomized control trials for SGLT2 inhibitors showed improved all-cause, and cardiovascular mortality, for empagliflozin but not all agents, suggesting it may not be a class effect. Several SGLT2 inhibitors have shown benefit in murine models of NAFLD, improving steatosis, inflammation, and fibrosis[67] and studies in humans with T2DM have demonstrated improved ALT and weight loss in patients with type 2 diabetes with ipragliflozin and canagliflozin[68] as well as reducing fatty liver index score with ipragliflozin.[69] There are no reported human studies assessing changes in liver histology in NAFLD with SGLT2 inhibitors, although one study[66] did demonstrate weight-independent improvements in serum ALT with ipragliflozin.

Rising demand and an improved understanding of NASH patho-physiology has led to a surge in development of new therapies. The therapeutic landscape for NASH is evolving rapidly with many compounds currently being assessed in phase II/III clinical trials.[69]

5) Treatment of Complications of Cirrhosis

Varices

Cirrhotic patients with NASH are at risk of varices to develop as with other liver diseases and their presence correlates with the severity of liver disease (40%-44% if Child–Pugh grade A, 75%-85% if Child–Pugh grade C).[70] Therefore, patients with NASH cirrhosis should undergo endoscopic screening for oesophageal and gastric varices at the time of diagnosis and at regular intervals according to British Society of Gastroenterology or American Association for theStudy of Liver Diseases guidelines.[71]

Hepatocellular Carcinoma (HCC)

The yearly cumulative incidence of HCC is 2.6% per year in patients with NASH cirrhosis. Therefore,

surveillance with abdominal ultrasonography (USS) should be performed every 6 months.[72] CT or MRI could be considered in subjects where ultrasound has failed to produce adequate examination of the liver, but these modalities have their own limitations in obese patients. ±-Fetoprotein is a tumor biomarker and has recently been removed from the European guidelines but is still widely used in clinical practice alongside 6-monthly ultrasound.[72] There is a clear association between obesity and malignancy[73] and hyperinsulinemia is well recognized as an independent risk factor for many malignancies. Metformin acts by inhibiting hepatic glucose production through an LKB1–AMPK-mediated mechanism. There is emerging evidence that metformin reduces the risk of cancer (including HCC) in patients with diabetes in a dose-dependent manner.[74]

There is also evidence that statins might reduce the incidence of HCC.[75] Statins appear to have anti-cancer effects through both 3-hydroxy-3-methyl-glutaryl-CoA (HMG-CoA) reductase dependent and HMG-CoA reductase independent pathways. They have a variety of actions including: anti-inflammatory and immune modulatory effects, inhibiting downstream production of mediators of cell growth and promoting programmed cell death.[75]

Liver transplantation

NASH cirrhosis is now the third commonest indication for liver transplantation in the USA and accounted for 12% of patients listed for transplantation in the UK in 2009.[74] Patient and graft survival in liver transplantation for NASH are comparable with other indications.[76]

Updates on Treatment of NAFLD and NASH

The therapeutic landscape for NASH is evolving rapidly with many compounds currently being assessed in phase II/III clinical trials:

1) Farnesoid X receptor bile acid axis

The nuclear hormone farnesoid X receptor (FXR) is primarily expressed in the liver, intestines, and kidneys, and has a key role in bile acid synthesis, and also influences carbohydrate and lipid metabolism, and insulin sensitivity.[77]Obeticholic acid (OCA) is a synthetic variant of the natural bile acid chenodeoxycholic acid, a potent activator of the farnesoid X nuclear receptor, which negatively regulate bile acid synthesis and down-regulates lipogenesis.[78] The FLINT study, a multicentre, phase IIb clinical trial conducted in subjects with NASH (NAS>4), compared placebo (n=142) with 25 mg OCA (n=141) for 72 weeks. OCA met the pre-defined stopping rule for efficacy, namely reduction in the NAFLD activity score (NAS) by 2 points with no worsening in fibrosis, although it did not impact on resolution of NASH (22% in OCA arm vs. 13% in placebo arm; P=.08). Notably, OCA improved fibrosis stage in 35% of patients vs. 19% (P=.004) in the placebo arm.[79]

2) Bile acid sequestrants /transporter inhibitors

Bile acid sequestrants bind bile acids in the gut and can be used to treat dyslipidaemia and hyperphosphataemia, although more recently these agents have been reported to lower blood glucose and increase insulin sensitivity through altered bile acid signaling pathways, possibly mediated by the presence of increased bile acids in the distal colon stimulating GLP-1 and peptide YY.[80]

3) Hormone signaling

Fibroblast growth factor 21 is a hormone secreted predominantly from the liver, which acts primarily in the fasting state to coordinate carbohydrate and lipid metabolism, enhance insulin sensitivity, decrease triglyceride levels, and cause weight loss, thus ameliorating obesity-associated hyper-glycaemia and hyperlipidaemia.[81]

