Bangladesh Medical & Dental Council (BM&DC) Recognized Journal

Green Life Medical College Journal

Volume 4 Number 1 January 2019

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Official Journal of
Green Life Medical College

Website: greenlife.edu.bd

GREEN LIFE MEDICAL COLLEGE JOURNAL
Vol. 4, No. 1, January 2019

Journal Committee

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ABOUT THE JOURNAL

Full Name of the Journal	:	Green Life Medical College Journal
Short Name	:	GMCJ
Nature of Publication	:	Bi-annual
Published From	:	Green Life Medical College
Accreditation	:	Recognized by Bangladesh Medical & Dental Council (BM&DC)
Address	:	31 and 32, Bir Uttam K.M. Shafiullah Sarak, Dhanmondi, Dhaka-1205 Phone: 9612345-50 Ext. 1251

AIMS & SCOPE:

The Green Life Medical College Journal is an english language scientific papers dealing with clinical medicine, basic sciences, epidemiology, diagnostic, therapeutics, public helath and healthcare in relation to concerned specialities. It is an official journal of Green Life Medical College and is published bi-annually.

This Joural is recognized by Bangladesh Medical & Dental Council (BM&DC).

The Green Life Medical College Journal of Bangladesh intends to publish the highest quality material on all aspects of medical science. It includes articles related to original research findings, technical evaluations and reviews. In addition, it provides readers opinion regarding the articles published in the journal.

INSTRUCTION TO AUTHORS:

Papers:

The Green Life Medical College Journal (published bi-annually) accepts contributions from all branches of medical science which include original articles, review articles, case reports, and letter to the Editor.

The articles submitted are accepted on the condition that they must not have been published in whole or in part in any other journal and are subject to editorial revision. The editor preserves the right to make literary or other alterations which do not affect the substance of the contribution. It is a condition of acceptance that the copyright becomes vested in the journal and permission to republish must be obtained from the publisher. Authors must conform to the uniform requirements for manuscripts submitted to biomedical journals (JAMA 1997; 277: 927-34).

Legal considerations:

Authors should avoid the use of names, initials and hospital numbers which may lead to recognition of a patient. A table or illustration that has been published elsewhere should be accompanied by a statement that permission for reproduction has been obtained from the author(s) or publisher(s).

Preparation of manuscript:

Each manuscript should indicate the title of the paper, and the name(s) and full address(es) of the author(s). Contributors should retain a copy in order to check proofs and in case of loss. Two hard copies of each manuscript (double-spaced) should be submitted. If a manuscript is accepted for publication in the GMCJ, the editor responsible for it and may request a soft copy (a CD or via internet) for the revision. Each paper will be reviewed for possible publication. The Editor may wish to see the raw data (electronic form) if necessary.

In preparing the manuscript, use double spacing throughout, including title, abstract, text, acknowledgement, references, table and legends for illustrations and font type and size ‘Times New Roman 12’. Begin each of the following sections on a separate paper. Number pages consecutively.

The standard layout of a manuscript:

- Title page
- Abstract, including Keywords
- Introduction
- Methods
- Results
- Discussion
- Acknowledgements
- Funding
- List of references
- Tables & Figures
- Illustrations

The pages should be numbered in the bottom right-hand corner and the title page being page one, etc. Start each section on a separate page.

Title page:

A separate page which includes the title of the paper. Titles should be as short and concise as possible (containing not more than 50 characters). Titles should provide a reasonable indication of the contents of the paper. This is important as some search engines use the title for searches. Titles in the form of a question, such as 'Is drinking frequent coffee a cause of pancreatic carcinoma?" may be acceptable.

The title page should include the name(s) and address(es) of all author(s). Details of the authors' qualifications and post (e.g., professor, consultant) are also required. An author's present address, if it differs from that at which the work was carried out, or special instructions concerning the address for correspondence, should be given as a footnote on the title page and referenced at the appropriate place in the author list by superscript numbers ($^{1, 2, 3}$ etc.) If the address to which proofs should be sent is not that of the first author, clear instructions should be given in a covering note, not on the title page.

Abstract:

The 'Abstract' will be printed at the beginning of the paper. It should be on a separate sheet, in structured format (Introduction/Background; Methods; Results; and Conclusions) for all Clinical Investigations and Laboratory Investigations. For Reviews and Case Reports, the abstract should not be structured. The Abstract should give a succinct account of the study or contents within 350 words. The results section should contain data. It is important that the results and conclusion given in the 'Abstract' are the same as in the whole article. References are not included in this section.

Keywords:

Three to six keywords should be included on the summary page under the heading Keywords. They should appear in alphabetical order and must be written in United Kingdom English spelling.

Introduction:

The recommended structures for this section are:

- Background to the study/Introduction
- What is known/unknown about it
- What research question / hypothesis you are interested in
- What objective(s) you are going to address

The introduction to a paper should not require more than about 300 words and have a maximum of 1.5 pages double-spaced. The introduction should give a concise account of the background of the problem and the object of the investigation. It should state what is known of the problem to be studied at the time the study was started. Previous work should be quoted here but only if it has direct bearing on the present problem. The final paragraph should clearly state the primary and, if applicable, secondary aims of the study.

Methods:

The title of this section should be 'Methods' - neither 'Materials and methods' nor Patients and methods'. The Methods section should give a clear but concise description of the process of the study. Subjects covered in this section should include:

- Ethics approval/license
- Patient/population
- Inclusion/exclusion criteria
- Conduct of the study
- Data handling
- Statistics
- Cognitive Task Analysis (CTA)

Ethical clearance:

Regardless of the country of origin, all clinical investigators describing human research must abide by the Ethical Principles for Medical Research Involving Human Subjects outlined in the Declaration of Helsinki, and adopted in October 2000 by the World Medical Association. This document can be found at: http://ohsr.od.nih.gov/guidelines/helsinki.html. Investigators are encouraged to read and follow the Declaration of Helsinki. Clinical studies that do not meet the Declaration of Helsinki criteria will be denied peer review. If any published research is subsequently found to be non-compliant to Declaration of Helsinki, it will be withdrawn or retracted. On the basis of the Declaration of Helsinki, the Green Life Medical Journal requires that all manuscripts reporting clinical research state in the first paragraph of the 'Methods' section that:

- The study was approved by the appropriate Ethical Authority or Committee.
- Written informed consent was obtained from all subjects, a legal surrogate, or the parents or legal guardians for minor subjects.

Human subjects should not be identifiable. Do not disclose patients' names, initials, hospital numbers, dates of birth or other protected healthcare information. If photographs of persons are to be used, either take permission from the person concerned or make the picture unidentifiable. Each figure should have a label pasted on its back indicating name of the author at the top of the figure. Keep copies of ethics approval and written informed consents. In unusual circumstances the editors may request blinded copies of these documents to address questions about ethics approval and study conduct.

The methods must be described in sufficient detail to allow the investigation to be interpreted, and repeated if necessary, by the reader. Previously documented standard methods need not be stated in detail, but appropriate reference to the original should be cited. However, any modification of previously published methods should be described and reference given. Where the programme of research is complex such as might occur in a neurological study in animals, it may be preferable to provide a table or figure to illustrate the plan of the experiment, thus avoiding a lengthy explanation. In longitudinal studies (case-control and cohort) exposure and outcome should be defined in measurable terms. Any variables, used in the study, which do not have universal definition should be operationalised (described in such terms so that it lends itself to uniform measurement). Where measurements are made, an indication of the error of the method in the hands of the author should be given. The name of the manufacturer of instruments used for measurement should be given with an appropriate catalogue number or instrument identification (e.g. Keyence VHX-6000 digital microscope). The manufacturer's town and country must be provided, in the case of solutions for laboratory use, the methods of preparation and precise concentration should be stated.

Single case reports:
Single case reports of outstanding interest or clinical relevance, short technical notes and brief investigative studies are welcomed. However, length must not exceed 1500 words including an unstructured abstract of less than 200 words. The number of figures/tables must not be more than 4 and references more than 25.

Animal studies:
In the case of animal studies, it is the responsibility of the author to satisfy the board that no unnecessary suffering has been inflicted on the animal concerned. Therefore, studies that involve the use of animals must clearly indicate that ethical approval was obtained and state the Home Office License number or local equivalent.

Drugs:
When a drug is first mentioned, it should be given by the international non-proprietary name, followed by the chemical formula in parentheses if the structure is not-well known, and, if relevant, by the proprietary name with an initial capital letter. Dose and duration of the drug should be mentioned in sufficient details. If the drug is already in use (licensed by appropriate licensing authority), generic name of the drugs should preferably be used followed by proprietary name in brackets.

Present the result in sequence in the text, table and figures. Do not repeat all the data in the tables and/or figures in the text. Summarize the salient points. Mention the statistics used for statistical analysis as footnote under the tables or figures. Figures should be professionally drawn. Illustration can be photographed (Black and White glossy prints) and numbered.

Discussion:
Comments on the observation of the study and the conclusion derived from it. Do not repeat the data in detail, already given in the results. Give implications of the findings, their strengths and limitations in comparison to other relevant studies. Avoid un-qualified statements and conclusions which are not supported by the data. Avoid claiming priority. New hypothesis or implications of the study may be labeled as recommendations.

Letters are welcome. They should be typed double-spaced on side of the paper in duplicate.

References:
References should be written in Vancouver style, numbered with arabic numerals in the order they appear in the text. The reference list should include all information, except for references with more than six authors, in which case give the first six names followed by et al.

Examples of correct forms of references:
Dorababu M, Prabha T, Priyambada S, Agrawal VK, Aryaa NC, Goel RK. Effect of Azadirachta indica on gastric ulceration and healing of bacopa monnierang in experimental NIDDM rats. *Indian J Exp. Biol 2004; 42: 389-397.*

Chapter in a book:
Hull CJ. Opioid infusions for the management of postoperative pain. In: Smith G, Covino BG, eds. Acute Pain. London: Butterworths. 1985,1 55-79.

All manuscripts for publication should be addressed to the executive editor.

LETTER TO THE EDITOR:
Any reader can provide feedback regarding published articles by writing letter to editor. The reader can also share any opinion in relation to medical science.

Professor M.A. Azhar
Editor-in-chief
Green Life Medical College Journal and
Principal
Green Life Medical College

ABOUT THE COLLEGE

INTRODUCTION

In 2005, about fifty distinguished physicians of the country started a hospital to give specialized care in the private sector. They named it Green Life Hospital and it turned out to be a great success. So in 2009, they decided to establish a medical college which will be a non-government, non-profit, self-financing project and will serve the humanity.

This College came into existence in 2009. The college commences its activities with the enrollment of 51 students in the 1st batch in 2010. Since inception, the college has undergone tremendous development and became a splendid centre for learning and development. At present we are enrolling 110 students each year. Among them, numbers of seats are reserved for overseas students.

We continue to evaluate and improve our programme to ensure the best medical education for the students. Our educational strategy is to create a conducive learning environment and to steer our students to acquire adequate knowledge, skills and temperament to practice medicine and be a competent health care professional group.

Green Life Medical College (GMC) is approved by the Ministry of Health and Family Welfare (MOHFW), Government of Bangladesh and Bangladesh Medical and Dental Council (BMDC) and affiliated to the University of Dhaka.

AIMS AND OBJECTIVES OF THE COLLEGE

Aims:

To create a diverse and vibrant graduate scholars in medical discipline and to create highly competent and committed physicians for the country.

Objectives:

- To provide an appropriate learning environment where medical students can acquire a sound theoretical knowledge and practical skills with empathetic attitude to the people.
- To carry out research in medical sciences to scale up the standard of medical education in the country.

LOCATION

The campus is located at 31 and 32, Bir Uttom K. M. Shafiullah Sarak (Green Road), Dhanmondi, Dhaka. The location is at the heart of the mega city Dhaka and is facilitated with very good communication networks.

The Medical College and the Hospital complexes have been raised in a multistoried fully air-conditioned building with an arrangement of approximately 500 patients. The building is equipped with state-of-the-art infrastructure, excellent with an out-patient department and adequate in-patient facilities.

EDITORIAL

Addition of Elective Programs in Undergraduate Medical Education Curriculum, Bangladesh

In a standard medical education program, all students pass through a set of prescribed course with few if any, opportunities to study a subject in more depth or to study a subject of their own choosing which has not been covered in the program. In recent years there has been a significant increase in curricular flexibility where electives are incorporated [1].

Elective program in a curriculum give students the opportunity to select subject or projects of their own choice.

The content and settings of the placement are largely decide by the student undertaking it and based on their field of interest [2].

Elective programs helps the students' to get an experiences in specialist field and place the students is considering working in future.

The electives gives the opportunity to the students to supplement required learning experiences and allowing them to gain exposure and deeper their understanding of medical specialties reflecting their career interest.

The introduction of the electives subjects programs in the undergraduate medical education program of Bangladesh will change the mode of the study among the students. During the student's undergraduate study, from they will get an opportunity to select their career in future and will experience in the field of the own interest.

Many Universities in Europe, like University of Amsterdam, Netherland, University College London, UK, and University of Toronto, Canada conducting the elective programs in their medical education program. The elective program for MD students of University of Toronto is an integral part of the Curriculum of 4th year [3]. The minimum length of an electives placement is 4 weeks and maximum length is 8 weeks. In Christian Medical College Vellore, India, invite foreign medical student to their available elective programs. Their electives for the foreign medical students not more than 12 weeks. Also, the Kasturba Medical College, Manipal, Karnataka, India offered elective programs to the undergraduate foreign medical students.

In Bangladesh the program for the electives could be launched as pilot program in the apex medical colleges in both public and private sectors, where efficient faculty members are available, malleable and interested for both local and international students. Such program might help to choose their future filed expertise from the very beginning of their medial educational life. The foreigner students will also, get the opportunity and experience to some prevailing health problems, quite prevalent in the South Asian region or health care delivery systems of the Bangladesh. By introducing the elective program in the undergraduate medial education would get acquainted our student to some extent with the same exposure or experience of students of the developed and other countries of the world.

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Prof. Ashraf Uddin Ahmed

Executive Editor, Green Life Medical College Journal
Head, Department of Community Medicine
Chairperson, Medical Education Unit (MEU) and Medical Skill Centre (MSC)
Green Life Medical College

References:

1. R.M. Harden, Susette Sowden and W.R. Dunn , Some educational strategies in curriculum development : the SPICES model, asme, Medical Education Booklet No.18
2. Mike Broad, going on medical electives: a guide for medical student; .Available from https://www.hospitaldr.co.uk/blogs/guidance/going-on-medical-elective-a-guide-for-students. Retrieved 23 December 2018.
3. Elective in MD program, University of Toronto, Available from: https://md.utoronto.ca/electives, Retrieved 11 November, 2018.

ORIGINAL ARTICLE

Cleft Palate and Cleft Lip: Observations In an Urban Setting

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Abstract

Introduction: Cleft lip and/or palate is a common congenital anomaly in the world as well as in Asia which can be corrected by cheiloplasty, palatoplasty, followed by speech therapy. Children with cleft lip with or without cleft palate or a cleft palate alone often have problem with feeding, speech delivery, ear infection, respiratory tract infection. This study was done in an urban setting of Bangladesh to describe the epidemic characteristics and categorized respondents according to the types, position and location of cleft lip/palate, types of surgery advised and suggestion of speech therapy after surgery.

Methods: A cross sectional type of descriptive study was done from 2009 to 2013 with 497 patient of diagnosed cleft lip or palate.

Results: One third (32.2%) of the patient were in the age group of one to five years with male prevalence of 60.6%. Among 497 respondents, majority (64.2%) came with cleft lip, 4% had cleft palate alone and 31.8% of the respondents had both congenital anomalies. Majority of the patients (80.3%) had complete type. Eighty-six percent of the patients were affected by unilateral cleft palate, majority (62.6%) of them were left sided. Only 13.6% of the patient had bilateral type of varieties. About half of the (50.3%) respondents had incomplete pattern of cleft lip, 44% had complete pattern and 5.7% had repaired pattern. For treatment purpose 92.4% of the respondents under went cheiloplasty, 6.6% palatoplasty, 23.6% were advised further surgery to correct cleft palate. 29% of the respondents were advised to take speech therapy.

Conclusion: Proper and meticulous documentation of birth registration along with congenital anomalies will help to evaluate the true values of incidence and prevalence of such inborn defects.

Keywords: Cleft lip, Cleft palate, Orofacial clefts, Urban children

Journal of Green Life Med. Col. 2019; 4(1): 3-7

Introduction:

Cleft lip and/or palate are a congenital abnormality that is seen frequently around the world. A cleft lip happens if the tissue that makes up the lip does not join completely before birth. This results in an opening in the upper lip. The opening in the lip can be a small slit or it can be a large opening that goes through the lip into the nose. A cleft lip can be on one or both sides of the lip or in the middle of the lip, which occurs very rarely. Children with a cleft lip also can have a cleft palate.

A cleft palate happens if the tissue that makes up the roof of the mouth does not join together completely during pregnancy. For some babies, both the front and back parts of the palate are open. For other babies, only part of the palate is open.