4) Anti-inflammatory and anti-apoptotic agents

Cenicriviroc, a C-C chemokine receptor types 2 and 5 antagonists, has been shown to reduce CD14 which is involved in inflammatory cell activation, and improve the aspartate Aminotransferase-to-Platelet Count Ratio Index (APRI) and fibrosis-4 scores in patients with HIV.[82]

5) Inhibition of de novo lipogenesis ("Lipid altering")

Aramchol is an arachidic and cholic acid conjugate that was shown to inhibit stearoyl CoA desaturase in vitro and de novo lipogenesis in animal models on high fat diet.[83]

6) Targeting the gut microbiome

IMM-124e is an IgG-rich extract of bovine colostrum from cows immunised against lipo polysaccharide (LPS), and is believed to reduce exposure of the liver to gut-derived bacterial products and LPS.[84]

Conclusion

NAFLD is a growing epidemic in worldwide in part due to obesity and insulin resistance leading to liver accumulation of triglycerides and free fatty acids. Between 10% and 30% of patients with NAFLD have non-alcoholic steatohepatitis (NASH) that can progress to cirrhosis. Additionally, access and decreasing cost for high quality and powered genetic scrutiny will no doubt provide future clinicians with a great deal of information and opportunity for enhanced targeted treatment. Patients should be encouraged to avoid a sedentary lifestyle by increasing daily activities, undertaking regular exercise and eating healthy foods in the defense of developing NAFLD. Patients who develop advanced disease, they require surveillance and management of the complications of cirrhosis. Currently several medications/supplements are being used in the treatment of NAFLD; however, none seem to be the "magic bullet" in curtailing this growing problem and it needs further research in understanding this disease and better managing NAFLD patients.

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A Middle Aged Woman with Giant Infected Hydatid Cyst in the Liver

Reza ST[1], Shahriar S[2], Jahan A[3], Hossain MI[4], Jahan SN[5]

Abstract

Hydatid cyst is a zoonotic disease caused by larvae of Echinococcus granulosus. It produces cyst in the liver and other organs. A middle aged woman was suffering from upper abdominal pain for a long time and was treated as peptic ulcer disease. Later, she was diagnosed as a case of giant cyst in the liver. She was admitted in MH Samorita Hospital and Medical College and underwent surgery. During operation, cyst wall was found thickened and organized pus came out with few scolices. The patient recovered very soon and discharged with medication and advices. Hydatid cyst may be undiagnosed as symptoms may mimic other common diseases like peptic ulcer disease or chronic cholecystitis. Ultrasound and CT imaging could identify the disease early. Surgery is needed in complicated cases.

Key words: *Giant Infected Hydatid Cyst, Liver.*

(MH Samorita Med Coll J 2019; 2(1): 32-34)

Introduction

Hydatid disease is an infection caused by a tapewarm, Echinococcus granulosus. The larvae reside in the liver and lung and produce fluid filled cyst. E. granulosus produces single, unilocular cyst. Free floating protoscolices (hydatid-sand) are common within the cyst cavity[1].

E. Granulosus requires two host types, definitive host and an intermediate host. The definitive host of this parasite are dogs and the intermediate host are most commonly sheep. However, cattle, horses, pigs, goats, and camels are also potential intermediate hosts.[1] Humans can also be an intermediate host for E. granulosus. However, this is uncommon and therefore humans are considered an aberrant intermediate host.[1]

E. granulosus is transmitted from the intermediate host (sheep) to the definitive host (dogs) by feeding of offal, also referred to as "variety meat" or "organ meat". Consuming offal containing E. granulosus can lead to infection, however, infection is dependent of many factors.[2] Eggs being shed in its scat . E. granulosus is ingested by dog and attaches to the mucosa of the dog's intestine and there the parasite grows into the adult one.[3]

Life cycle:

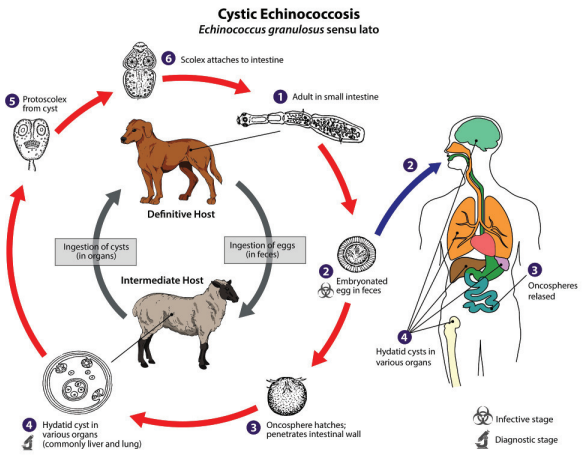

Fig.-1

1. Dr. Syed Tariq Reza, Assistant Professor, Critical Care Medicine, Dhaka Medical College, Dhaka.
2. Dr. Shaon Shahriar, Junior Consultant, Surgery, OSD, DGHS
3. Dr. Ahsina Jahan, Registrar, ICU, MH Samorita Hospital & Medical College.
4. Dr. Md. Iqbal Hossain, Associate Professor, Department of Forensic Medicine, MH Samorita Hospital and Medical College, Dhaka.
5. Syeda Nusrat Jahan, Lecturer, Community Medicine, Shaheed Suhrawardy Medical College, Dhaka.

Address of Correspondence: Dr. Md Iqbal Hossain , Associate Professor, Department of Forensic Medicine, MH Samorita Hospital and Medical College, Dhaka. Email- iqbalsacrifice @yahoo.com; Phone-+8801711123997

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The life cycle alternates between hervivores and carnivores. Adult E. granulosus releases eggs within the intestine of dog which is transported out of the body via faeces. The sheep ingests the eggs and the eggs hatch in its small intestine. The larvae passes through the wall of small intestine and enter the blood and reach the liver. In the liver they produce cyst. The larvae can travel to lung, brain and other organ. When dogs eat the sheep viscera infested with hydatid cyst, the protoscolices attach to the small intestine of the dog. Then the protoscolices begin to form proglottids. The gravid proglottids lay eggs. The eggs pass out in the faeces of the dog. This way life cycle continues. Animals like cows and sheep again are infested by contaminated grass. Man is the accidental host. Contaminated vegetables are one of the common sources of infestation in man.

Case report

A 47-year-old lady living in Dhaka city was admitted in MH Samorita Hospital and Medical College on 7th May'2018 with the history of mild to moderate upper abdominal pain off and on for 3 years. She took analgesic and antiulcer drugs but did not get any improvement. She was normotensive, nondiabetic, a known case of subclinical hypothyroidism and osteo arthritis (OA). She had hysterectomy 7 years back. . On examination, patient was mildly pale, had mildly enlarged liver and otherwise normal findings. Then she was advised for some investigations. She had Hb 11.6 gm/dl, high ESR (100 mm in 1st hour), and TSH was mildly elevated (4.2 mIU/L). Her USG of abdomen revealed a large hepatic SOL measuring 7.7×7 cm (Fig 1). CT scan of abdomen showed a giant hepatic cyst in right lobe of liver (Fig 2). IHA (indirect hemagglutination test) for Echinococcus was negative. Upper GI endoscopy was normal. After diagnosis of a giant hepatic cyst in the liver, she has been operated at MH Samorita Hospital and Medical College. With all aseptic precaution abdomen was opened by right subcostal incision and a large cyst was found in segment VII and VIII of liver. Cyst wall was thickened contained organized thick pus and a few scolices. De-roofing was done with marsupialization, pus was sent for C/S and cyst wall for histopathology. The post operative period was uneventful and the patient got recovery within 3 days. Later C/S report was found negative and histopathology showed inflammatory cells without any malignancy. The patient was discharged with anthelmintic drug and advised for follow up.

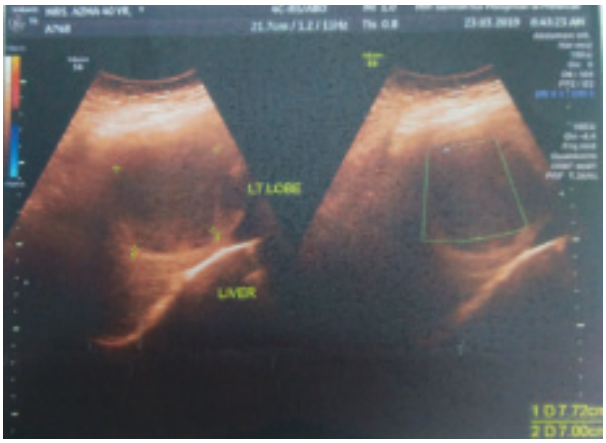

Fig. 1: *Cyst in right lobe of liver*

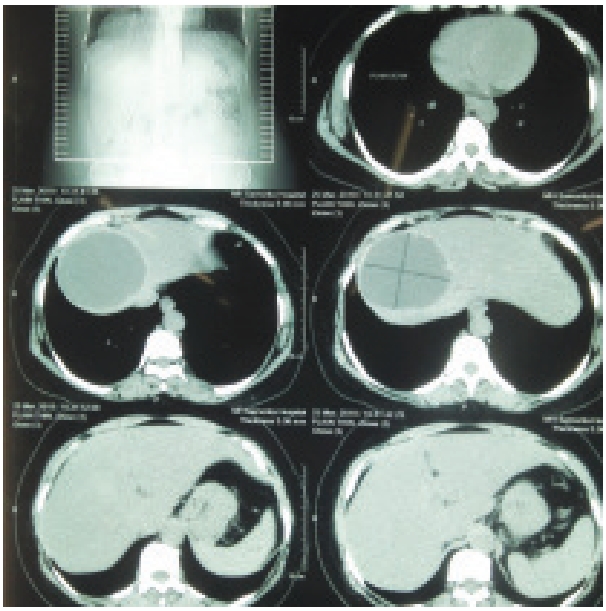

Fig. 2: *Giant cyst in the liver*

Discussion

Hydatid cyst, caused by Echinococcus is seen worldwide. More than 1 million people are affected with echinococcosis. It is common in some part of the world like Eurasia (the Mediterranean regions, southern and central parts of Russia, central Asia, China), Australia, some parts of America (especially South America) and north and east Africa.[4]