Cleft of the lip (CL), palate (CP), or both is one of the most common congenital abnormalities and has a birth prevalence rate ranging from 1/1000 to 2.69/1000 amongst different parts of the world.[1] Birth prevalence ranges from one in every 500 to 1,000 in the white population and one in every 2,000 births in the African-American population. On average, about 1 in every 500-750 live births result in a cleft.[2] Furthermore, in the U.S., the prevalence for cleft lip with or without cleft palate (CL +/- CP) is 2.2 to 11.7 per

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Received: 23 October 2018 **Accepted:** 24 December 2018

10,000 births. CDC recently estimated that, each year in the United States, about 2,650 babies are born with a cleft palate and 4,440 babies are born with a cleft lip with or without a cleft palate.[1] Cleft palate alone (CP) results in a prevalence rate of 5.5 to 6.6 per 10,000 births [3] a higher rate was seen for Asians, specifically in Pakistan, with the prevalence rate being 1.91 per 1,000 live births.[4]

Children with a cleft lip with or without a cleft palate or a cleft palate alone often have problems with feeding, speech and can have recurrent infections in ear and respiratory tract. They also have hearing problems and dental problem.

The causes of orofacial clefts among most infants are unknown. Some children have a cleft lip or cleft palate because of changes in their genes. Cleft lip and cleft palate are thought to be caused by a combination of genes and other factors, such as things the mother comes in contact with in her environment, or what the mother eats or smoke, drinks, or certain medications she uses during pregnancy.[2,4,5,6,7]

Isolated orofacial clefts, or clefts that occur with no other major birth defects, are one of the most common types of birth defects in the United States.[8] Depending on the cleft type, the rate of isolated orofacial clefts can vary from 50% to 80% of all clefts.[9-11]

Etiologic and genetic factors contributing to CL and CP development are unknown though extensive research has been conducted.

Methods:

A Cross sectional type of descriptive observational study was done on 497 patients with diagnosed cleft lip or cleft palate who received operative treatment for Cleft lip & Cleft palate to describe the epidemic characteristics of the patient and to find out a number of variables like types, position and location of cleft lip/palate, types of surgery advised and suggestion of speech therapy after surgery. Data collection was conducted between 2009 to 2013. All patients who attended aged from 5 months to 60 years were included. Data were collected by face to face interview using check list. After collection, each questionnaire was sorted for its consistency and comprehensiveness. Then cleaned data were analyzed through computer based software SPSS 16 and Microsoft Excel 2007. Then various tables were made and analyzed according to the objectives.

Consent was sought from each of the respondents of the study after reading to them the reasons of the study. Confidentiality was maintained throughout by ensuring that no name or number would be used that can identify the respondents.

Results:

A total of 497 patients with congenital cleft lip and cleft palate deformities were included in the study. The duration of the study was from 2009 to 2013 which was done in urban setting of Bangladesh. About one third of the patients (32.2%) were in the age group of 1 to 5 years (Figure-1). The second highest number that is 20.3% was in 0 to 11 months. Only 0.2% were in age group above 50 years. Majority of the patient (60.6%) were male and the rest (39.4%) were female (Table-1).

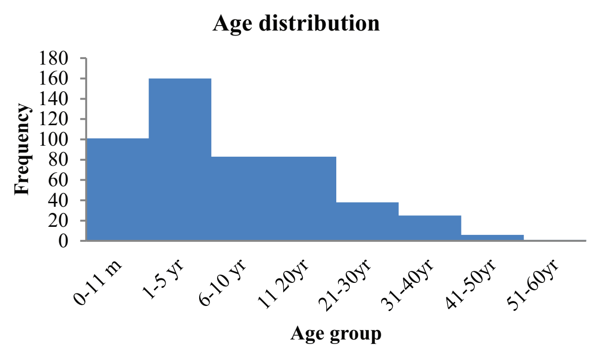

Fig-1: *Distribution of the respondents by age group (n= 497)*

Table-I

Gender Distribution of the respondents (n= 497)

Sex	Frequency	Percentage (%)
Male	301	60.6
Female	196	39.4
Total	497	100.0

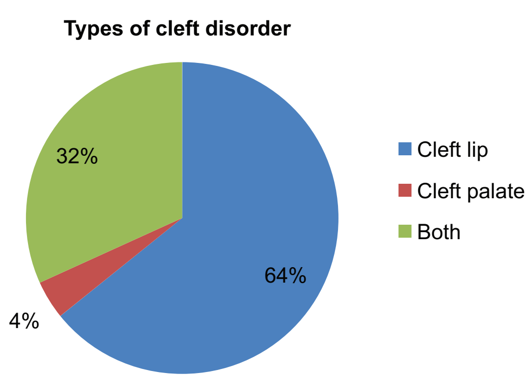

Fig-2: *Distribution of the respondents by type of cleft disorder (n= 497)*

Majority of the respondents (64.2%) came with cleft lip, 31.8% came with both and only 4% had isolated cleft palate.

Table-II

Distribution of the respondent by the characteristics of cleft palate (n= 178)

Types of cleft palate	Frequency	Percentage (%)
Complete	147	80.3
Incomplete	26	14.2
Fistula	2	1.1
Other	3	4.4
Total	178	100.0

Among 497 respondents, 178 patients were presented with cleft palate. Majority (80.3%) had complete type of cleft palate; only 14.2% had incomplete and very few had other varieties.

Table-III

Distribution of the respondent according to position of cleft lip (n= 477)

Type of cleft lip	Frequency	Percentage (%)
Unilateral	412	86.4
Bilateral	65	13.6
Total	477	100.0

Among 477 respondents with cleft lip, 86.4% had unilateral cleft palate and only 13.6% had bilateral types.

Table-IV

Distribution of the respondent according to the location of cleft lip (n= 412)

Side of cleft lip	Frequency	Percentage (%)
Right	154	37.4
Left	258	62.6
Total	412	100.0

Among 412 respondents who had presented with unilateral cleft lip, majority (62.6%) had the deformity on the left side and only one third (n= 154) had right sided cleft lip.

Table-V

Distribution of the respondent by pattern of cleft lip (n= 477)

Presentation of cleft lip	Frequency	Percentage (%)
Complete	210	44.0
Incomplete	240	50.3
Repaired	27	5.7
Total	477	100.0

About half of the respondents (50.3%) had incomplete pattern of cleft lip, 44% had complete and only 5.7% had repaired pattern.

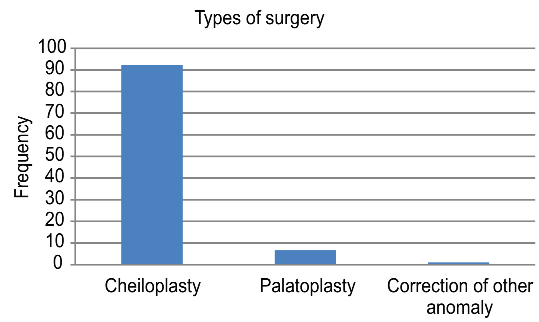

Fig-3: *Distribution of the respondent by type of surgery advised*

Among 497 respondents, cheiloplasty was done for 92.4% and palatoplasty was advised for 6.6% of the respondents.

Table-VI

Distribution of the respondent by further surgery advised (n= 497)

Further surgery advised	Frequency	Percentage (%)
Yes	119	23.9
No	378	76.1
Total	497	100.0

Among 497 patients treated, only 23.9% were advised further surgery.

Table-VII

Distribution of the respondents according to the requirement of speech therapy (n= 497)

Required speech therapy	Frequency	Percentage (%)
Yes	144	29.0
No	353	71.0
Total	497	100.0

Among 497 respondents, only 29% (n = 144) was advised the speech therapy from specialized rehabilitation centre.

Table-VIII

Distribution of the respondent by distance from hospital (n= 497)

Distance from hospital	Frequency	Percentage (%)
0-20 km	250	50.3
21-40 km	140	28.2
41-60 km	51	10.3
61-80 km	18	3.6
81-100 km	14	2.8
>100 km	24	4.8
Total	497	100.0

Among 497 respondents, half of them (50.3%) had their residence within the distance of 0 to 20 km, 28.2% came from 21 to 40 km and only 4.8% came from far away that is above 100 km.

Discussion:

In this present study about one third of the patients (32.2%) were in the age group of 1 to 5 years. The second highest number that is 20.3% was in 0 to 11 months. Only 0.2% was in age group above 50 years. In Brazil, Age between 0 and 4 years old was most prevalent (53%).[14]

In this study majority of the patient (60.6%) were male and the rest (39.4%) were female. This result is coincide with the study conduct in Spain (Male patients 60.97%).[15] In Brazil prevalence is almost same that is male gender is (54%).[14] In Jordan higher prevalence rates were found for boys than girls (55% boys versus 45% girls).[12] In other studies similar results were observed, where the majority of subjects with oral clefts were males. [16-18]

This study shows among 497 respondents majority of them (64.2%) came with Cleft lip, 31.8% came with both and only 4% had Cleft palate. In Jordan the overall prevalence rate for live births with cleft lip, cleft palate, or both was 1.39 per 1000 live births. Thirty percent of the clefts identified affected the lip, 22 percent affected the palate, and 48 percent involved the clefts of the lip and palate.[12] But in California the scenario is quite different , the birth prevalence of nonsyndromic CL/P was 0.77 per 1000 births (CL, 0.29/1000; CP, 0.48/1000), and the prevalence of nonsyndromic CP was 0.31 per 1000 births.[13] In Bolivia the total birth prevalence of CL +/- P was 1.23/1000 live births per year. There were 12 clefts of the lip alone (birth prevalence 0.53/1000 per year), 15 cleft lip and palate (0.66/1000 per year), and one cleft palate only (0.04/1000 per year).[19]

In this study, patients with cleft palate, majority 80.3% had complete type; only 14.2% had incomplete and very few had other varieties. 86.4% had unilateral cleft palate and only 13.6% had bilateral types. Regarding unilateral cleft lip, majority (62.6%) had left sided and only one third had right sided cleft lip. About half of the respondents (50.3%) had incomplete pattern of cleft lip, 44% had complete and only 5.7% had repaired pattern. In a study conduct in Spain shows the unilateral complete cleft lip and palate (54.4%) was most frequently found, followed by the bilateral complete cleft lip and palate (16.3%). Left side (41.46%) were the most affected.[15] Genetic factors are passed to the next generation, thus creating an increased risk for CL/P in offspring. From a clinical point of view, two factors that are sex and severity of the effect in the patient (eg, unilateral vs bilateral), the most important way in evaluating the risk of recurrence for CL/P. The highest risk of recurrence of CL/P is for the subcategory of female patients affected with a bilateral CL/P.[20]

Among 497 respondents of the study, cheiloplasty was done for 92.4% and palatoplasty for 6.6% and only 23.9% was advised further surgery to correct their cleft palate. Twenty nine percent of the respondents were advised to take speech therapy from specialized rehabilitation centre like CRP, Savar.

In this study among 497 respondents half of them had their residence within the distance of 0 to 20 km, 28.2% came from 21 to 40 km and only 4.8% came from far away that is above 100 km. most of them were from the countryside (58.0%)[14] or metropolitan area.[21]

Conclusion:

The researchers who would attempt to investigate cleft lip and palate should be intrigued and challenged by the perplexing questions with which they have faced. Some cases of cleft lip and palate may be purely genetically determined and others were purely environmentally determined.

The present study focused on some of the epidemiological aspects of cleft lip and cleft palate. Precise documentations of birth and death registry along with congenital anomaly registration will help evaluate the true values of incidence of such congenital conditions.

Ethical consideration:

The researchers have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

Funding:

The principal investigator did all the economical expenses in this research.

Conflict of interest:

The authors declare no conflict of interest for this study.

References:

1. McLeod, N.M.H., Arana-Urioste, M.L., & Saeed, N.R. (2004). Birth prevalence of cleft lip and palate in Sucre, Bolivia. Cleft Palate-Craniofacial Journal, 41(2), 195-198.
2. Peterson-Falzone, S.J., Hardin-Jones, M.A., & Karnell, M.P. (2001). Cleft palate speech. (3rd edition). St. Louis: Mosby, Inc.
3. Elahi, M.M., Jackson, I.T., Elahi, O., Khan, A.H., Mubarak, F.M., Tariq, G.B., & Mitra, A. (2004). Epidemiology of cleft lip and cleft palate in Pakistan. Plastic & Reconstructive Surgery, 113(6), 1548-1555.

4. Cheng, L.L. (1990). Asian-American cultural perspectives on birth defects: Focus on cleft palate. Cleft Palate Journal, 27(3), 294-300.

5. Forrester, M.B., & Merz, R.D. (2004). Descriptive epidemiology of oral clefts in a multiethnic population, Hawaii, 1986-2000. Cleft Palate-Craniofacial Journal, 41(6), 622-628.

6. Kim, S., Kim, W.J., Oh, C., & Kim, J.C. (2002). Cleft lip and palate incidence among the live births in the Republic of Korea. Journal of Korean Medical Science, 17(1), 49-52.

7. Kirby, R., Petrini, J., & Alter, C. (2000). Collecting and interpreting birth defects surveillance data by Hispanic ethnicity: A comparative study. Teratology, 61, 21-27.

8. Parker SE, Mai CT, Canfield MA, Rickard R, Wang Y, Meyer RE, Anderson P, Mason CA, Collins JS, Kirby RS, Correa A; for the National Birth Defects Prevention Network. Updated national birth prevalence estimates for selected birth defects in the United States, 2004-2006. Birth Defects Research (Part A): Clinical and Molecular Teratology 2010;88:1008-16.

9. Little J, Cardy A, Munger RG. Tobacco smoking and oral clefts: a meta-analysis. Bull World Health Organ. 2004;82:213-18.

10. Honein MA, Rasmussen SA, Reefhuis J, Romitti P, Lammer EJ, Sun L, Correa A. Maternal smoking, environmental tobacco smoke, and the risk of oral clefts. Epidemiology 2007;18:226–33.

11. Yazdy MM, Autry AR, Honein MA, Frias JL. Use of special education services by children with orofacial clefts. Birth Defects Research (Part A): Clinical and Molecular Teratology 2008;82:147-54.

12. Al Omari, F., & Al-Omari, I.K. (2004). Cleft lip and palate in Jordan: Birth prevalence rate. Cleft Palate-Craniofacial Journal, 41(6), 609-612.

13. Croen, L.A., Shaw, G.M., Wasserman, C.R., & Tolarova, M.M. (1998). Racial and ethnic variations in the prevalence of orofacial clefts in California, 1983-1992. American Journal of Medical Genetics, 79, 42-47.

14. Andrea Luiza[1] Diego Noronha de Góis[1] et. al. A Descriptive Epidemiology Study of Oral Cleft in Sergipe, Brazil,Int. Arch. Otorhinolaryngol. vol.17 no.4 São Paulo 2013.

15. Rosa P, Yanez M et.al. A descriptive epidemiologic study of cleft lip and palate in Spain. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology.Volume 114, Issue 5, Supplement, November 2012, Pages S1-S4.

16. .Ajike SO, Adebola RA, Efunkoya A, Adeoye J, Akitoye O, Veror N. Epidemiology of adult cleft patients in North-western Nigeria: our experience. Ann Afr Med 2013;12:11-15.

17. Martelli DR, Machado RA, Swerts MS, Rodrigues LA, Aquino SN, Martelli Júnior H. Non syndromic cleft lip and palate: relationship between sex and clinical extension. Braz J Otorhinolaryngol 2012;78:116-120.

18. Costa CH, Diniz LV, Lacerda RH, Forte FD, Sampaio FC. Prevalence of dental anomalies in patients with cleft lip and palate, Paraiba, Brazil: clinic and radiographic study. Acta Odontol Latinoam 2012;25:181-185.

19. McLeod NM[1], Urioste ML, Saeed NR. Birth prevalence of cleft lip and palate in Sucre, Bolivia. Cleft palate craniofac.j, 2004 Mar, 41(2); 195-8.

20. Marie M Tolarova, MD, PhD, DSc; Chief Editor: Ravindhra G Elluru, Pediatric Cleft Lip and Palate, Feb 01, 2018, Medscape.

21. Baptista EVP. Malformações congênitas associadas à fissura labial e/ou palatal em pacientes atendidos em um serviço de referência para tratamento de defeitos da face: um estudo de série de casos [dissertation]. Recife, Brazil: Instituto Materno Infantil Prof. Fernando Figueira; 2007:67

ORIGINAL ARTICLE

Estimation of the Stature From The Selected Cranial Measurements – An Anthropometric Study On Adult Bangladeshi Manipuri Females

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Abstract

Introduction: ***Cranial anthropometry involves measurements of parameters on the skull. It can be used for racial identification, physical anthropologists, forensic scientists, genetic counselors, beauticians as well as plastic and reconstructive surgeon. On the other hand, stature means a person's natural height. For personal identification, the stature is one of the important criteria which have been found to show definite and proportional biological relationships with different parts of the human body like head, face, trunk and extremities. These measurements and relationships are also considered as characteristics useful data for differentiating between various ethnic groups of the world. There is no formally produced data on the cranial measurements or stature of the Manipuris of Bangladesh. This study was anticipated to develop a baseline quantitative data on the linear selected cranial measurements and the stature adult Bangladeshi Manipuri females. This study was undertaken to determine whether there is any significant correlation between the stature of an individual and the selected cranial measurements of adult Bangladeshi Manipuri females.***

Methods: The study was observational, cross-sectional with descriptive and some analytical components. The study was carried out in the Department of Anatomy, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka and the period of study was March, 2015 to February 2016. A total of 130 adult Bangladeshi Manipuri females were selected using the convenience sampling technique from the Madhavpur village of the Kamalganj Police Station in Maulavibazar district of Sylhet, Bangladesh. Cranial dimension such as the maximum cranial length, the maximum cranial breadth, the head circumference and the stature were measured directly from the subjects by using a measuring tape, spreading caliper, steel plate and steel tape. Multiplication factors were estimated for estimating stature from the cranial measurements. The agreement between the corresponding 'measured' stature and the 'estimated' stature using the Bland-Altman method.