Ultrasonography is very sensitive to diagnose hepatic cyst in most cases. CT scan has a role in diagnosing complicated cases and screening of multiorgan involvement. Diagnosis of echinococcosis can also been done through serological tests. Indirect Hemagglutination test (IHA) and Enzyme linked immunosorbent assay

(ELISA) are two popular tests for the diagnosis. There are multiple treatment options for hydatid cyst. Medical therapy with anthelmintic drugs is most popular. PAIR (Percutaneous aspiration, irrigation with scolicide and re-aspiration) can be done in some cases. Surgery is the definitive treatment for large (> 5 cm), symptomatic and complicated cyst.

Conclusion

Hydatid cyst remains undiagnosed as patient is asymptomatic or have symptoms mimicking other common disease like peptic ulcer diseases. Strong clinical suspicion and radiological evaluation is necessary for early diagnosis and treatment. Surgery is the definitive treatment for large and complicated cyst.

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Sirenomelia (Mermaid Syndrome) - A Case Report of a Rare Congenital Malformation

Haque GMI[1], Yeasmin N[2]

Abstract

Sirenomelia, also known as sirenomelia sequence or Mermaid Syndrome, is a birth defect of the lower part of body characterized by apparent fusion of legs into a single lower limb. It is found in approximately 1:100000 live births, 15% of which are associated with twin and most often monozygotic. The cases are usually fatal within first day because of agenesis or abnormal development of urinary system and failure to develop normal vascular branches of abdominal aorta. Maternal diabetes has been associated with caudal regression syndrome and sirenomelia. Throughout the world nine mermaid syndrome have been reported surviving after reconstructive surgery. The most important characteristics of survival of the affected newborn is the presence of functional kidney. Here, we report a case, born to a primigravida mother. Diagnosis was made according to clinical findings. X-Ray and ultra-sonogram were done to confirm the case.

Key words: *Sirenomelia, mermaid syndrome, caudal regression syndrome.*

(MH Samorita Med Coll J 2019; 2(1): 35-38)

Introduction

Sirenomelia is a rare and fatal congenital anomaly. According to Greek mythology, the Sirens were the creatures with the head of a woman and the body of a bird from wings downward.[1] They were dangerous to sailors, whom they narcotized with their enchanting music voices to later kill them.[1] Over time, these bird-women were portrayed as more aquatic creatures, and eventually with a full mermaid-like appearance. In humans, fusion of the legs in sirenomelia occurs in a spectrum of morphologies, ranging from the mildest cases, in which all bones of the two fused lower limbs are discernible to the most severe cases, in which there is only single rudimentary lower limb that is derived from fusion of the two.[1] Sirenomelia is a multisystem condition with severe malformations of the gastrointestinal, genitourinary, cardiovascular and musculoskeletal systems.[2] Duhamel in 1961 defined all anomalies of Mermaid syndrome and commented as more severe form of caudal regression syndrome.[3] Caudal regression syndrome is a rare disorder which represents a spectrum of congenital malformations ranging from lumbosacral agenesis to the most severe case of fused lower extremities.[4]

Owing to visceral abnormalities, sirenomelia is usually incompatible with life; death occurs in the perinatal period.[5] The diagnosis, which is obvious at birth, is currently performed by antenatal ultrasonography. Antenatal ultrasonography clues include oligohydramnios, renal agenesis and a fibula positioned between the tibiae. All human cases of sirenomelia analyzed thus far show a variable degree of renal and urethral dysplasia, with total renal agenesis being very frequently reported.[5] Malformation of the urinary tract is consistently associated with genital malformations. These mainly affect the external genitalia, which are either absent or represented by an indistinct tag of tissue, whereas the gonads are usually unaffected.[6] The presence of gastrointestinal anomalies in sirenomelia is also common, the most frequent being a blind-end colon, rectal atresia and imperforate anus.[7]

Case Repot

Mina Akter, a primi gravida 25-year-old lady was on irregular antenatal check- up, hailing from Pallabi, Mirpur-12, admited at Islami Bank Hospital, Mirpur on 9th October 2016. She had USG for pregnancy profile before delivary, which revealed no

1. Dr. Gazi Mohammad Imranul Haque, Assistant Professor, Neonatology, Department of Paediatrics, MH Samorita Hospital and Medical College, Dhaka.
2. Dr. Nahida Yeasmin, Junior Consultant, Department of Gynae & Obs. Shahid Suhrawardy Medical College Hospital.

Address of Correspondence to: Dr. Gazi Mohammad Imranul Haque, Assistant Professor, Neonatology, Department of Paediatrics, MH Samorita Hospital and Medical College, Dhaka. Email-litons.imran@gmail.com; Phone-+8801712561176

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abnormality except severe oligohydromnios. LUCS was done due to less foetal movement at 36 weeks of gestation and delivered a baby with fused and tappered lower limbs from hip downwards which ended like a tip of a finger. There was ambiguaty of genitalia, presented like a slit. The baby cried immediately after birth and there was no respiratory distress. APGAR score was 6/10 at 1 minute and 8/10 at 5 minutes. Mother had no history of ingestion of tobaco leaves, no exposure to teratogenic agent, homeopathy or oral contraceptive. Mother was non-diabetic. Birth weight was 1420 grams with no facial dysmorphism. But Baby had gross anomaly in lower limbs in the form of fusion.There was absence of external genitalia. Imperforate anus with a swelling over anal region. A babygram was done which showed normal bone distribution in head, neck, thorax and upper extrimities. Upper vertibraes were normal but there was absence of hip bone and sacrum. There was presence of single lower extrimity bone probably femur without any distal bones. On ultrasonogram of abdomen, liver and spleen were present but genitourinary system were found to be absent. As patient party refused to take the baby to NICU, so managed in same hospital with IV infusion, Inj-ceftazidim and incubator care with other routine care. Random blood sugar was 4.1 m.mol/L. Baby developed gasping respiration about 14 hours after birth. Oxygen inhalation was started with mask @ 2 liters/min. Considering acidosis Infusion normal saline was given bolus @ 10 ml/kg. Inspite of all these efforts the baby died 15 hours after birth.

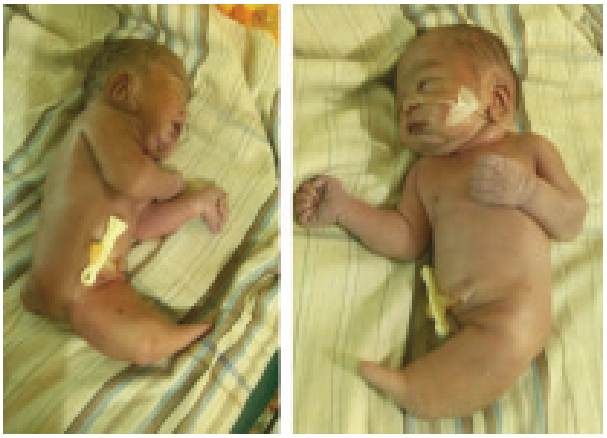

Fig. 1: *Fused lower limbs, no external genitalia,swelling of anal region without any anal opening.*

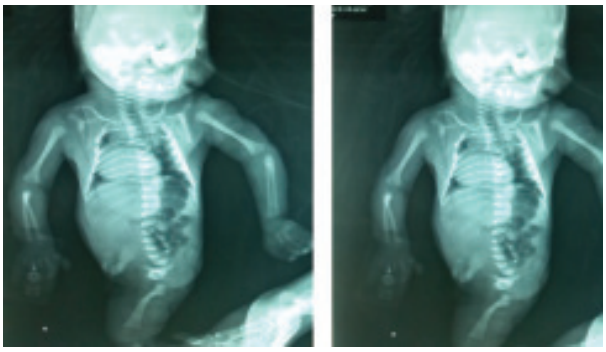

Fig. 2: *Babygram showing normal upper bony skeleton, absence of sacrum, hip bones, single femur, absent tibia, fibula and other distal bones.*

Discussion

Mermaid syndrome or Sirenomelia is a rare congenital anomaly.Till date around 300 cases have been reported in literature.[8] Most of those newborns were stillborn or died immediately after birth; death is due to renal agenesis, which is incompitible with life. Most of the time it is presented with lumbosacral agenesis.[9] In most severe cases of sirenomelia both sacral agenesis and caudal regression syndrome are related. Caudal regression syndrome has variable spinal anomalies varying from partial sacral agenesis to complete absence of lumbosacral spine.[9]

Various theories have been postulated to explain the aetiology of sirenomelia. The "pressure theory"[5] which proposed that some intrauterine force, probably amniotic, firstly acts on the tail of embryo. This leads to defective development of the caudal region including the allantoic structures. Ballantyne[10] supports this view, noting that the allantois fails to develop completely and that the placenta is vitelline in origin. Bolk[11] in 1899 proposed a theory of "primary failure", suggested that sirenomelia resulted from failure of development of the caudal somites leading to greater or lesser hind part deficiency. Weigert coined the theory of "nutritional deficiency".[5] He observed the vitelline nature of the umbilical artery and hypoplasia of the remaining vasculature.[5] In 1961 Duhamel proposed the theory of "Caudal regression" resulting in a spectrum of malformation including anal imperforation and the mermaid syndrome.[3] In 1970 Davies[12] suggested that sirenomelia results from injury to the caudal mesoderm between 28-32 days of fetal development. The primitive streak at the lumbar and sacral areas undergoes a developmental

arrest allowing the halves of the hind limb buds to move medially and dorsally fusing along their postaxial surfaces. Midline structures i.e. cloacal and urogenital derivatives are destroyed. This arrest of primitive streak due to unknown aetiology initiates formation of a second primitive streak, giving rise to a second normal embryo. This explains the association of sirenomelia in monozygotic twins. Smith supported this theory in 1980.[13]