Results: The maximum cranial breadth and head circumference showed significant positive correlations with the stature but the maximum cranial length showed positive correlations with the stature did not reach statistically significant level. No significant difference was found between the 'measured' stature and the 'estimated' stature for the maximum cranial length, the maximum cranial breadth and the head circumference. So, the measured' stature and the 'estimated' stature in adult Bangladeshi Manipuri females agreed with each other.

Conclusions: The results of the present study can provide the basic framework for formulating the interrelationships between the selected cranial measurements and the stature in adult Bangladeshi Manipuri female population. However, using larger samples with more sophisticated measurement techniques and with more specified inclusion and exclusion criteria are recommended for standardization of these anthropometric measurements.

Keywords: Anthropometry, Stature, correlation, Maximum cranial length, Maximum cranial breadth, Head circumference, Multiplication factor.

Journal of Green Life Med. Col. 2019; 4(1): 8-13

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Received: 18 November 2018 **Accepted:** 24 December 2018

Introduction:

The term 'anthropometry' is a Greek word ('anthropos', meaning 'human' and 'metron', meaning 'measurement'). So, 'Anthropometry' means the measurement of the human body and its individual parts. Cranial anthropometry means measurement of the head of living subjects. Traditional anthropometry uses simple instruments to take measurements.

However, human body dimensions are affected by ecological, biological, geographical, racial, gender, age related and nutritional factors but bodily measurements are the basis of the anthropological research.[1] Anthropometry is a series of systematic measurement techniques that expresses quantitative representation of the human individual for the purpose of understanding human physical variations.[2] Stature has a proportional biological relationship with each other and every part of the human body, i.e. head, face, trunk and extremities and this relationship also helps a forensic scientist to calculate stature from dismembered and mutilated body parts in forensic examinations.[3] Mansur et al. also stated that "Although a number of long bones are used for this purpose but cranial dimensions are more reliable and precise mean of predicting the stature".[4] Therefore, the present study was conducted with the intension to investigate whether there is any possible significant correlation between the stature and the cranial dimension in human body.

Methods:

An observational, cross-sectional with descriptive and some analytical components were present in this study which was carried out on the adult Bangladeshi Manipuri females lived in Madhavpur village of the Kamalganj Police Station in Maulavibazar district of Sylhet, Bangladesh. The study was conducted among 130 adult Bangladeshi Manipuri females, 25 to 45 years of age. This sample size was calculated following Liao (2009).[5]

Sample size when all individuals but one pair agree with each other (k=1)

		Tolerance probability				
		80%	85%	90%	95%	99%
Discordance Rate	0.01	299	337	388	473	662
	0.05	59	67	77	93	130
	0.10	29	33	38	46	64
	0.15	19	22	25	30	42
	0.20	14	16	18	22	31

The study population does not have any known history of interethnic mixing three generations or that of any acquired or genetic craniofacial anomalies. Ethical clearance was taken from the Institutional Review Board (IRB) of BSMMU and written informed consent was taken from all participants.

The researcher requested to each participants with a proper and respectful approach to give the measurements. The participants were told to sit on the chair in a relaxed mood with head in anatomical position.[6] There were 2 milimetre errors in the spreading caliper. For getting actual measurement 2 milimetre was discarded from the measured value. All the measurements were taken twice to control for measurement error and were recorded on the data sheet with the help of a volunteer.

The final value that was used for the study was the average of the two obtained values. A third reading was taken if the initial two measurements showed some discrepancy and the two closer reading then be used.[7]

The stature was taken by using steel plate and steel tape after removing any hair ornaments, jewelry, buns, or braids from the top of the head. The maximum cranial length and the maximum cranial breadth were measured by using spreading caliper. The head circumference was measured by using flexible measuring tape.

Mean and standard deviation were calculated for each measurement. Correlation was tasted between the selected cranial measurements and the stature. Multiplication factor was calculated for estimating stature from the selected cranial measurements. The effectiveness of the use of the multiplication factor in these estimations was tested whether there was a significant difference or not.

Calculation of multiplication factors:

Each multiplication factor is the ratio of the stature to the respective physical measurements or respective cranial measurements. A mean multiplication factor was then calculated for each measurement. These mean multiplication factor was used for estimating the stature from that measurements.[8]

The multiplication factor (MF) of each cranial measurement was calculated using the following formula:

$$\text{MF} = \frac{\text{Stature}}{\text{Respective cranial measurement}}$$

(maximum cranial length or maximum cranial breadth or head circumference)

Results:

Table-I showed that the mean values of the stature, the maximum cranial length, the maximum cranial breadth and the head circumference. The mean (±SD) of the stature, the maximum cranial length, the maximum cranial breadth and the head circumference were 149.95 (±6.07), 17.65 (±0.74), 14.31 (±0.99) and 54.32 (±1.51) respectively (Table-I).

Table-I

Selected linear craniofacial measurements and the stature (N=130)

Measurement	Value (cm)	
	Range	Mean (±SD)
Stature	130.60 – 163.20	149.95 (±6.07)
Maximum cranial length	16.00 – 19.00	17.65 (±0.74)
Maximum cranial breadth	11.00 – 16.00	14.31 (±0.99)
Morphological face height	9.58 – 11.82	10.72 (±0.45)
Head circumference	51.00 – 58.20	54.32 (±1.51)

Here, the maximum cranial breadth and the head circumference showed significant positive correlations with the stature but the maximum cranial length showed positive correlation with the stature but did not reach statistically significant level (Table-II & Fig. 1, 2, and 3).

Table-II

Correlation coefficients of selected cranial measurements with the stature in adult Bangladeshi Manipur females (N= 130)

Measurement	Correlation coefficient (r)	Significance (p) of correlation with the stature	Mean multiplication factor MF* (± SD) to estimate the stature
Maximum cranial length	0.092	0.300 (NS)	8.512 (± 0.489)
Maximum cranial breadth	0.221	0.012(S)	10.529 (±0.815)
Head circumference	0.265	0.002 (S)	2.762 (±0.117)

$p \leq 0.05$ was considered as significant

S= significant

NS= Non-significant

*The mean multiplication factor for each craniofacial variable was calculated for estimating the stature from that variable. It was determined by dividing the stature of each individual subject by the individual value for the variable in question and then calculating the mean of the 130 individual multiplication factors.

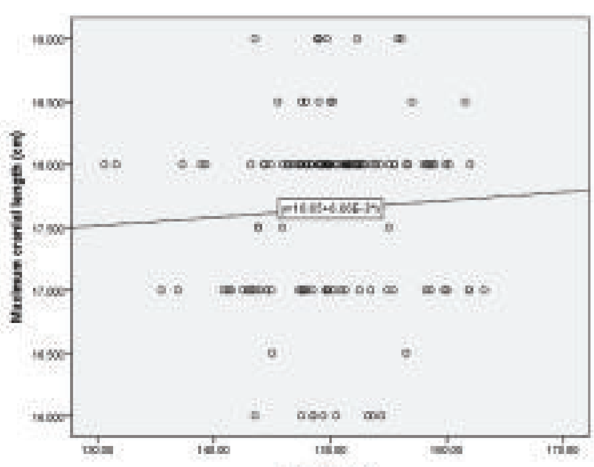

Fig.-1: *Regression analysis, showing non-significant positive correlation (r = +0.092, p = 0.300) between the maximum cranial length and the stature (N= 130).*

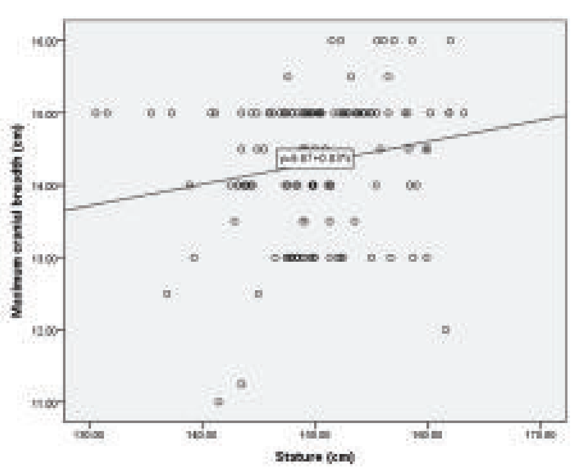

Fig.- 2: *Regression analysis, showing significant positive correlation (r = +0.221, p = 0.012) between the maximum cranial breadth and the stature (N= 130).*

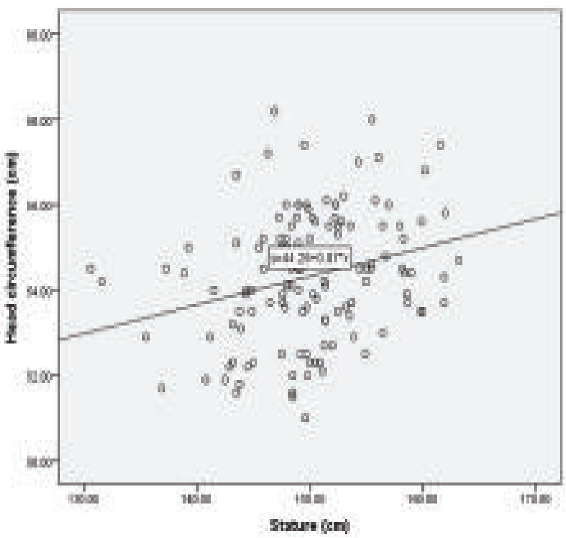

Fig. -3: *Regression analysis, showing significant positive correlation (r = +0.265, p = 0.002) between the head circumference and the stature (N= 130).*

No significant difference was found between the 'measured' stature and the 'estimated' stature for the maximum cranial length, the maximum cranial breadth and the head circumference (Fig. 4, 5 and 6).

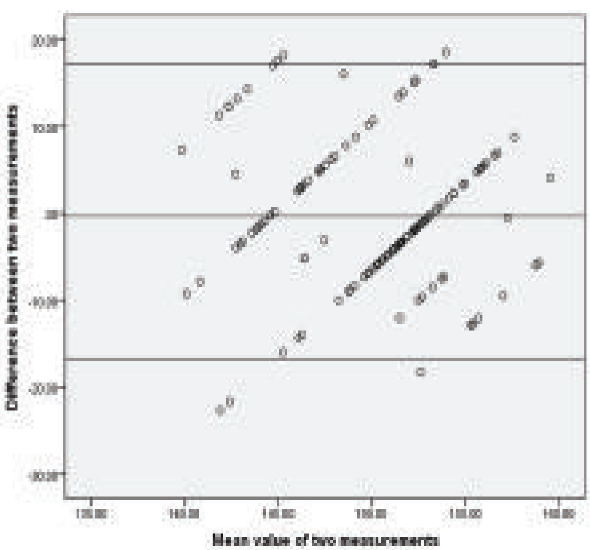

Fig. 4 *Bland-Altman plot of measured stature and estimated stature for cranial length showing upper and lower lines that indicate the limits of agreement, and the middle line that indicates the mean values of the two measurements.*

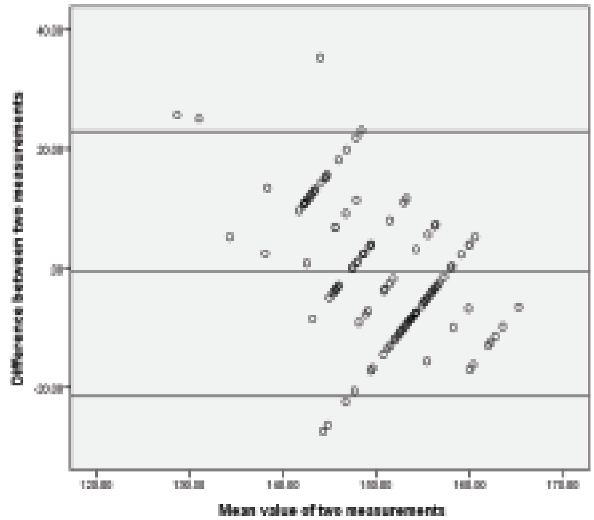

Fig. 5 *Bland-Altman plot of measured stature and estimated stature for cranial breadth showing upper and lower lines that indicate the limits of agreement, and the middle line that indicates the mean values of two the measurements.*

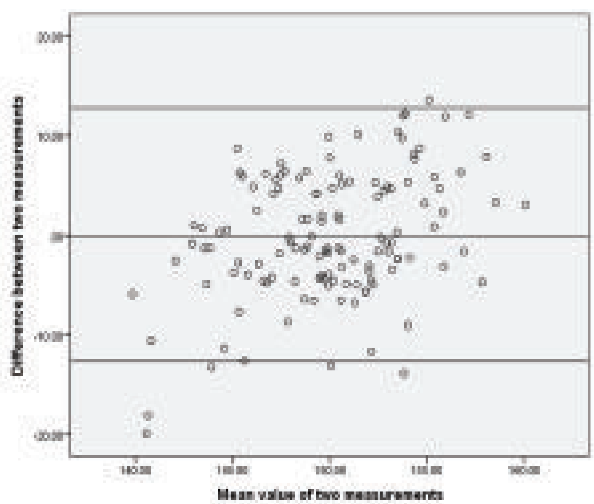

Fig. 6 *Bland-Altman plot of measured stature and estimated stature for head circumference showing upper and lower lines that indicate the limits of agreement, and the middle line that indicates the mean values of the two measurements.*

Discussion:

The present anthropometric study was carried out among adult Bangladeshi Manipuri females with a view to measuring the stature, the maximum cranial length, the maximum cranial breadth and the head circumference. It also looked for any correlation between the stature and the maximum cranial length, the maximum cranial breadth and the head circumference. Estimation of stature was made from the maximum cranial length, the maximum cranial breadth and the head circumference using multiplication factors and effectiveness of the estimation was tested by Bland-Altman method. Manipuri tribe belongs to the race, some similarities are expected to exist with other ethnic groups of the Mongoloid race and some with other races.

In general, the present study data were satisfactorily in concurrence with previous reports. For example, the mean stature of the adult Bangladeshi Manipuri females was almost similar to those of the adult Santal females, Bangladesh[9] and Santal population, Bankura District, West Bengal, India.[10] The mean maximum cranial length was similar to those of the Mongoloid adult Christian Garo females, Dhaka, Bangladesh[11] and Taiwanese people, Taiwan.[12] They also showed a similar mean value to those of the adult Santal females, Bangladesh[9], Gurung community, Nepal[13] and adult male and female ethnic population (Indigenous, Indo-Nepalese and Tibeto-Nepalese), Sunsari and Morang Districts, Nepal.[14] The mean maximum cranial breadth was similar to those of the Mongoloid adult Christian Garo females, Dhaka, Bangladesh11, the Austroasiatic Santal population Bankura District, West Bengal, India[10], the Caucasoid Latvian residents, Latvia[15], the Negroid Onge males and females tribe, Andaman and Nicobar Island, India[16] and adult male and female ethnic population (Indigenous, Indo-Nepalese and Tibeto-Nepalese), Sunsari and Morang Districts, Nepal.[14] The head circumference had a mean value similar to those of the dental students of a University, Tehran, Iran[17], the Negroid adult Omoku, Ogba/Egbema/ Ndoni, River State, Nigeria.[18] They also showed a similar mean value to those of the adult male and female students, a medical school, Kathmandu, Dhulikhel, Nepal.[4]

When the age groups progress from the younger age group to the older age group, usually there is a gradual increase of the mean values of all the parameters.[19] According to Shah, Koirala and Khanal (2014. P. 7) racial factor, gender, geographical and regional variations influence the craniofacial anthropometry especially stature and head region.[20]

Significant correlations between some of the measurements can be utilised in the determination of proper multiplication factors to be useful in estimating one measurement from another. This should encourage others in taking up further research in this field. This measurements and multiplication factors would be useful in the field of physical anthropometry, archeology, genetic counselors, forensic scientists, plastic surgery, oral surgery, ophthalmology, otolaryngology as well as to the beautifications.

Conclusion:

The results of the present study can be useful for the anatomists and anthropologists to serve as a basic framework. Multiplication factors were calculated which will be useful for the estimation of stature from the selected cranial measurements.