Gardner and Brener[14] proposed a theory of "neural tube over distension". According to them neural tube over distension in the caudal area may lead to a roof plate expansion of the tube leading to lateral rotation of mesoderm by 180°. This results in fusion of the lower limb buds, closing off the midline primitive gut and urethra.[14] Stevenson in 1986[15] postulated that a "vascular steal" accounts for caudal regression. He dissected abdominal vasculature in 11 cases of sirenomelia and observed that in all cases there was a single umbilical artery that entered aorta in the upper abdomen. The vessels distal to this aberrant umbilical artery were under-developed and malformed resulting in diversion of blood flow and nutrients from all caudal structures of embryo to the placenta. The phenomenon is known as steal phenomenon of umbilical artery. This decreased delivery of nutrients cause subsequent developmental arrest or malformation.[15] Maternal diabetes increases the risk of congenital malformation in the offspring of affected pregnancies. This increase arises from the teratogenic effect of the maternal diabetic milieu on the developing embryo, although the mechanism of this action is poorly understood.[16,17]

There are three different variant of sirenomelia depending on degree of fusion of the lower limbs.[8]

1. **Symelia apus:** No feet are present and the limbs are completely fused into a single limb; One femur and one tibia are present. In this case there was no foot, lower limb was rather ended like a tip.
2. **Symelia unipus:** One foot is present (a partial fusion of both feet), 2 femur and 2 tibiae and fibulae.
3. **Symelia dipus:** Two feet are present giving the appearance of fins, hence the 'mermaid', foetuses for this condition. The fusion of the limbs extends as far as the ankles.

The condition is seen in 100-150 times more frequently in monozygotic twin or singletons and males are three times more affected than females.[16,17]

In sirenomelia, third trimester ultrasonography for diagnosis is usually hampered by severe oligohydromnios related to bilateral renal agenesis, whereas during early second trimester the amount of amniotic fluid may be sufficient to allow diagnosis earlier and less traumatic abortion.[18] Although sirenomelia has been described as a rare lethal anomaly, nine mermaid syndromes have been reported surviving after reconstructive surgery.[19] The most important characteristic of survival of the affected newborn is the presence of functional kidney.

Conclusion

Sirenomelia is a lethal congenital anomaly where neonates die within 24 hours of age without any medical intervention. For further details regarding visceral anomalies operational procedure in survivors and autopsy of dead foetus is required. Early termination of pregnancy may be possible if diagnosis is made early.

Conflict of Interest: No

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Abstract From Current Literatures

(MH Samorita Med Coll J 2019; 2(1): 39-41)

PRENATAL TETANUS, DIPHTHERIA, ACELLULAR PERTUSSIS VACCINATION AND AUTISM SPECTRUM DISORDER

Becerra-Culqui TA, Getahun D, Chiu V, Sy LS, Tseng HF

Pediatrics. 2018;142(3):e20180120. doi: 10.1542/peds.2018-0120.

Background: Increasing vaccination of pregnant women makes it important to assess safety events potentially linked to prenatal vaccination. This study investigates the association between prenatal tetanus, diphtheria, acellular pertussis (Tdap) vaccination and autism spectrum disorder (ASD) risk in offspring.

Methods: This is a retrospective cohort study of mother-child pairs with deliveries January 1, 2011 to December 31, 2014 at Kaiser Permanente Southern California hospitals. Maternal Tdap vaccination from pregnancy start to delivery date was obtained from electronic medical records. A diagnosis of ASD was obtained by using *International Classification of Diseases, Ninth* and *Tenth Revision* codes. Children were managed from birth to first ASD diagnosis, end of membership, or end of follow-up (June 30, 2017). Cox proportional hazards models estimated the unadjusted and adjusted hazard ratios (HRs) for the association between maternal Tdap vaccination and ASD, with inverse probability of treatment weighting to adjust for confounding.

Results: Women vaccinated were more likely to be Asian American or Pacific Islander, be nulliparous, have a higher education, receive influenza vaccination prenatally, and give birth at term. ASD was diagnosed in 1341 (1.6%) children, and the incidence rate was 3.78 per 1000 person years in the Tdap exposed and 4.05 per 1000 person years in the unexposed group (HR: 0.98, 95% confidence interval: 0.88-1.09). The inverse probability of treatment weighting-adjusted analyses revealed that prenatal Tdap vaccination was not associated with an increased ASD risk (HR: 0.85, 95% confidence interval: 0.77-0.95).

Conclusions: Prenatal Tdap vaccination was not associated with an increased ASD risk. We support recommendations to vaccinate pregnant women to protect infants, who are at highest risk of death after pertussis infection.

CEREBRAL OXYGENATION IN PRETERM INFANTS RECEIVING TRANSFUSION

Jain D, D'Ugard C, Bancalari E & Claure N

Pediatric Research 2018;85(6): 786-89.

Background: The influence of severity of anemia and cardiac output (CO) on cerebral oxygenation ($CrSO_2$) and on the change in $CrSO_2$ following packed red blood cell (PRBC) transfusion in preterm infants has not been evaluated.

Objectives: To evaluate the effect of pre-transfusion hemoglobin (Hb) and CO-weighted oxygen delivery index (ODI) on $CrSO_2$ and on the post-transfusion $CrSO_2$ change.

Methods: Preterm infants of <32 weeks gestational age (GA) receiving PRBC transfusion were enrolled. Infants received 15 ml/kg PRBC over 3 h. $CrSO_2$ by near-infrared spectroscopy and CO by electrical velocimetry were recorded for 1 h pre- ransfusion and post transfusion. ODI was defined as pre-transfusion Hb × CO.

Results: Thirty infants of 26.6 ± 2.0 weeks GA were studied at 19 ± 12 days. Pre- transfusion Hb was 9.8 ± 0.6 g/dl. Pre-transfusion CrSO2 correlated with pre- transfusion ODI (R_2 = 0.1528, p = .044) but not with Hb level. The pre- transfusion to post-transfusion $CrSO_2$ change correlated with pre-transfusion ODI (R2 = 0.1764, p = .029) but not with Hb level. CrSO2 increased from 66 ± 6% to 72 ± 7% post transfusion (p < .001), while arterial oxygen saturation, heart rate, and CO did not change.

Conclusion: In these infants, the pre-transfusion ODI was a better indicator of brain oxygenation and its improvement post transfusion than Hb alone. The role of CO and tissue oxygenation monitoring in assessing the need for transfusion should be evaluated.

ROLE OF BARIATRIC SURGERY IN REDUCING THE RISK OF COLORECTAL CANCER: A META ANALYSIS

Almazeedi S, El Abd R, Al Khamis A, Albatineh AN, Al Sabah S

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Background: Obesity increases the risk of multiple co-morbidities such as type 2 diabetes, cardiovascular disease and most cancers, including colorectal cancer.

Currently, the literature presents conflicting results regarding the protective effects of bariatric surgery on the incidence of colorectal cancer. This meta analysis was conducted to investigate the effect of bariatric surgery on the risk of developing colorectal cancer in obese individuals.

Methods: Ovid Embase, Ovid MEDLINE, Cochrane CENTRAL and Web of Science were searched for relevant articles. Articles published by the end of December 2018 were retrieved; data were extracted according to evidence based PICO (population, intervention, control, outcome) model and analysed using a random effects model to estimate the pooled relative risk (RR) and its 95 per cent confidence interval. The heterogeneity of studies was tested and quantified using Cochran's Q and I^2 statistics. Meta regression was used to investigate the association of year of study, region, mean length of follow up and sample size with RR.

Results: Seven articles, involving a total of 1 213 727 patients, were included in the meta analysis. The pooled estimate of the RR was 0·64 (95 per cent c.i. 0·42 to 0·98). The test of asymmetry found no significant publication bias. Meta regression showed that sample size was a statistically significant factor (P = 0·037), but year of publication, region and mean duration of follow up were not significant.

Conclusion: Patients who underwent bariatric surgery had a greater than 35 per cent reduction in the risk of developing colorectal cancer compared with obese individuals who had no surgery.

ASSOCIATION OF SERUM C-REACTIVE PROTEIN IN PREECLAMPSIA AND ITS EFFECT ON FETAL BIRTH WEIGHT – A CASE CONTROL STUDY

Sharmin S, Chowdhury S, Alam D, Banu N, Rashid F, Kabir S

Bangladesh J Obstet Gynaecol, 2016; 31(2): 75-80

Objective(s): The aim of this study was to evaluate the association of serum C-reactive protein (CRP) in preeclampsia (PE) and its effect on fetal birth weight.

Materials and methods: This case control study was conducted in Chittagong Medical College Hospital, Bangladesh, from July 2013 to June 2014. Study population was pregnant women of third trimester with preeclampsia (case group) and normal blood pressure (control group). The maternal serum C-reactive protein (CRP) levels were measured by immune turbidometric assay between 32 weeks to term. The women were divided into three groups: mild PE, severe PE (according to ACOG criteria) and normal healthy group. The value of CRP and its correlation with birth weight was compared between groups.

Results: One hundred and fifty (150) pregnant women were analyzed. Among them 50 were case and 100 control. There was no difference between age of the patients of both groups. The mean systolic blood pressure was 148.40 ± 12.35 mm Hg in case and 122.15 ± 6.44 mmHg in control group. The mean diastolic blood pressure was 100.00 ± 9.74 mmHg in case and 74.05 ± 5.97 mmHg for the control. The systolic and diastolic blood pressure was significantly higher in preeclamptic group (P < 0.000). C-reactive protein was 10.28 ± 7.25 mg/ mL in mild PE and 10.94 ± 6.32 mg/mL in severe PE and 3.45 ± 1.71 mg/mL in normotensive group, which was significantly higher in case than control group (P = 0.000). Preeclamptic women delivered at a significantly shorter gestational age than normal pregnant women. Mean gestational age during delivery for the case group and control group was 39.02 ± 1.6 and 39.58± 0.8 weeks respectively. Mean birth weight in PE (2.52 ± 0.42 kg) was significantly lower than normal pregnancies (2.88 ± 0.29 kg). The sensitivity and specificity of CRP were 68% and 98% respectively. Multiple regression analysis showed that there is a strong association between CRP levels and PE and birth weight.