References:

1. Shah S, Koirala S. Role of Craniofacial Anthropometry in Medical Science. International Invention Journal of Medicine and Medical Sciences 2015; 2 (4): 44-48.
2. Al-jassim NH, Fathallah ZF, Abdullah NM. Anthropometric measurements of human face in Basrah. Basrah Journal of Surgery 2014; 29-40.
3. Mansur DI, Haque MK, Sharma K, Mehta DK, Shakya R. Use of Head Circumference as a Predictor of Height of Individual. Kathmandu Univ Med J 2014; 12 (2): 89-92.
4. Ahmmed MF, Singh L. State of the Rural Manipuris in Bangladesh, Ethnic Community Development Organization (ECDO) press 2006, Sylhet, Bangladesh.
5. Liao JJZ. Sample size calculation for an agreement study. Pharmacutical Statistics 2009; 9 (2): 125-132.
6. Anibor E, Etetafia MO, Eboh DEO, Akpobasaha O. Anthropometric study of the nasal parameters of the Isokos in Delta State of Nigeria. Annals of Biological Research 2011; 2 (60: 408-413.
7. Hansi B, Ashish B. An Estimation of Correlation between the Head length and the Stature of the Children aged between 6-10 Years. Research Journal of Forensic Sciences 2013; 1(2): 1-5.
8. Waghmare V, Gaikwad R, Herekar N. Estimation Of The Stature From The Anthropometric Measurement Of Hand Length. The Internet Journal of Biological Anthropology 2012; 4(2): 2-4.
9. Talukder M. Craniofacial Measurements and Their Relationships with Each Other and with the stature in Adult Bangladeshi Santal Females, masters thesis, Bangabandhu Sheikh Mujib Medical University, Dhaka, 2014.
10. Ghosh S, Malik SL. Sex differences in body size and shape among Santhals of West Bengal. Anthropologist 2007; 9 (2): 143-149.
11. Ferdousi A. Comparative Craniofacial Anthropometry of Adult Christian Bangladeshi Garo Males and Females, masters thesis, Bangabandhu Sheikh Mujib Medical University, Dhaka 2011.
12. Liu BS. Incorporating anthropometry into design of ear-related products. Applied Ergonomics 2007; 499 (4): 115-121.
13. Lobo SW, Chandrashekhar TS, Kumar S. Cephalic index of Gurung community of Nepal - an anthropometric study.

Kathmandu University Medical Journal 2005; 3 (3): 263-265.

14. Shah S, Koirala S, Jha CB. Effect of ethnicity on head form anthropometry of 17-26 year old normal population in Eastern Nepal. Eur. J. Anat. 2014; 18 (3): 135-139.

15. Nagle E, Teibe U, Kapoka D. Craniofacial anthropometry in a group of healthy Latvian residents. Acta medica Lituanica 2005; 12 (1): 47–53.

16. Pandey AK. Cephalo-facial Variation Among Onges. Anthropologist 2006; 8 (4): 245-249.

17. Amini F, Mashayekhi Z, Rahimi H, Morad G. Craniofacial Morphologic Parameters in a Persian Population: An Anthropometric Study. The Journal of Craniofacial Surgery 2014; 25 (5): 1874-1881.

18. Oladipo GS, Okoh PD, Akande PA, Oyakhire MO. Anthropometric study of some craniofacial parameters: head circumference, nasal height, nasal width and nasal index of adult Omoku indigenes of Nigeria. American Journal of Scientific and Industrial Research 2011; 2 (1): 54-57.

19. Ewunonu EO, Anibeze CIP. Anthropometric study of the Facial Morphology in a South-Eastern Nigerian Population. Human Biology Journalb2013; 2 (4): 314-323.

20. Shah S, Koirala S, Khanal L. Variation in Craniofacial Anthropometry of 17-25 Years Old Adult Population of Nepal. European Journal of Forensic Sciences 2014; 1 (1): 5-8.

ORIGINAL ARTICLE

Pattern of Dyslipidaemia Among Patients With Newly Diagnosed Type 2 Diabetes Mellitus in Bangladeshi Population

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Abstract

Introduction: Dyslipidaemia is one of the major risk factors for developing cardiovascular disease (CVD) in diabetic patients. However, the magnitude and characteristic features of dyslipidaemia among Bangladeshi people with newly diagnosed diabetes is not well studied. We aimed to study the pattern of dyslipidaemia in a cohort of patients with newly diagnosed type 2 diabetes mellitus (T2DM).

Methods: This study was carried out in 101 newly diagnosed T2DM patients attending a specialist Endocrinology outpatient department of a tertiary hospital of Bangladesh. Dyslipidaemia was diagnosed if patients had one or more parameters of lipid profile outside the target values recommended by the AmericanDiabetes Association (ADA).

Results: This study revealed, nearly 96% patients had some form of dyslipidaemia at the time of diagnosis of T2DM. Combined dyslipidemia including high LDL and low HDL was the commonest abnormality affecting 56% of male and 81% of female. Isolated raised LDL-C was relatively less common than combined dyslipidemia affecting 10% of male and 5% of female only. Females had higher prevalence of unfavorable lipid abnormalities including raised LDL-C and low HDL –C than male. However, Isolated raised TG and Low HDL was more prevalent in males than in females. Factors such as degree of hyperglycemia at diagnosis, showed significant effect on prevalence and pattern of dyslipidaemia. BMI and WC showed no significant effect on prevalence and pattern of dyslipidaemia

Conclusions: This study revealed a higher prevalence and a different pattern of dyslipidaemia among newly diagnosed patients with T2DM. Finding of raised LDL with relatively low HDL observed in this study was different to the pattern of diabetic Dyslipidaemia described in Caucasians.

Keywords: Diabetic dyslipidaemia, Type II DM, Cardiovascular disease

Journal of Green Life Med. Col. 2019; 4(1): 14-19

Introduction:

Type II Diabetes Mellitus is a heterogenous condition characterized by hyperglycemia due to abnormalities in both insulin secretion and insulin resistance. Type II diabetic patients are at increased risk of developing microvascular and macrovascular complications like coronary artery disease (CAD), Ischemic stroke etc. The risk is due to a greater burden of atherogenic risk factors among diabetics including dyslipidemia, hypertension, and obesity.[1] Beyond glycemic control, management of hypertension, dyslipidemia can prevent or delay the onset and severity of diabetic complications. Different studies have shown the beneficial effect of treating dyslipidemia in diabetic patients on cardiovascular morbidity and mortality.[2, 3]

Dyslipidemia is elevation of plasma cholesterol, triglycerides or both or low HDL level that contributes to

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Received: 29 October 2018 **Accepted:** 24 December 2018

the development of atherosclerosis. It is traditionally classified by patterns of elevation in lipids and lipoproteins.Dislipidemia is a well-recognized and modifiable risk factor that should be identified early to institute aggressive cardiovascular preventive management.

The American Diabetes Association (ADA) recommends screening for dyslipidemia at the time of diabetes detection and every five years thereafter, or more frequently if needed to achieve goals.[4] In 2016, ADA recommends moderate intensity statin treatment for all patients with diabetes agede"40 years and high dose statin for those with increased cardiovascular risk(e.g. High blood pressure, smoking, albuminuria, LDL cholesterol>100mg/dl and family history of premature CAD.[5] Spectrum of dyslipidemia in diabetic patients can vary; however the typical pattern of dyslipidemia seen in diabetic patients is high TG, elevated LDL cholesterol and low HDL-C.Low HDL-C,high TG and LDL-C are important risk factors for CAD.[6,7] Morbidity and mortality in diabetic patients differ in different ethnic groups. South Asian has a higher risk of developing CAD than Caucasians.[8] Pattern of dyslipidemia could be a contributing factor for developing CAD among South Asians.

Adequate knowledge on the pattern of dyslipidemia will be useful in the proper management of Type II DM.Therefore, this study was designed to determine the pattern of dyslipidemia among newly diagnosed diabetic patients and evaluate its association with risk factors such as hypertension, BMI, Waist circumference and FBG.

Methods:

The study was conducted in the department of Endocrinology, Enam Medical College and Hospital,Savar,Dhaka during the period of January 2016 to June 2016.It was a cross sectional study. The subjects were selected purposively. One hundred and one subjects with newly diagnosed type 2 diabetes aged 20-70 years were included in this study. Exclusion criteria include history of diabetes more than one year, presence of endocrine disorders other than diabetes, pregnant and lactating mother, patients receiving any medication that may alter blood glucose level, patients currently on treatment (pharmacological or non-pharmacological) for dyslipidemia, patients with known history of liver or renal disease, acute infection, and when type 1 diabetes or secondary causes for diabetes were more likely on clinical grounds than T2DM. They were selected from outpatient clinic of endocrinology department at Enam Medical College Hospital on the basis of availability.

Selection of the subjects:

Study subjects were selected who were newly diagnosed diabetic patients according to ADA criteria. Diabetic patients labeled as newly diagnosed referred to those with a known duration of up to one year. Those who had history or clinical features of endocrinopathies,renal disease, liver disease, acute infection ,history of taking medication that may alter blood glucose level, history of taking medication for dyslipidemia were excluded from the study. After primary selection, patients were referred to perform following investigation: SGPT, S.creatinine, fasting lipid profile, CBC, USG of whole abdomen to exclude renal disease, liver disease and acute infection. Study protocol was approved by local Ethical committee and all patients were briefed about the study and informed written consent was obtained. At baseline, demographic data were collected and a detailed physical examination was done. Sitting blood pressure was measured in both arms after at least ten minutes of rest with an appropriate sphygmomanometer.

Dyslipidemia was diagnosed if any component of lipid profile exceeds recommended targets. Those with dyslipidemia were further subdivided into mixed dyslipidaemia (all three parameters outside the recommended ADA targets), combined dyslipidaemia (two parameter outside the recommended target) or isolated dyslipidaemia (only one parameter outside the recommended target. Patients with combined dyslipidaemia were further classified in to different patterns of dyslipidaemia (high LDL and TG, high LDL and low HDL, high TG and low HDL).

Anthropometric measurement:

Standing height and weight was measured using appropriate scales. BMI was calculated using standard formula, BMI=weight (kg)/height (m2).Waist circumference was measured at the plane between anterior superior iliac spine and lower costal margin at the narrowest part of the waist line on the midaxillary line while the patient was standing and at the end of normal expiration. It was measured with a soft non elastic measuring tape.

Blood sample collection:

Subjects were requested to fast at least eight hours and fasting venous blood sample was collected between 7am to 8 am. Serum fasting glucose was measured at the day of sample collection. Serum fasting lipid profile, SGPT, creatinine, CBC, HbA1c were measured within three days of sample collection.

Statistical Analysis:

Statistical analysis was made by using SPSS for windows 16 software package. All data were expressed as mean, SD, median and or percentage as appropriate. Chi-square test was used to test difference between groups. P level‹0.05 was considered significant and 95% confidence interval was calculated. Z-proportion test and one way ANOVA were used to test significant as appropriate.

Results:

Table 1 shows demographic characteristics of the 101 newly diagnosed patients with T2DM in the study. There were more male (58.42%) than female (40.58%), and nearly 40% of the male had younger onset of T2DM (<40 years).Females were significantly older and had lower level of HDL-C than males. Although there was a higher prevalence of central obesity in females (78%) compared with males (17%), the mean difference was not statistically significant.

Mean levels of total cholesterol, HDL-C, LDL-C, and TG were 202.26± 45.90, 36.39±7.8, 128.58± 30.83, and 212.95± 135.28 respectively. Of note, the mean LDL-C of 128.58± 30.83, mg/dL was well above the LDL-C target of 100 mg/dL recommended by ADA.

Combined dyslipidemia including high LDL and low HDL was the commonest abnormality affecting 56% of male and 81% of female followed by high TG and high LDL affecting 54% of male and 71% of female, and High TG and low HDL affecting 45% of male and 64% of female. Isolated raised LDL-C was relatively less common than combined dyslipidemia affecting 10% of male and 5% of female only. Females had higher prevalence of unfavorable lipid abnormalities including raised LDL-C and low HDL –C than male. However, Isolated raised TG and Low HDL was more prevalent in males than in females.

Table II presents the pattern of dyslipidemia in newly diagnosed patients with T2DM according to their gender. Among males, combined dyslipidemia with raised LDL-C and low HDL-C was the commonest pattern seen around 56%, followed by raised LDL-C and TG was 54% and high TG and low HDL-C was around 46%. Mixed dyslipidemia with all three abnormal lipid parameters (high TG and LDL-

Table l

Baseline characteristics of the study sample according to gender

Variables Number and percentages	Male (n=59)		Female (n=42)		*p*
	n	%	n	%	
Age (years)					
<40	23	38.98	13	30.95	-
≥40	36	61.02	29	69.05	-
BMI					
<23	17	28.81	7	16.66	-
≥23	42	71.19	35	83.44	-
Waist circumference (cm)					
Normal	49	83.05	9	21.42	-
High	10	16.95	33	78.58	-
HbA_1C					
<7%	3	5.08	3	7.14	-
≥7%	56	94.92	39	92.86	-
	Mean	SD	Mean	SD	*p*
Age	43.42	10.72	44.17	11.62	0.741
BMI	24.96	4.02	26.66	4.06	0.039*
Waist circumference	83.88	5.24	85.33	5.92	0.197
HbA_1C %	10.75	2.37	9.71	2.30	0.029*

*significant at 95% CI

Table ll

Pattern of dyslipidaemia according to gender

	Male (n=59)		Female (n=42)	
Pattern of dyslipidemia	n	%	n	%
Isolated single parameter dyslipidemia	13		2	
Raised LDL cholesterol	06	10.16	02	4.76
Raised triglyceride	02	3.38	00	00
Low HDL cholesterol	05	8.47	00	00
Combined dyslipidemia	92		91	
High TG and low HDL-C	27	45.76	27	64.28
High TG and high LDL-C	32	54.23	30	71.42
High LDL and low HDL-C	33	55.93	34	80.95
Mixed dyslipidemia(high TG,high LDL-C,and low HDL-C)	22	37.28	26	61.90
Total	127		119	

C and low HDL-C) was also common, 37% in males. Isolated raised LDL-C was less common, around 10%. In females too, isolated raised LDL-C was very rare (4.76%). Among females, most common pattern of dyslipidemia was combined dyslipidemia with raised LDL-C and low HDL-C(81%) followed by high TG and high LDL-C was 71% and High TG and low HDL-C was 64%. Mixed dyslipidemia with all three abnormal lipid parameters (high TG and LDL-C and low HDL-C) was also common, 62% in females.

We analyzed prevalence and pattern of dyslipidemia according to degree of hyperglycemia at the time of diagnosis of T2DM. HbA_1C value less than 7% was considered as lower degree of hyperglycemia whereas values 7% or more was considered as higher degree of hyperglycemia. As shown in table III, there was significant effect of degree of hyperglycemia on the pattern and the prevalence of dyslipidemia.

Table lll

Lipid abnormalities according to degree of hyperglycaemia

Prevalence of	Degree of hyperglycemia		
dyslipidaemia	HbA_1C <7% (%)	HbA_1C ≥7% (%)	***p***
Raised LDL	4.95	75.24	0.001*
Raised TG	4.95	64.35	0.001*
Low HDL	5.94	69.30	0.001*
Total			

LDL-Low density lipoprotein, HDL-High density lipoprotein, TG-triglycerides* significant by Z proportion test.

We also evaluated whether the age at diagnosis of T2DM had any effect on the prevalence and pattern of dyslipidemia. As shown in table 4, raised TG was different between groups with higher mean value observed in patients with younger (<40 years) onset T2DM compared older onset. Other lipid parameters including raised LDL and low HDLwere higher in older individuals.

Table lV

Lipid abnormalities according to age at diagnosis

Age categories		TC			LDL			TG			HDL		
(in years)	n	Mean	SD	*p*	Mean	SD	*p*	Mean	SD	*p*	Mean	SD	*p*
<40	36	202.26	45.91	0.295	128.58	30.83	0.466	212.95	135.28	0.156	36.40	7.81	0.128
40-59	54	216.64	39.87		137.51	0.34		209.07	108.54		39.30	7.89	
≥60	11	209.70	42.60		133.80	39.27		187.64	158.14		35.06	9.92	

LDL-Low density lipoprotein, HDL-High density lipoprotein, TG-triglycerides by One way ANOVA test

Discussion:

In the wake of rising incidence of diabetes and cardiovascular diseases in Bangladesh, this study is a step towards evaluating the prevalence and the pattern of diabetic dyslipidaemia. This study revealed, close to 96% patients had some form of dyslipidaemia at the time of diagnosis of T2DM. The mean levels of total cholesterol and LDL-Cfound in our study were comparable to the mean totalcholesterol levels and LDL-C reported in the United Kingdom Prospective Diabetes Study (UKPDS).[9] However, raised TG observed in our study (211mg/dL in males and 230mg/dL in females) was higher than the mean TG levels of UKPDS(159mg/dLin both genders) . MeanHDL-C levelof 35.8mg/dL in males and 37.11mg/dL in females found in our study were also close to the meanHDL-C levels of 39mg/dl in males and 43mg/dl infemales reported in UKPDS study. The UKPDS study also reported higher prevalence of adverse dyslipidaemia in females than in males.[10] Such difference was not observed in our study; howeverthere was a significantly lower HDL-C level observed in males in our study.