Conclusion: This small study showed that in case group CRP was raised in 68% cases which is much higher in comparison to healthy control group where CRP was high only in 2% cases. High CRP has association with low birth weight. Therefore, CRP may be used as cost effective investigation to identify the risk of preeclampsia and its effect on fetal birth weight.

THYROID STATUS AND TREATMENT RESPONSE OF HYPOTHYROID INFERTILE WOMEN IN TERTIARY CARE CENTER OF BANGLADESH

Deeba F, Fatima P, Banu J, Ishrat S, Begum N, Anwary SA

Bangladesh J Obstet Gynaecol, 2016; 31(2): 86-89

Objective(s): Aim of this study was to find out the prevalence of hypothyroidism in infertile women as well as to assess their response to treatment.

Materials and methods: This descriptive study was conducted in Infertility unit of Department of Obstetrics and Gynecology, Bangabandhu Sheikh Mujib Medical University, Bangladesh, a tertiary care centre from January 2014 to December 2014. Four hundred women who visited infertility clinic of the department for fertility treatment were the target population for this study.

Routine investigations such as CBC, Blood sugar 2 hours postparandial or GTT, TSH and Prolactin was done. Subclinical hypothyroidism was diagnosed when there was increased TSH and normal FT4.

Hypothyroid patients were given Levothyroxin depending upon TSH levels and continued until end of the study. Patients were followed up for six months even if pregnancy was attained.

Results: Out of 400 women 55% were primary and 45% were secondary subfertility. Mean duration of infertility was 4.5 ± 1.2 years. Ninety two (23%) patients were hypothyroid. Among them 66 (16.5%) were subclinical hypothyroid and 26 (6.5%) were frank hypothyroid. The mean TSH levels were 7.34 ± 2.13 μIU/ml, and the mean PRL levels were 52.46 ± 11.17 ng/ml. Out of 92 infertile women diagnosed as hypothyroidism 75 (81.52%) women conceived after treatment with drugs for hypothyroidism (dose depending upon severity of hypothyroidism, i.e. TSH levels). More than 90% women had regular ovulation for consecutive three cycles after initiation of treatment.

Conclusion: The normal TSH levels are the pre-requisite for fertilization. The decision to initiate thyroid replacement therapy in both clinical and subclinical hypothyroidism at early stage is justified in infertile women.

(MH Samorita Med Coll J 2019; 2(1): 42)

CME Presentations
(July – December 2018)

No.	Date	Department	Presenter	Topic
1.	07-07-2018	Gynaecology & Obstetrics	Dr. Faria Rashid Asst. Professor	A Case report on "Pregnancy with acute pancreatitis
2.	22-07-2018	Community Medicine	Dr. Anindita Aich Lecturer	From millennium Development goal (MDG) to sustainable development goad (SDD) – Bangladesh Health Perspective
3.	04-08-2018	Cardiology	Dr. Umme Salma Khan Associate Professor	Coronary artery disease in practice
4.	27-08-2018	Dermatology	Dr. Md. Mujibur Rahman Associate Professor	Chicken pox and pemphigus vulgaris
5.	09-09-2018	Ophthalmology	Dr. Ummay Kawsar Assistant Professor	Thyroid Associated Ophthalmopathy
6.	23.-09-2018	Paediatrics	Dr. Israt Jahan Chowdhury Registrar	A Child with fever and rash – Dengue fever
7.	02-10-2018	Otolaryngology & Head-Neck Surgery	Dr. Khwaja Sawda Tabassum Registrar	Role of endoscope in diagnosis and treatment of sinunasal diseases
8.	21.10.2018	Pharmacology	Dr. Israt Jahan Assistant Professor	MBBS Curriculum in Bangladesh – Past, Present & Future
9.	01-11-2018	Nephrology	Dr. Muhammad Ehsan Jalil Assistant Professor	Anaemia in chronic kidney disease
10.	18-11-2018	Conservative dentistry and endodontics	Dr. Kamrun Nahar Shanta Assistant Professor	Dental caries and prevention
11.	06-12-2018	Critical care medicine	Dr. Syed Tariq Reza Associate Professor	ABG interpretation
12.	15-12-2018	Medicine	Dr. Mir Md. Mostafizur Rahman Registrar	Wilson's Disease

CORRIGENDUM

Wrong Information: (bienial publication) and 'the subject'
Place: Volume 1, Both Issue 1 and 2; Page No. (i) in first sentence of **Guide to Authors**.
Correct Information to replace the (bienial publication) by (twice in a year) and 'the subject' by 'different subjects'

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