The most prevalent type of dyslipidaemia in this study was combined dyslipidemia followed by mixed dislipidemia. Prevalence of raised LDL-C, Triglyceride and low HDL was comparatively low. These findings are different to diabetic dyslipidaemia described in Caucasians.[11] However, some studies including one conducted in Sri Lanka also have reported higher LDL cholesterollevel than TG in patients with T2DM.[12] Raised LDL cholesterol with relatively lower TG was alsoreported among patients with diabetes in otherdeveloping countries such as Nigeria, and India.[13] But, unlike in our study most of these studies-showed higher prevalence of low HDL. There could be many reasons for the different patternof diabetic dyslipidaemia in the cohort of patientsthat we studied. Unlike some of the published studies on diabetic dyslipidaemia, we studied only the newlydiagnosed patients with T2DM who were not on any treatment for dyslipidaemia. Therefore, the patternof Dyslipidaemia observed in this study reflects thetrue picture of diabetes dyslipidaemia. Higher prevalence of TG with low LDL reported in previous studies could be an effect of statins as it lowers LDL more than TG. The typical changes of raised TG withlow HDL-C seen in diabetic dyslipidaemia is thought to be due to insulin resistance together with dysfunction of the enzyme lipoprotein lipase (LPL).It was postulated that insulin resistance in adipocytespromote lipolysis, resulting in excessive free fattyacid (FFA) release into the blood and higherproduction of TG-rich very low-density lipoproteins (VLDL) by liver. Higher production of VLDLtogether with blunted LPLactivity contributes raisedTG levels.[14] Therefore, one of the possibilities forcomparatively lower levels ofTGseen in our study iseither lower degree of insulin resistance or higherLPL activity or both. Further studies are needed to evaluate insulin resistance among patients withraised LDL-C and raisedTG to answer this question. Higher prevalence of mixed dyslipidemia in this study may indicate higher prevalence of raised LDL and triglyceride in thegeneral population. Even though the prevalence oflipid abnormalities in the general population of Bangladesh is not well studied, studies conducted in urbanIndia had shown higher prevalence of raised LDL ingeneral population.[15] One of the other possibilities is the rising incidence of obesity in thelocal population.[16] As substantial proportion of subjects (60%) were obese in our study it could contribute for raised LDL levels.

Gender differences in the pattern of altered plasma lipids observed in this study are similar to major epidemiological studies from Western populations.However, in contrast to females who were more dyslipidemic with higher LDL and triglycerides in those studies, our study revealed higher proportions of males having raised triglycerides compared to females. Our study also showed that diabetes occurring in young individuals (<40 years) had significantly higher prevalence of elevated triglycerides. Even though not reaching significant level, other lipid parameters (raised LDL) were higher in individuals (age 40-59 years). This may be significant in the light of the finding that new onset diabetes in the South Asians is occurring at a relatively younger age and they need to be treated accordingly with appropriate measures.[17]

Other factors such as degree of hyperglycemia at diagnosis, showed significant effect on prevalence and pattern of dyslipidaemia.BMI and WC showed no significant effect on prevalence and pattern of dyslipidaemia. Therefore, predicting underlying dyslipidaemia based on BMI, and WC may not be appropriate.

Conclusion:

The prevalence of dyslipidaemia is high among newly diagnosed patients with T2DM affecting over 95%. In both genders, the most common pattern of dyslipidaemia is combined dyslipidaemia with raised LDL and low HDL-C. Typical pattern of diabetic dyslipidaemiawith raised TG and low HDL is not observed in themajority. Other associated and contributing factorsof dyslipidaemia such as obesity, waistcircumference at diagnosis had no significant effect on the pattern and prevalence of dyslipidaemia. But degree of hyperglycemia at diagnosis had significant effect on the pattern and prevalence of dyslipidaemia. Based on the findings of our study, we recommend lipid profile to be carried out in all patients at the diagnosis of T2DM and decision to perform lipid profile should not be based on factors such as obesity and WC.

Limitations:

The main limitations of our study include its single center study design and small sample size. Therefore, generalization of ourfindings accurately to whole diabetes population of Bangladesh may not be possible. The other limitationis not performing lipoproteins and genetic studies for further characterization of dyslipidaemia.

References:

1. Turner RC, Millns H, Neil HA, Stratton IM, Manley SE,MatthewsDR,et al. Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus: United Kingdom Prospective Diabetes Study (UKPDS: 23).BMJ1998;316 (7134): 823-8.
2. Cholesterol Treatment Trialists C, Mihaylova B, EmbersonJ, Blackwell L, Keech A, Simes J, et al.The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomisedtrials.Lancet 2012;380 (9841): 581-90.
3. Battaggia A, Font M. Statins for people at low risk of cardiovascular disease. Lancet 2012;380 (9856): 1815.
4. Haffner SM, American Diabetes A. Dyslipidaemia management in adults with diabetes.Diabetes Care 2004;27(Suppl 1): S68-71.
5. Cardiovascular Disease and Risk ManagementDiabetes Care.2016; 39(Suppl 1):S60-71.
6. Miller M, Kwiterovich PO. Isolated low HDL-cholesterol as an important risk factor for coronary heart disease.European Heart Journal 1990;11(Suppl H): 9-14.
7. Haffner SM. Management of Dyslipidaemia in adults with diabetes. Diabetes Care 1998;21(1):160-78.
8. Karter AJ, Ferrara A, Liu JY, Moffet HH, Ackerson LM,Selby JV. Ethnic disparities in diabetic complications in an insured population. JAMA: the journal of the American Medical Association. 2002; 287(19): 2519-27.
9. Davis TM, Millns H, Stratton IM, Holman RR, Turner RC.Risk factors for stroke in type 2 diabetes mellitus: United Kingdom Prospective Diabetes Study (UKPDS) 29.Archives of Internal Medicine 1999:159(10)1097-103.
10. UK Prospective Diabetes Study 27. Plasma lipids andlipoproteins at diagnosis of NIDDM by age and sex. Diabetes Care 1997;20 (11): 1683-7.
11. Stern MP, Haffner SM. Dyslipidaemia in type II diabetes.Implications for therapeutic intervention.Diabetes Care 1991;14 (12): 1144-59.
12. HettihewaLMG,Jayasinghe SS*et al*. Lipid abnormalities intype 2 diabetes mellitus patients in Srilanka .Galle Medical Journal 2007
13. Subburam R, Manohar CR, SubramaniyamP,Sachithanantham S, Paul AV, SankarapandianM.Dyslipidaemia among type 2 diabetes mellitus patients in a rural hospital in Erode district, Tamilnadu. *Journal of the Indian Medical Association* 2013;111 (1): 10-3.
14 Ginsberg HN. Diabetic dyslipidaemia: basic mechanisms underlying the common hypertriglyceridaemia and low HDL cholesterol levels. Diabetes1996; 45(Suppl 3):S27-30.
15. MisraA, Shrivastava U. Obesity and Dyslipidaemia in SouthAsians.Nutrients2013; (7): 2708-33.
16. Katulanda P, Jayawardena MA, Sheriff MH, Constantine GR, Matthews DR. Prevalence of overweight and obesity in Sri Lankan adults. Obesity reviews: an official*Journalof the International Association for the Study of Obesity*2010;11 (11): 751-6.
17. Mohan V, Deepa M, Deepa R, Shanthirani CS, FarooqS,GanesanA,et al.Secular trends in the prevalence of diabetes and impaired glucose tolerance in urban South India – the Chennai Urban Rural Epidemiology Study (CURES-17). *Diabetologia*2006; 49(6): 1175-8.

Prophylactic Effect of Pethidine In Preventing Postoperative Shivering: A Prospective Clinical Trial

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Abstract

Introduction: Postoperative shivering (POS) is rhythmic vibratory motions in one or more groups of muscles that caused after general or regional anesthesia. Prevention and early treatment of POS lead to not conflict with patient monitoring and also reduce cardio-respiratory and metabolic side effects in patients. The aim of this study is to assess the efficacy of pethidine on reducing postoperative shivering.

Methods: This randomized placebo-controlled double blind clinical trial included 80 patients scheduled for elective ENT operations, randomly divided to two groups. Group I (pethidine group) received intravenous pethidine 0.5 mg/kg and Group II (placebo group) received 2 ml normal saline 30 minutes before the anticipated completion of surgery. Anesthesia was induced and maintained equivalently for all. Patients were observed incidence and severity of shivering in postoperative period.

Results: There was no significant difference in terms of age, body weight, sex, ASA status, hemodynamics and temperature between the groups. In pethidine group four (10%) out of the 40 patients had postoperative shivering (POS), whereas 18 (45%) out of the 40 patients had POS in placebo group ($P<0.05$).

Conclusion: Intravenous pethidine is significantly effective in reducing post-operative shivering after general anesthesia.

Key words: Pethidine, General anesthesia, Postoperative shivering (POS).

Journal of Green Life Med. Col. 2019; 4(1): 20-23

Introduction:

Postoperative shivering (POS) is an accompanying part of general anesthesia, with an estimated rate up to 50%.[1] It has different unpleasant and stressful consequences for patients undergoing surgery due to some physiological changes including increasing oxygen consumption, hypoxemia, lactic acidosis and hypercarbia.[2] These changes, in addition to increasing intraocular and intracranial pressure, may complicate the recovery process during anesthesia and increase the wound pain.[3]

Several studies have investigated the underlying mechanisms of POS. Accordingly, thermoregulatory and non-thermoregulatory reactions are responsible in this regard.[4-6] So, different pharmacologic interventions were studied considering these reactions, but the precise origin of it is not understood yet.[7]

Various pharmacological agents have been used for prevention of POS, including pethidine, tramadol, clonidine, ketamine,nefopam, doxapram, ondansetron and physostigmine; but none of them have shown promise.[8-10]

Opioids (pethidine, sufentanyl, alfentanyl, tramadol) have significant role among the identified drugs because the effects of different opioids have been studied frequently in this regard and some of them are used for both treatment and prevention of POS and it considered as very effective anti-shivering drug.[5,11-13]

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Received: 30 September 2018 **Accepted:** 24 December 2018

Since POS is an accompanying part of general anesthesia with different unpleasant and stressful consequences for patients undergoing surgery, it seems that its proper management is necessary for both treatment and prevention. The aim of this study was to investigate the effect of pethidine preventing postoperative shivering in general anesthesia.

Methods:

In this prospective, randomized, double blind, clinical trial, 80 patients aged 20 50 years, scheduled for elective surgery under general anesthesia at National Institute of ENT Dhaka during the period of November 2017 to February 2018, were enrolled. Patients with a history of convulsions, history of using tricyclic antidepressants (TCAs), monoamine oxidase (MAO) inhibitors, and other cardiorespiratory, renal or hepatic insufficiency were excluded from the study. In addition, patients who were hemodynamically unstable or had severe bleeding during surgery were excluded. Written informed consent was obtained from every patient.Patients then were randomly selected to Group I (pethidine group) who received 0.5 mg/kg intravenous pethidine and Group II (placebo group) who received 2ml normal saline 30 minutes before the anticipated completion of surgery. The responsible anesthesiologist was blinded to the drug available in same 2 ml syringes. Anesthesia was induced by 1.5 mcg/kg fentanyl, 2 mg/kg propofol and 1.5 mg/kg succinylcholine and maintained by halothane in an inspired mixture of 40% oxygen and 60% N_2O after intubation. Vecuronium was administered to keep muscle relaxation. The patients were mechanically ventilated. Room temperature was set at 22°-24 °C. After completion of surgery muscle relaxant was reversed by neostigmine and atropine then extubated the patient and shifted to recovery room. Heart rate, blood pressure and body temperatures were measured every 5 minutes interval throughout the intraoperative and postoperative period. Patients were observed for shivering in postoperative period as per shivering scale described by Crossley & Mahajan[14] and Tsai & Chu.[15]

Grading scale of postoperative shivering validated by Crossley & Mahajan[14] and Tsai & Chu.[15]

0 = No shivering.

1 = Piloerection or peripheral vasoconstriction but no visible shivering.

2 = Muscular activity in only one muscle group.

3 = Muscular activity in more than one muscle group but not generalized shivering.

4 = Shivering involving the whole body.

Statistical analysis

Quantitative data were expressed as mean ± standard deviation (SD). Qualitative data were expressed as frequency and percentage. The following tests were done: Independent-samples t-test of significance was used when comparing between two means. Chi-square test of significance was used in order to compare proportions between two qualitative parameters.

P-value <0.05 was considered significant. P-value >0.05 was considered insignificant.

Results:

There was no significant difference in terms of age, body weight, sex, ASA status, hemodynamics and temperature between the groups (Table I). In group I four (10%) out of the 40 patients had postoperative shivering (POS), whereas 18 (45%) out of the 40 patients had POS in group II (P<0.05). Grade 1 POS was lower number of patients in group I when compared with group II (3 versus 12; P<0.05). Grade 2 POS was also lower number of patients in group I when compared with group II (1 versus 4; P<0.05) and grade 3 POS was only present in group II (0 versus 2; p<0.05.There was no grade 4 POS in either of the two groups (Table II). The baseline values of systolic and diastolic blood pressure, heart rate and temperature in both groups were similar and there was no any adverse effect.

Table-I

Demographic and operative details of patients between Pethidine and Placebogroup

Demographic and operative details	Group I (Pethidine group)n=40	Group II (Placebo group)n=40
Age (Years)	36.7±7.3	37.4±8.2
Weight (Kg)	66.4±8.2	64.7±7.3
Sex M/F	23/17	24/16
ASA physical status I/II	37/3	36/4
Mean basal heart rate (bpm)	80.6±7.8	82.8±9.6
Mean basal systolic BP (mm Hg)	116.7±9.2	118.5±9.7
Mean basal diastolic BP (mm Hg)	80.5±8.3	82.8±7.8
Mean duration of surgery (min)	72.8±9.4	74.4±7.9
Mean body temperaturesduring surgery (^{0}C)	36.28±0.38	36.33±0.34
Mean body temperaturein recovery room (^{0}C)	36.47±0.43	36.49±0.48

Table II

Incidence and severity of postoperative shivering

Postoperative shivering(POS)	Group I(Pethidine group) n=40	Group II(Placebo group) n=40	p value
Incidence of POS number (%)	4 (10%)	18 (45%)	p<0.05
Grading of POS number (%)			
0	36 (90%)	22 (55%)	p<0.05
1	3 (7.5%)	12 (30%)	p<0.05
2	1 (2.5%)	4 (10%)	p<0.05
3	0	2 (5%)	p<0.05
4	0	0	-

Discussion:

Postoperative shivering is one of the most common problems in the early recovery phase following general anesthesia.Several medications have been suggested for the prevention and treatment of postoperative shivering.

The present study shows valuable preventive effect of pethidine on postoperative shivering compared to placebo group, here pethidine group 4 (10%) out of the 40 patients had postoperative shivering (POS), whereas 18 (45%) out of the 40 patients had POS in placebo group (P<0.05). Grade 1 POS was lower number of patients in pethidine group when compared with placebo group (3 versus 12; P<0.05). Grade 2 POS was also lower number of patients inpethidine group when compared with placebo group (1 versus 4; P<0.05) and grade 3 POS was only present in placebo group (0 versus 2; p<0.05.There was no grade 4 POS in either of the two groups.

Many studies have confirmed the effectiveness of pethidine in preventing postoperative shivering. Similar to present study Ramalingarajuet al[16] compared prophylactic effect of pethidine and ketamine on postoperative shivering after general anesthesia. Postoperative shivering was reported in only 3.7% patients in pethidine group in their study, which is less than present study.

A comparative study done by Zavarehet al[17] on prevention of postoperative shivering, using ketamine, dexamethasone and pethidine. They found 11.1% patients had POS in pethidine group.

Another study of Asl et al[1] recorded 20% patients had postoperative shivering when they used pethidine as pretreatment for prevention of POS.

So, result of present study is similar to those of the result of Zavarehet al[17] and higher than those ofAsl et al.[1]

Conclusion:

Intravenous pethidine 0.5 mg/kg body weight 30 minutes before the anticipated completion of surgery could effectively decreases the incidence and severity of postoperative shivering.

References:

1. Asl ME, Isazadefar K, Mohammadian A, Khoshbaten M. Ondansetron and meperidine prevent postoperative shivering after general anesthesia. Middle East J Anesthesiol. 2011;21:67–70.
2. Gecaj-Gashi A, Hashimi M, Sada F, Salihu S, Terziqi H. Prophylactic ketamine reduces incidence of postanaesthetic shivering. Niger J Med. 2010;19:267–70.
3. Sessler DI. Temperature monitoring. In: Miller RD, editor. Miller's anesthesia. 6th ed. Philadelphia: Churchill Livingstone; 2005; 1571–97.
4. Bhattacharya PK, Bhattacharya L, Jain RK, Agarwal RC. Post Anesthesia Shivering: A review. Indian J Anaesth. 2003;47:88–93.
5. Shrestha AB. Comparative study on effectiveness of doxapram and pethidine for postanaesthetic shivering. J Nepal Med Assoc. 2009;48:116–20.
6. Alfonsi P. Postanaesthetic shivering: Epidemiology, pathophysiology, and approaches to prevention and management. Minerva Anestesiol. 2003;69:438–42.
7. Mahamood MA, Zweifler RM. Progress in shivering control. J Neurol Sci. 2007;261:47–54.
8. Dal D, Kose A, Honca M, Akinci SB, Basgul E, Aypar U. Efficacy of prophylactic ketamine in preventing postoperative shivering. Br J Anaesth 2005;95:189 92.
9. Tewari A, Katyal S, Singh A, Garg S, Kaul TK, Narula N. Prophylaxis with oral clonidine prevents perioperative shivering in patients undergoing transuretheral resection of prostate under subarchanoid blockade. Indian J Urol 2006;22:208 12.
10. Alfonsi P, Adam F, Passard A, Guignard B, Sessler DI, Chauvin M. Nefopam, a nonsedativebenzoxazocine analgesic, selectively reduces the shivering threshold in unanesthetized subjects.Anesthesiology 2004;100:37 43.

11. Roy JD, Girard M, Drolet P. Intrathecalmeperidine decreases shivering during cesarean delivery under spinal anesthesia. AnesthAnalg. 2004;98:230–34.
12. Techanivate A, Dusitkasem S, Anuwattanavit C. Dexmedetomidine compare with fentanyl for postoperative analgesia in outpatient gynecologic laparoscopy: A randomized controlled trial. J Med Assoc Thai. 2012;95:383–90.
13. Miller RD (eds). Anesthesia, 5th ed. Philadelphia: Churchill Livingstone; 2005.
14. Crossley AWA, Mahajan RP. The intensity of postoperative shivering is unrelated to axillary temperature Anaesthesia 1994;49:205-7.
15. Tsai YC, Chu KC. A comparison of tramadol, amitriptyline, and meperidine for postepidural anesthetic shivering in parturients.AnesthAnalg 2001;93:1288-92.
16. Ramalingaraju A.V.S, Ranjan DS, Neethika M. Post Operative Shivering: Prophylactic Effects of Ketamine and Pethidine, A Comparative Study in Tertiary Care Hospital. IOSR Journal of Dental and Medical Sciences. 2017; 16 (3): 12-15.
17. Zavareh SMHT, Morovati L, Koushki AM. A comparative study on the prophylactic effects of ketamine, dexamethasone and pethidine in preventing postoperative shivering. J Res Med Sci 2012; 17(2): 175-81.

ORIGINAL ARTICLE

Measles In Vaccinated and Unvaccinated Patients In Urban Setting

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Abstract

***Introduction:** Measles is a highly contagious viral illness that occurs worldwide. Following exposure, approximately 90 percent of susceptible individuals will develop measles. It may cause mild to severe complications such as diarrhea, pneumonia, otitismedia, bronchitis and infection of brain leading to mortality and morbidity of a large number of children. The objectives of this study was to find out measles cases among vaccinated and unvaccinated children with their age distribution in urban setting.*

***Methods:** This cross sectional descriptive study was done in the pediatric department (both in and out) of Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh. All of 80 clinically suspected measles cases who attended the department during the period from March 2011 to September 2012 were studied. Under full aseptic preparation blood samples were collected and tested for IgM antibody against measles virus. Out of these 80 cases, 62 patients were laboratory confirmed measles by the presence of Anti IgM and were finally enrolled in the study.*

***Results:** Out of 80 clinically suspected measles cases 62 (77.5%) were positive for measles anti IgM. Of these 62 patients 43 (69.35%) were unvaccinated and 19 (30.65%) were vaccinated with a single dose of measles vaccine. Among the confirmed measles cases 19 (30.65%) patients were below 9 months of age and none of them was found vaccinated as they did not attain the minimum age of measles vaccination as per EPI schedule. On the other hand, 19 (52.78%) out of 36 patients who were above 12 month of age, were found vaccinated but developed measles.*

***Conclusion:** Measles may occurs before 9 months, even in a significant number before 6 months of age due to fall of protective maternal immunity; and a decline of protective level of immunity or failure of seroconversion of vaccine is also evident after one dose of vaccination against measles.*

***Keywords:** Measles cases in urban settings, Seroconversion of measles vaccine, Protective maternal immunity for measles.*

Journal of Green Life Med. Col. 2019; 4(1): 24-27

Introduction:

Measles is a highly contagious infectious disease caused by the measles virus. It is an airborne disease and spreads easily through cough and sneezing. It may also spread through contact with saliva or nasal secretion. Measles affects about 20 million people a year, and those are mainly in the developing areas of Asia and Africa.[1] In the prevaccine era more than 90% of people experienced the infection during their childhood. Natural measles infection results in life-long immunity. The number of deaths reported worldwide between 2000 and 2010 decreased from 7,50,000 in 2000 to 1,97,000 in 2007.[2] The infection is characterized by fever, rash, cough, coryza, and conjunctivitis. Following exposure 90 percent of the susceptible individuals will develop measles.[1,3] It is contagious from 5 days before the appearance of rash to 4 days after ward[4]. Measles virus infection can cause a variety of clinical syndrome depending on the immunity status against measles, which are clinical measles infection, modified measles infect and atypical measles infect.[3]

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Received: 20 November 2018 **Accepted:** 24 December 2018

About 30% of the reported cases of measles may develop one or more complication. These complications observed are otitis media (7-9%), pneumonia (1-6%), diarrhoea (6%), blindness and post-infectious encephalitis (1/1000 cases). Though less common, but a very severe complication is subacute selerosing pan encephalitis (SSPE), which may occurs in 1 per 1,00,000 cases of measles.[4] Case-fatality is highest among infants aged under 12 months. Measles vaccine is one of the most effective vaccine in the world. A single dose of vaccine will give around 93 percent protection and a second dose (booster dose) increase the effectiveness of vaccine to more than 97 percent.[5,6] So, there will be cases who may develop measles even after 2 doses of measles vaccination.[5,8-10]

In Bangladesh, like developed and some other developing countries 2 doses of measles vaccine have been introduced from 2012, with a goal to eliminate measles and rubella by 2020.[10] In this schedule the 1st & 2nd doses are given after the age of 9 months & 15 months respectively. But, both sporadic and outbreak of measles are ongoing in Bangladesh and it has picked up in 2016 – 17.[10,11] In Bangladesh and some other countries, studies showed that immunity gap is persisting primarily in infants and young children, but it is also observing in adolescents.[10,12,13] There are reports about development of measles in a significant number of infants below 9 months of age[10,13] and occurrence of measles even after receiving 2nd dose of measles vaccine due to decline of protective level of antibody titer.[13,14] Rationale for offering the vaccine at an earlier time is based on an outbreak of measles and this schedule may be conducted routinely in high – risk areas.[14,15] But there is chance of less seroconversion in early 2- doses schedule.8,13,16 So, questions are there about early 2-doses also. Our study will help to find out the situation in Bangladesh and thereby to adopt more effective measles elimination program in our country.

Methods:

This cross sectional descriptive study was done in the in and out patient department of pediatrics, Sir Salimullah Medical College and Mitford Hospital, Dhaka. Study period was from March 2011 to September 2012. Parents or care givers of all the 80 clinically suspected measles cases attending the in or out patient department were interviewed through a structured questionnaire. A clinical measles was diagnosed as a case with fever and rash, with the presence of any one of cough, coryza and conjunctivitis.[1,10,16] Vaccination status was recorded from documents or from the verbal history given by the parent or caregiver if no record was available.[17] After obtaining consent from the parents or caregivers blood samples were collected from the cases. Under full aseptic preparation 3 ml of blood was collected from infants and 5 ml from other children within 3 – 28 days of appearance of rash. The samples were preserved at 2º- 8ºC temperature and send to National Polio and Measles Laboratory, Institute of Public Health (IPH), Mohakhali, Dhaka for serological diagnosis of measles. Measles cases were diagnosed by detection of IgM antibody against measles by enzyme linked immunosorbent assay (ELISA) as the standard test for routine measles surveillance, as per recommendation of WHO.[4,10,16,17] Serum specimens that give negative result for measles IgM were tested for rubella – specific IgM. The lab test results were analyzed age wise among vaccinated and unvaccinated children. Finally, the measles IgM positive (confirmed) cases were enrolled in our study. Data were stored and analyzed with standard computer software (SPSS-15). Ethical clearance was taken from concerned authority.

Results:

Of our study population 17 (27.14%) came from urban (Dhaka city) area, 22 (35.48%) from semi- urban and 23 (37.09%) from rural areas. Out of 80 clinically diagnosed measles 62 (77.50%) were found positive for IgM against measles, 3 (3.75%) were positive for IgM against rubella and 15 (18.75%) were discarded for improper or inadequate sampling, Fig – 1. Among the 62 lab confirmed measles patients 43 (69.35%) were unvaccinated and 19 (30.65%) were vaccinated, Fig–2.

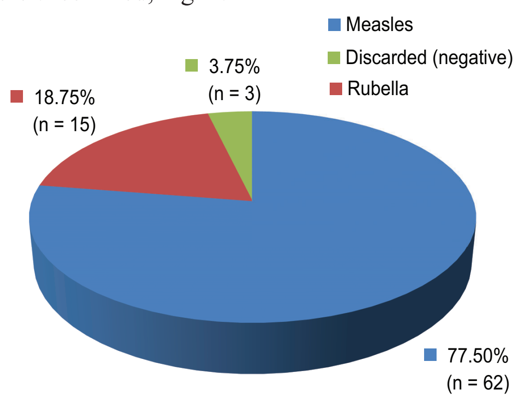

Fig - 1. *Shows percentage of IgM positive measles among clinical measles (n = 80).*

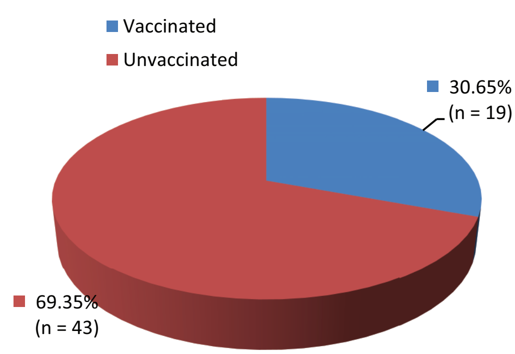

Fig - 2. *Shows percentage of unvaccinated & vaccinated child among IgM positive measles (n = 62).*

Among the 62 confirmed measles patient fever and rash was present in all (n =62, 100%) cases; but among other features cough was the most common (n = 58, 93.55%) clinical feature followed by coryza (n = 45, 72.58) and conjunctivitis (n = 26, 41.94%) Table–1.

Table - I

Showing clinical features of measles as observed (n = 62).

Clinical Features	Number of patient shows	Percentage
Fever	62	100%
Rash	62	100%
Cough	58	93.55%
Coryza	45	72.58%
Conjunctivitis	26	41.94%

Regarding age distribution of the patients 10 (16.13%) were below 6 months, 9 (14.52%) were between 6 – 9 months and 7 (11.29%) patients fall between 9 months – 12 months of age. None of these 26 (41.94%) patients was found vaccinated. Above these age group there were 36 patients, of which 22 (35.48%) were between 12 months – 36 months of age and 14 (22.58%) were above 36 months of age. Among these 36 patients 19 (52.78%) were vaccinated but 17 (47.22%) were unvaccinated. But between 12 months to 36 months age group majority (n = 14, 63.64%) were found unvaccinated, on the other hand above 36 months of age majority (n = 11, 78.57%) of children were vaccinated. Table-II.

Table-II

Shows age distribution of measles among unvaccinated and vaccinated children (n = 62).

Age	Number	%	Unvaccinated		Vaccinated	
			N	%	N	%
< 6 months	10	16.13	10	100	0	0
6 – 9 months	9	14.52	9	100	0	0
9 – 12 months	7	11.29	7	100	0	0
12 – 36 months	22	35.48	14	63.64	8	36.36
≥ 36 months	14	22.58	3	21.43	11	78.57

Discussion:

In our study out of 62 IgM antibody positive measles patient 19 (30.65%) were vaccinated and 43 (69.35%) were found unvaccinated. Of this 19 vaccinated child 11 (78.57%) were ≥36 months of age out of a total 14 child of this age group. This indicate declining of protective level of antibody or failure of seroconversion after 1 dose of measles vaccination. Declining of antibody after 1 dose of vaccination were found in other studies from United States,[6] Wales[7] and Canada[8] . In this study out of 43 unvaccinated patients 19 (30.65%) were below 9 months of age and of these child 10 (16.13%) were below 6 months. This significant number (30.65%) of patients developed measles before reaching the age of 1st dose measles vaccination as per present EPI schedule.[11] Development of measles before this optimum age of measles vaccination had been reported in other studies from Bangladesh[10,15,16] and Florida, USA.[13]

In this study, 21 (72.41%) patients of 9 months to 36 months of age was found unvaccinated out of total 29 measles cases of this age group. So, immunity gap is persisting and this observation is supported by other studies.[10,14] To ensure individual protection against measles the WHO recommends application of two doses of the vaccine to achieve coverage higher than 95% to ensure herd immunity.[2] Bangladesh introduced first dose of measles vaccine at 9 months in 1989 and second dose at 15 months of age in 2012. The coverage in 2016 was 94% and 93% for measles containing vaccine (MCV1) and MCV2.[10,17] In developed countries recommended time for 1st and 2nd dose vaccination against measles is 12 months to 15 month and 3 years to 6 years respectively.[6,7,14,18]

The case fatality from measles is highest in infants under 12 months of age and highest number of measles deaths are observed in Africa and South-East Asia.[4] In Bangladesh, a significant number of measles patients are being observed before the age of 9 months,[10,15,19] and on the other hand cases are also reported even after two dose of vaccination from developed countries.[5,9] Strategies recommended for elimination of disease depends on local epidemiology, vaccination coverage and ability of the health system to deliver vaccine. So, from our study and report from other observers, in high-risk areas vaccination at 6 months may be started followed by routine revaccination of two more doses (one dose at 12 – 15 months of age and other through 3 - 6 years of age or at least 28 days later).[13,20]

Limitations of the study:

Small sample size and single center study.

Conclusion:

In our study of 62 IgM antibody positive confirmed measles patients 43(69.35%) were unvaccinated, of which 19 (30.65%) were below 9 months and10(16.13%)were below 6 months of age. On the other hand, 11 (78.57%) child out of 14 patients of ≥ 36 months developed measles even after vaccination with MCV1. Results of this study clearly indicates fall of protective maternal immunity in a large number of children before 9 months, and in a significant number before 6 months of age; on the other hand decline of protective level of immunity or failure of seroconversion is also evident after the MCV1 vaccination.

The 1st dose of vaccination against measles may be started at 6 months of age, and there-after 2 doses of vaccines at 12-15 month and at 4-6 years should be given to eliminate measles and thereby reducing mortality and morbidity from measles.

Acknowledgement:

We express our heartfelt gratitude and regards to Dr. Anwarul Haque Chowdhury, Virologist National Polio and Measles Laboratory, IPH, Mohakhali, Dhaka. We are also highly grateful to the authority of IPH for extending hands to diagnose measles by laboratory investigations.

References:

1. Wikipedia.Measles.https://en.wikipedia.org/wiki/Measles.
2. Smetana J, Chlibek R, Hanovcova I, Sosovickova R, Smetanova L, Gal P, etal. Decreasing Semoprevalence of Measles Antibodies after Vaccination – Possible Gap in Measles Protection in Adults in the Check Republic. http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0170257.
3. G Hayley, M A Yvonne. Measles : clinical manifestations, diagnosis, treatment, and prevention – update. https://www.update.com/contents/measles-clinical-manifestation-diagnosis-treatment-and-prevention.
4. Measles rubella and CRS : disease description, epidemiology and diagnosis. Surveillance Guidelines for Measles, Rubella and congenital Rubella Syndrome in the WHO European Region Geneva : World Health Organization; 2012 Dec. https://www.ncbi.nlm.nih.gov/books/NBK143257/
5. Palmen K M. Why did Vaccinated People Get Measles at Disneyland? Blame the Unvaccinated. https://www.wired.com/2015/01/vaccinated-people-get-measles-disneyland-blame-unvaccinated/
6. Iannelli V. Measles Prevention. https://www.verywellhealth.com/how-to-prevent-measles-2633847
7. Petrovic M, Roberts R, Ramsay M. Second dose of measles, mumps, and rubella vaccine: questionnaire survey of health professionals.BMJ 2001; 322 doi: https://doi.org/10.1136/bmj.322.7278.82. Cite this as: BMJ 2001;322:82
8. Serres G D,Boulianne N, Defay F, Brousseau N, Benoit M, Lacoursiere S, etal. Higher Risk of Measles when the First dose of a 2- Dose Schedule of Measles Vaccine is Given at 12 -14 Months Versus 15 Months of Age. Clinical Infectious Diseases 2012;55(3):394-402.
9. Cherry, et al. Measles in Vaccinated and Unvaccinated Patients. Clin Infect Dis; ePub 2018 June 7. doi : 10. 1093/cid/ciy286. https://www.mdedge.com/ familypracticenews/clinical-edge/summary/vaccines/measles-vaccinated-unvaccinated-patients.
10. Strategic Plan for the Elimination of Measles, Rubella and CRS in Bangladesh 2017. Expanded Program on Immunization, Directorate General of Health Services, Ministry of Health & Family Welfare, Govt. of the People Republic of Bangladesh.
11. Expanded Programme on Immunization (EPI) Fact Sheet, Bangladesh 2017. Directorate General of Health Services, Ministry of Health & Family Welfare, Govt. of the People Republic of Bangladesh.
12. Baron CWL, Becler J, Sullivan B J et al. Persistance of Measles Antiobodies After 2 Doses of Measles Vaccine in a Post Elimination Environment. Arch Pediatr Adolesc Med. 2007;161(3):294-301.doi:10.1001/archpedi.161.3.294.
13. Hutchins S S, Dezayas A, Blond K L, Health J, Bellini W, Audet S, etal. Evaluation of an Early Two – Dose Measles Vaccination Schedule. American Journal of Epidemiology 2001;154(11):1064-1071.
14. Herrera O R, Thornton T A, Helms R A, Foster S L. MMR Vaccine : When is the Right Time for Second Dose? J Pediatr Pharmacol Ther.2015;20(2):144-148.
15. Wiesen E, Wannemuehler K, Goodson J L, Anand A, Mach D, Thapa A, etal. Stability of the Age Distribution of Measles Cases Over Time During Outbreaks in Bangladesh, 2004-2006. The Journal of Infections Diseases 2011;204(1): S414-S420.
16. Akramuzzaman S M, Cutts F T, Hossain M J, Wahedi OK, Nahar N, Islam D, etal. Measles Vaccine effectiveness and risk factors for measles in Dhaka, Bangladesh. Bulletin of the World Health Organization 2002;80:776-782.
17. Khanal S, Bohara R, Chacko S, Sharifuzzaman M, Shamsuzzaman M, Goodson J L, etal. Progress Toward Measles Elimination – Bangladesh, 2000 – 2016. Centers for Disease Control and Prevention Weekly July2017:66(28):753 – 757.
18. Peltola H, Heinonen O P, Valle M, Paunio M, Virtanen M, Kamrenko V, etal. The Elimination of Indigenous Measles, Mumps, and Rubella from Finland by a 12-Years, Two Dose Vaccination Program. N Engl J Med 1994;331:1397 – 1402.
19. Bangladesh : Measles Outbreak – Sep 2017. Overview https://reliefweb.int/ disaster/ep-2017-000187-bgd.
20. Measles Vaccination. LSU Health, New Orleans. Measles Outbreak 2014 – 15. Isuhsc.edu/measles/Measles_ Vaccination aspx.

Role of FNAC in the Management of Breast Lump and its Correlation with Histopathology

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Abstract

Introduction: Breast lump is a common surgical problem. A descriptive cross-sectional study was carried out to evaluate the effectiveness of fine needle aspiration cytology (FNAC) as a diagnostic method. One hundred cases of clinically palpable breast lumps were subjected to FNAC. In all cases tissues were examined histologically after excisional biopsy or definitive surgery. Results of FNAC were compared with histological diagnosis.

Objective of study: To evaluate the role of FNAC in the management of breast lump and its correlation with histopathology.

Methods: This cross-sectional descriptive study was carried out at Bangabandhu Sheikh Mujib Medical University & Dhaka Medical College Hospital during the period from March, 2011 to August, 2011. Total number of 100 female patients between 10-60 years of age with palpable breast lump were selected. Patients with no discrete palpable lump and in whom excision of lump was not done were excluded.

Results: In this study the sensitivity of FNAC was 85.7% for the presence of carcinoma and the specificity was 100% for the absence of malignancy. The Positive predictive value was 100% and negative predictive value was 93.5% and the overall diagnostic accuracy of this series was 95.3%. In this study false negative rate of cytological diagnosis was 6.45% but there was no false positive diagnosis.

Conclusion: Due to high diagnostic accuracy, FNAC can be used as a diagnostic method in the management of breast diseases which is of reasonable sensitivity and specificity. The cytological diagnosis is highly reliable when diagnosis of cancer is made. If there is clinical suspicion of malignancy, final diagnosis should be done by surgical excision followed by histopathology.

Key words : FNAC, Breast lump, Biopsy, Carcinoma

Journal of Green Life Med. Col. 2019; 4(1): 28-33

Introduction

Breast is the common organ for cancer worldwide.[1] Benign breast diseases are also common.[2] Palpable breast abnormalities are described as breast lump or breast thickening.[3] For women, the risk of developing breast cancer up to age of 74 years is approximately 8% and it is the commonest cause of death from cancer in women after lung cancer.[4] Various diagnostic methods have been developed to evaluate palpable & non palpable breast lump like mammography, USG, core needle biopsy, open excisional biopsy, thermography, fine needle aspiration cytology (FNAC).[5,6] FNAC of breast is a sensitive and specific method for diagnosis of breast tumor before or instead of surgical biopsy. It is a very simple & safe procedure which can be performed at OPD without much discomfort to the patient. Results of FNAC can be obtained more quickly than surgical biopsy.[7,8] Moreover, the procedure is cost effective than other routine histological section.[9] Although FNAC does not replace frozen section histology, it is valuable in the identification of unsuspected malignancy & by giving accurate preoperative diagnosis

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Received: 29 November 2018 **Accepted:** 24 December 2018

in preparation of patients for mastectomy. Complications of FNAC are rare. If clear fluid is obtained, a clinical diagnosis of benign cystic disease can be made with high degree of reliability. If the findings of breast aspiration cytological examination is suspicious or to disclose malignancy that patient should be referred as soon as possible for definitive open biopsy & appropriate surgical treatment.

Methods :

The present study was a descriptive cross-sectional study carried out at Bangabandhu Sheikh Mujib Medical University and Dhaka Medical College Hospitals during the period from March 2011 to August 2011. All the female patients between 10-60 years of age with palpable breast lump were evaluated. Patients were selected from outdoor department as well as from surgical wards of Dhaka Medical College Hospital and Bangabandhu Sheikh Mujib Medical University.

Patient with no discrete palpable lump and in whom excision of lump was not done were excluded. Cystic lumps and patients with fungating, ulcerated lumps were not included in this study. A total of 100 patients meeting all the enrollment criteria were selected consecutively from the study population.

Results:

Table 1

Distribution of patients according to the age group

Age (years)	Number of patients (n=100)	Percentage (%)
0-9	0	0.0
10-19	16	16.0
20-29	18	18.0
30-39	28	28.0
40-4	0	
9	24	24.0
50-60	14	14.0

Table 1 shows distribution of the patients according to the age group majority of the patients were in the age group of 30-39 years.

Table-II

Clinical diagnosis

Clinical diagnosis	Number of patients (n=100)	Percentage (%)
Benign	56	56.0
Malignant	32	32.0
Equivocal	12	12.0

Table 2 shows the clinical diagnosis. Before aspiration, clinical diagnosis were established by taking thorough history and performing physical examination Clinically a reasonably confident diagnosis could be made in 88 (88.0%) cases of which 56 (56.0%) were diagnosed to be benign and 32 (32.0%) were to be malignant. Of the remaining 12 (12.0%) cases the clinical diagnosis were uncertain and a suspicion of malignancy could not be ruled out.

Table-III

Cytological diagnosis

Cytological diagnosis	Number of patients (n=100)	Percentage (%)
Benign	62	62.0
Malignant	24	24.0
Suspicious of malignancy	55.0	
Atypical	22.0	
Unsatisfactory smears	77.0	

Table 3 shows the cytological diagnosis. Out of 100 cases cytologically 62 (62.0%) cases were found to be benign, 24 (24.0%) cases diagnosed as malignant, 5 (5.0%) cases were found to be suspicious of malignancy, 2 (2.0%) cases were reported atypical and in 7 (7.0%) cases smears were unsatisfactory for cytological examinations.

Table-IV

Comparison of clinical and cytological diagnosis

Clinical diagnosis	Number of patients (n=100)	Percentage (%)	Cytological diagnosis	Number of patients (n=100)	Percentage (%)
Benign	56	56.0	Benign	62	62.0
Malignant	32	32.0	Malignant	24	24.0
Equivocal	12	12.0	Suspicious	5	5.0
			Atypical	2	2.0

Table 4 shows that 56 cases (56.0%) were diagnosed to be benign clinically but cytologically 62 cases (62.0) were benign. Clinically 32 (32.0%) cases were malignant but cytologically 24 (24.0%) cases were malignant. Clinically in 12 (12.0%) cases diagnosis were uncertain cytologically 5 cases (5.0%) were suspicious of malignancy and 2 (2.0%) cases were atypical

Finally histologic diagnosis was made in every case.

Table - V

Histological Diagnosis

Histological Diagnosis	Number of patients (n=100)	Percentage (%)
Benign	66	66.0
Malignant	34	34.0

Table V shows histological diagnosis of all patients. Histologically 66 (66.0%) cases were benign and 34 (34.0%) cases were malignant.

The table 6 shows the comparison of cytological diagnosis with the final histological diagnosis shows that out of 62 benign lesions diagnosed cytologically, 4 cases were found malignant histologically i.e.false negative 4 (6.45%) 24 malignant cases diagnosed cytologically were confirmed histologically (i.e. no false positive case was found). Out of 5 suspicious cases reported cytologically, 1 (20.0%) case was found to be benign and 4 (80%) cases were to be malignant histologically and 2 (100.0%) atypical cases were benign after histology. Out of 7 unsatisfactory smears 5 (71.43%) cases were benign and 2 (28.57%) cases were malignant histologically.

Table-VI

Comparison of cytological diagnosis and histological diagnosis.

Cytological Diagnosis	Histological Diagnosis				
Diagnosis	Benign No of patients	Malignant No of patients	Percentage (%)	No of patients	Percentage (%)
Benign	62	58	93.55	4	6.45
Malignant	24	0	0.0	24	100.0
Suspicious of malignancy	5	1	20.0	4	80.0
Atypical	2	2	100.0	0	0.0
Unsatisfactory	7	5	71.43	2	28.57

Table-VII

*Sensitivity and Specificity of aspiration cytology**

	Histological diagnosis				
Cytological diagnosis	No	Malignant	Benign	Sensitivity	Specificity
Malignant	24	24	0	85.7%	100.0%
Benign	62	4	58		

*Only cases with a definitive diagnosis of benign and malignant were considered.

Table -VII shows that sensitivity of aspiration cytology was 85.7% for the presence of malignancy and specificity 100% for the absence of carcinoma.

Table -VIII

*Diagnostic accuracy of aspiration cytology**

Cytological diagnosis	No of cases	No of cases with correct diagnosis	Diagnostic accuracy Predictive value
Benign	62	58	93.5%
Malignant	24	24	100.0%
Total	86	82	95.3%

* Only cases with a definitive diagnosis of benign and malignant were considered.

Table 8 shows that the negative predictive value of aspiration cytology for benign was 93.5% and positive predictive value for malignancy was 100% and overall diagnostic accuracy is 95.3%.

Discussion:

Fine needle aspiration cytology is a diagnostic method which has been thoroughly validated in many tissues including thyroid, breast, lymph node, salivary gland, prostate and other tissues. In essence the technique requires the insertion of a fine bore hollow needle through which groups of cells are withdrawn under negative pressure. Cellular material thus obtained is smeared on a slide and examined cytologically.

The method does not permit histological diagnosis, but the accuracy with which malignant cells can be identified by an experienced cytologist leaves the diagnosis of malignancy beyond doubt in cases where an adequate cytological sample is obtained. It is now widely used as a method of detecting the nature of tumor mass of various organs. Although the principal objective was to assess the value of aspiration cytology in confirming the clinical diagnosis in cases of breast cancer, an important result has been the diagnosis of malignancy in some clinically benign lesions. An equally important aspect of the method is the advantage of accurate preoperative diagnosis in the management of the patients with breast cancer.[5]

Fine needle aspiration cytology can be performed as an outpatient procedure. It eliminates the hazards of anesthesia. It is less traumatic, less expensive than open biopsy. It yields results more quickly than surgical biopsy. FNAC is a safe procedure with low complication rate. The accuracy of FNAC exceeds that of other preoperative diagnostic method. It provides better opportunity of treatment planning and more definitive discussion with the patient. It avoids open biopsy in inoperable cases. It is valuable in the identification of unsuspected malignancy. Needle aspiration is valuable technique for the diagnosis of breast cancer and follow-up examinations (Clinical, Mammographic) as may be the case with surgical| biopsies. It can be repeated without causing much discomfort.[2,5,10,15]

Patient with a breast mass or abnormal tissue area should have needle aspiration biopsy.[10] Morphological diagnosis of lesion from cytological smears needs experience. In this study the slides were grouped into five a) unsatisfactory smears b) benign c) atypical d) suspicious of malignancy and e) malignant.

The clinical diagnosis was recorded as benign, malignant and equivocal in which diagnosis was not certain. The final diagnosis was obtained in each case by paraffin section histology. In the present study examination was done on 100 cases.

In the present study satisfactory material for cytological examination were obtained from 93 cases 7 smears were unsatisfactory for cytological examination. Cytological assessment showed 62 (62.0%) benign, 24 (24.0%) malignant and 5 (5.0%) cases were suspicious of malignancy and 2 (2.0%) cases were atypical. All lesions with a cytological diagnosis of malignancy were confirmed by histological examination. So there was no false positive cytological diagnosis in this study Histological examination in 62 cytologically benign lesions showed that 4 were malignant giving a false negative rate of 6.45%. Four suspicious (80.0%) and 2 unsatisfactory smears (28.57%) were also malignant histologically. Two atypical cases were benign after histological examination.

Considering only the definitive diagnosis of benign and malignant, the sensitivity of this study was 85.7% for the presence of carcinoma and specificity was 100% for the absence of malignancy. The negative predictive value of this series for benign was 93.5% and positive predictive value for malignancy was 100% and the overall diagnostic accuracy was 95.3%. These findings are almost consistent with the result of other studies.[5,13,14]

Furnival[5] found that the diagnostic accuracy of aspiration cytology was 96.0% for benign and 96.1% for malignant and overall accuracy was 95.5%. The diagnostic accuracy of the study was almost identical with their series. Eisenberg[12] et al. found in their study of a large series that aspiration cytology would have a sensitivity of 84% for the presence of carcinoma a specificity of 97% for the absence of carcinoma, a predictive value of 99% for a positive diagnosis and a predictive value of 56% for a negative diagnosis; the diagnostic efficiency would be 86%. The finding of this study appears better. This is probably due to small number of this series. Hammond[14] found in their study that aspiration cytology would have a sensitivity of 94% while the specificity was 98%, the overall efficiency of the test was thus 96%. The predictive value were 98% for a positive test and 94% for a negative test. The result of this series is roughly consistent with their study. Cook and Robinson (1991) achieved a diagnostic accuracy of 96.9%, a positive predictive value of 98.4% and a negative predictive value of 95.7%. These figures are again roughly consistent with the results of this study.

In this study the number of unsatisfactory smears for cytology were 7 (7.0%), cytologically 5 (5.0%) cases were diagnosed as suspicious of malignancy and 2 (2.0%) cases were atypical. This may be due to technical error, nature of the lesions, in experience on the part of aspirator and cytologist.

In this study there was no false positive cytological diagnosis but there were 4 false negative cytological diagnosis giving a false negative rate of 6.45%. False negative rate in the literature ranges from 1.8 percent to 26.3 percent.[2] The false negative rate in this study is within the range of rate in the literature.

Factors that affect false negative rate include experience of those performing fine needle aspiration cytology and interpreting its result and the size and nature of the tumour[2]. Nature of the lesions may influence the false negative or inconclusive diagnosis. In patient with small tumors, masses seated deep in the breast or cell poor cancer (scirhhous carcinoma), aspirates may be cell free or cell poor and provide unsatisfactory material for evaluation may give false negative or suspicious diagnosis. Also aspirates from benign lesions may be cell poor.[10] High degree differentiation may be a cause of false negative aspirate.[11]

The number of inconclusive diagnosis, false negative results, unsatisfactory smears, which were higher in the early part of the study, can be reduced with increasing experience and practice. More over if the cytologist knows the clinical features diagnostic accuracy may improve.[5]

In patients who undergo a coordinated programme of clinical examination, mammography and fine needle aspiration biopsy, 99% of those with breast cancer will be detected but a few patients with breast cancer will still be missed. Clinicians must realize that a negative result of fine needle aspiration biopsy does not necessarily rule out the presence of cancer.

The cytological diagnosis is highly reliable when the diagnosis of cancer is made,[11] but negative or inconclusive cytological diagnosis should not be regarded as a definitive diagnosis if there is clinical suspicion of malignancy.[10] In any patient with a negative aspiration but with clinical suspicion or mammographic indication of cancer, surgical biopsy or definitive treatment should be performed.[10]

In this study out of 66 benign disease, (histologically diagnosed) 59 (89.4%) cases were below the age of 40 years. Out of 34 malignant lesions 31 (91.2%) were above the age of 40 years, i.e.- the malignant lesions are common in elderly patients. In this study it was found that common benign lesions were fibroadenoma (54.55%) and fibrocystic disease (36.36%). Common malignancy was invasive ductal carcinoma (76.47%).

Complication of FNAC is rare and usually not significant. There may be occasional pain, oedema, haematoma, infection.[2,10] Although dissemination of malignancy and implantation along the needle tract had been feared for a long time, but no study has substantially demonstrated it.[10] In this study minor discomfort and mild pain has been complained by some patients but significant hematoma, edema have not resulted, as firm pressure for 3-4 minutes has been maintained in every case. No patient developed infection after aspiration. So it can be said that FNAC is a safe procedure with minimum or no complication.

Due to high diagnostic accuracy, FNAC can be reliably used as a diagnostic tool in the management of breast lump. The sensitivity and specificity of FNAC is also acceptable and reasonable. It is less time consuming, can be performed in the outpatient department without any anesthesia. It is also a safe procedure with an accuracy exceeding that of other preoperative diagnostic methods.[5] Its complication is rare and usually not significant. It does not replace histology but improves the management of breast disease by giving reliable preoperative diagnosis.

For it to be fully and accurately utilized clinicians must understand the strength and weakness of the procedure also the technical difficulties involved in obtaining diagnostic material.

Limitation of the study:

The study was conducted in a small number of subjects in the teaching hospitals only for six months. All these factors limit generalization of the results.

Conclusion

Despite of availability of newer invasive techniques, FNAC still plays an important role in the diagnosis of clinically palpable breast lump in all age groups.

References

1. Aziz M,Ahmad N, Zahid J, Faizullah and Aziz M. comparison of FNAC and Open Biopsy in palpable breast lumps. JCPSP 2004; Vol. 14 (11): 654 656.
2. Abdullah P,MalikA,Zaheer N, Zahur-ur-Rehman, Mehmood A. Breast lump what they actually mean. JCPSP 2003;9:46-48.
3. Grady D, Hodgikns ML, Godsin WH 3rd. The lumpy breast west J med. 2005; 149:226-9
4. Cuschieri A. Giles GR. Moossa AR. Essential surgical practice 4th edition Vol-II oxford Buttarworth-Heinemann Ltd; 2002:61-93.

5. Muscovite M. Minimal breast cancer, radix. Radial north Am 2003;21:93 113.

6. Rosenburgh Al, Schwartz GF, FeigSA,PatchfskyAS. Clininically occult breast lesions: Localization and significance. Radiology 1997;162;167-170.

7. Teixidor HS, Wojtasek DA, Reiches EM, Santos CA, Minick R.V. Fine needle aspiration of Breast biopsy specimen: Correlation of Histologic and Cytologic findings Radiology July 2002; vol 184(1): 55-58.

8. Durfee GK, Cytologic techniques. In Ross LG (editor). diagnostic Cytology and its histopathologic basis. 2nd ed. Philadephia. JB Lippincott company 1998:597-628.

9. Furnival CM, Hocking MA, Hughes HE, Reid MM, Bough ust LH. Aspiration cytology in breast cancer: its relevance to diagnosis, Lancet 2001;6:446-449.

10. Ellis F. Needle biopsy in the clinical diagnosis of tumours. Br. J. Surgery 2000;34:240-261.

11. Wanbo HS, Felnen PS, Wilhelon MC, Covel JL, Binns RL. Fine needle aspiration cytology in lieu of open biopsy in the management of primary breast cancer. Ann surg. 2009, 569-579.

12. Eisenberg AJ, Hajdu, Wilhelmus J, Melamed MR, Kine D. Preoperative aspiration cytology of breast tumours. Actacytol. 2006; 30: 135-146.

13. Domugnez F, Riern JR, Tojos and Janco P. Fine needle aspiration of breast masses. An analysis of 1,398 patients in a community hospital ActaCytol Mar-Apr;41(2): 341-347.

14. Gupta RK, Dowle C, FNAC of tubular carcinoma of the breast in young woman. Diagn-Cytopathol 2001; 7:72-74.

15. Palombini L Fine-needle aspiration biopsies of breast masses: A critical analysis of 1956 cases in 8 years (1976-1984). *Cancer1988;61:2273 2277*.

Evaluation of Barium Esophagography in patients with Dysphagia

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Abstract:

Dysphagia is one of the common complaints in patients presenting for Barium Swallow Esophagography or endoscopic examination of the esophagus and is usually due to functional or structural abnormalities of the esophagus. A cross-sectional descriptive study was carried out on a total of 100 cases having dysphasia to evaluate barium Esophagography as a primary diagnostic tool, during January 2018 to December 2018. Barium esophagography successfully diagnosed carcinoma with a sensitivity of 60.00%, specificity of 90.00 %, positive Predictive Value of 72.00%, negative Predictive Value of 84.00 %. On the other hand, Sensitivity was 80.00%, Specificity was 94.44%, Positive Predictive Value was 61.54%, Negative Predictive Value was 97.70 % for diagnosing nonmalignant conditions, like Benign growth/ leomyoma/ submucosal growth without any mucosal involvement. High sensitivity and specificity was also found in Achalesia cardia , (100 %,100%) , strictures (100%,92.3%), hiatus hernia (83.3%,98.9%) etc. This study shows that barium swallow esophagography was an effective primary diagnostic tool for evaluating dysphagia.

Key words: Dysphagia, barium swallow Esophagography, upper GI endoscopy

Journal of Green Life Med. Col. 2019; 4(1): 34-38

Introduction

Dysphagia is one of the common complaints in patients presenting for Barium swallow Esophagography or endoscopic examination of the esophagus and is usually due to functional or structural abnormalities of the esophageal body or esophagogastric region.[1,2] Dysphagia is the perception of difficulty in swallowing or abnormal sensation of blockage of swallowed material.[3,4] There are multiple causes of dysphagia; which can be divided into broadly oropharyngeal causes and esophageal causes. In oropharyngeal causes, patients find difficulty initiating a swallow. Causes are mainly neurological causes like stroke, other causes of bulbar and pseudobulbar palsy.[5,6,7] On the other hand, esophageal causes are due to obstruction due to malignant or benign causes in esophagus. Physicians always use Barium Swallow Esophagography and upper GI endoscopy for investigating dysphagia. Endoscopy is useful in assessing a variety of pathologies of GI tract. Endoscopy is not only a diagnostic tool but also used for therapeutic procedure. It has indeed become a cost effective and reliable tool to modern medical science.[8] However, there is a huge debate over the preference by the doctors of one procedure over the other. The objective of this study was to evaluate Barium Esophagography as a primary diagnostic tool for evaluating dysphasia.

Methods

The study was an observational type of cross sectional study which was conducted on 100 patients with dysphagia presenting to the tertiary care hospital for a period of one year from January 2018 to December 2018. All cases were recruited by convenience sampling from radiology department who were sent for barium swallow study for evaluation of dysphagia. Informed consent was obtained from each patient.

Patients were clinically evaluated by detailed history and clinical examination. Patients were then sent for videofluoroscopy. The patients were explained regarding the procedure. The unit used was Listem (model-Rex-525RF). In the standing left lateral position, patients were given a few spoon thick paste (Max 100ml) of barium

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Received: 15, November 2018 **Accepted:** 21 December 2018

sulphate mixture (Viewgut 250% w/v 100 ml) mixed in water, for initial assessment of the upper aero digestive tract and to rule out any signs of aspiration. This was followed by a mouthful of barium (approx. 25 ml) and the patients were asked to hold the barium in the mouth till the fluoroscopic unit is ready for exposure. Continuous screening of the oropharynx, proximal and distal esophagus and gastroesophageal junction was done for free flow of the barium. Spot film in erect, AP, erect RAO, erect Rt/Lt and last a semi prone images were taken in left posterior oblique position. Both single contrast and mucosal relief contrast films were taken and any pathology noted was documented. The reporting of radiological investigations was done by experienced consultants. Endoscopic examination was done using endoscopy with 10% Xylocaine. Biopsy and relevant histopathological investigations were done wherever indicated.

Statistical analysis was performed with SPSS Version 24 statistic software package.

Results:

In the study 100 cases of dysphagia were included. The youngest patient was 14 years old and the eldest person was 75 years old. However, maximum number of cases were in the age group 30-60 years. Sixty one participants were male and 39 were female (M: F ratio 1.6:1).

Findings of Barium Swallow Study:

Barium swallow study revealed that out of 100 cases of dysphagia, 38 cases were benign and malignant case of neoplastic lesions. Among these 38 cases 25 cases are suspected to malignant growth (esophageal carcinoma) and rest 13 cases were benign lesions like leomyoma, compression from outside of esophagus. The researchers kept all compressive lesions under benign growth if there was no mucosal involvement as to evaluate barium study with diagnostic endoscopy. Achalasia cardia / bird-beak signs were found in 4 cases. Only in 2 cases of dysphagia barium Esophagography revealed impacted foreign body. Esophageal strictures were found in 15 cases, 4 of them were suffering from tracheoesophageal fistulas (all 4 of them suffering from corrosive burn) revealed by barium study, presbyoesophagus found in 5 cases. Hiatus hernia were found in 6 cases, Candidiasis in 5 cases, Schatzki's ring in 1 case, Esophageal webs in 2 cases and Zenker's diverticulum in 1 case. Out of 100 cases 20 cases were found to be normal in barium study.

Table-I

Summarization of Findings of barium swallow study in 100 cases of dysphagia

Barium swallow finding	Percentage
Esophageal Carcinoma	25
Benign growth/ leomyoma/ submucosal growth without any mucosal involvement	13
Achalasia cardia/ bird-beak sign	4
Foreign body	2
Stricture/ stenosis	15
Presbyesophagus	5
Hiatus hernia	6
Candidiasis	6
Schatzki's ring)	1
Esophageal webs	2
Zenker's diverticulum	1
Normal	20
Total	100

Endoscopy Findings:

In the present study, carcinoma of upper gastrointestinal tract was the most common diagnosis during endoscopy of upper GIT who presented with dysphagia. Thirty cases out of 100 cases were diagnosed as caricinoma of esophagus in endoscopy. Twenty eight cases were normal in endoscopic evaluation. Other findings were Leomyoma/ submucosal growth in 4 cases, esophageal polyps in 5 cases, compression outside esophagus in 1 case, Achalasia cardia in 4 case, spasm at lower end \Achalasia cardia in 4 cases, foreign body in 2 cases, stenosis/ Stricture in 8 cases, esophageal candidiasis in 10 cases, schatzki's ring in 1 case, Zenker's diverticulum in 1 case, Hiatus hernia in 6 cases.

Table-II

Endoscopy findings in 100 cases of dysphagia

Endoscopic finding	Percentage
Esophageal carcinoma	30
Leomyoma/ submucosal growth	4
Esophageal polyp	5
Compression outside esophagus	1
Spasm at lower end (Achalasia cardia)	4
Foreign body	2
Stenosis/Stricture	8
Esophageal candidiasis	10
Schatzki's ring)	1
Zenker's diverticulum	1
Hiatus hernia	6
Normal	28
Total	100

Evaluation of Barium Esophagography with Endoscopic Findings:

In this study 100 cases of dysphasia were evaluated by barium swallow esophagography followed by upper GI endoscopy. Here, upper GI endoscopy considered as gold standard for evaluating dysphasia. It was found that Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value for barium Esophagography were 60.0%, 90.0 %, 72.0%, and 84.0 % respectively, in diagnosing Esophageal Carcinoma. On the other hand, for diagnosing non malignant conditions like benign growth/ leomyoma/ submucosal growth without any mucosal involvement, it was found that Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value for barium Esophagography were 80.0%, 94.4%, 61.5%, and 97.7 % respectively.

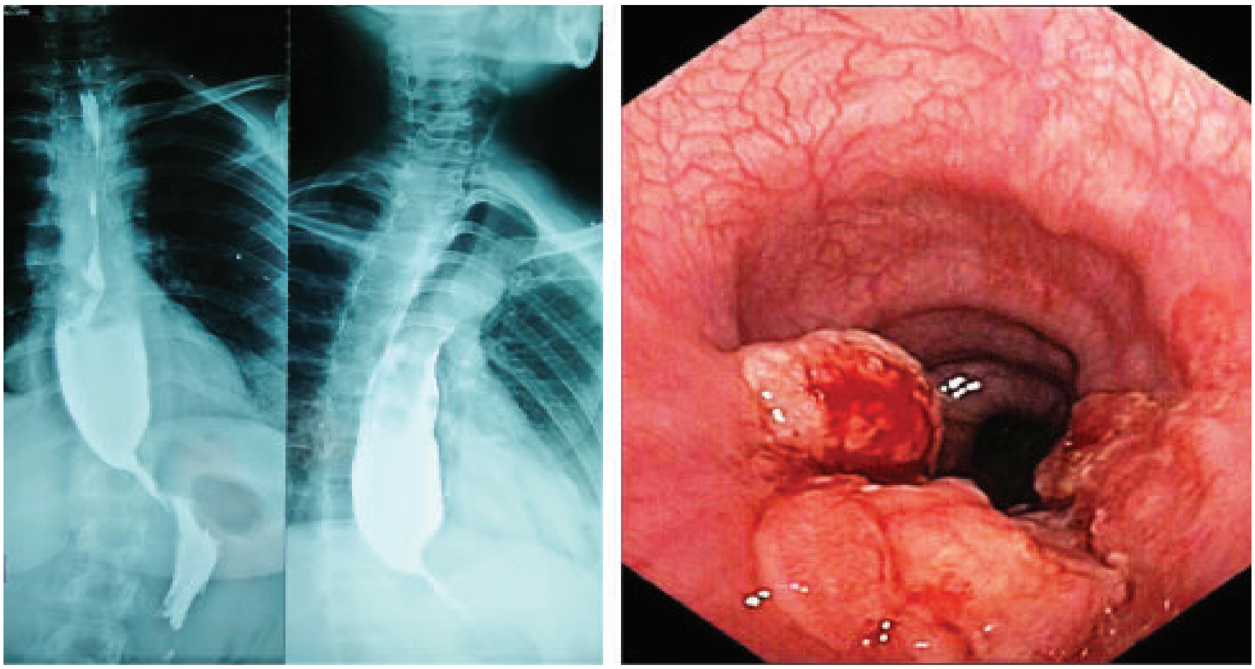

Fig 1: *Showing esophageal carcinoma in Barium study & endoscopy*

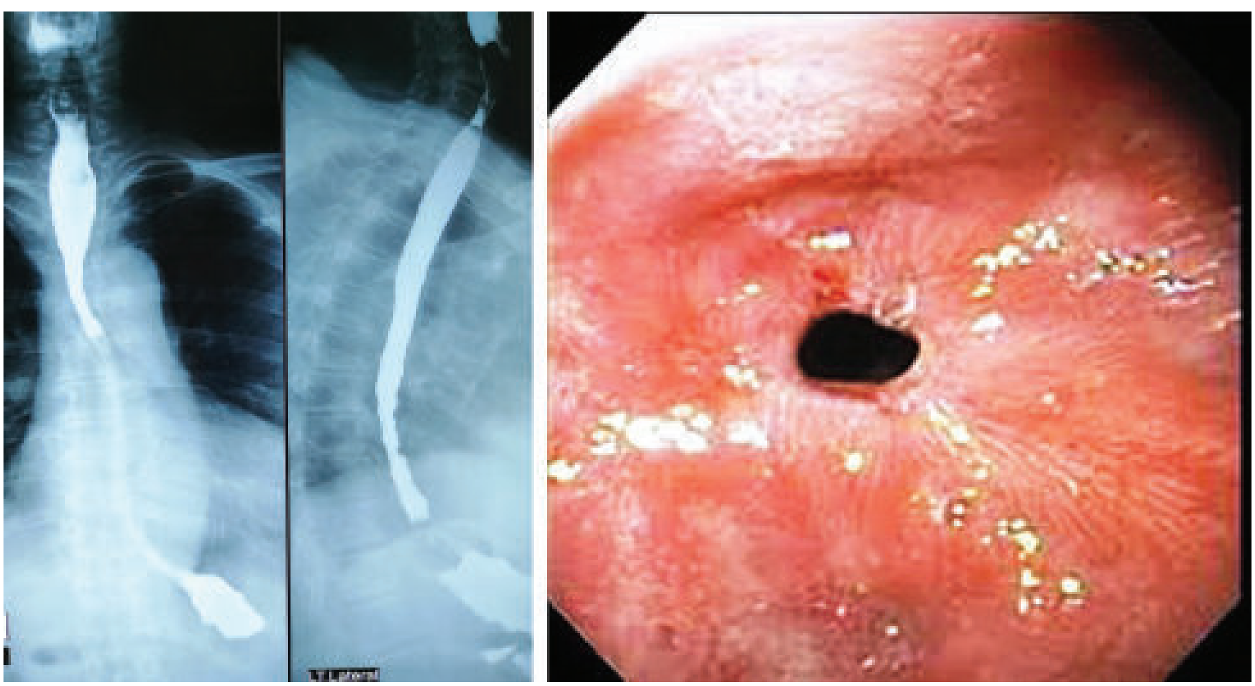

Fig.-2: *Showing esophageal stricture in Barium study & endoscopy*

Table-III

Evaluation of Barium Esophagography with Endoscopic Findings

		Endoscopic findings		Sensitivity=60.00%	Specificity=90.00 %	PPV=72.00%	NPV=84.00 %
Barium X-ray findings		Esophageal carcinoma in Endoscopy=30	No esophageal carcinoma in Endoscopy =70				
	Positive for Esophageal Carcinoma in barium study	18	7				
	Negative for Esophageal Carcinoma in barium study	12	63				
		Total=30	Total= 70				
		Endoscopic findings		Sensitivity=80.00%	Specificity=94.44 %	PPV=61.54%	NPV=97.70%
		Esophageal Benign growth/ leomyoma/ submucosal growth without any mucosal involvement in Endoscopy=10	No esophageal Benign growth/ leomyoma/ submucosal growth without any mucosal involvement in Endoscopy =90				
Barium X-ray findings	Positive for Benign growth/ leomyoma/ submucosal growth without any mucosal involvement	8	5				
	Negative for Benign growth/ leomyoma/ submucosal growth without any mucosal involvement	2	85				
		Total=10	Total= 90				
		Endoscopic findings		Sensitivity=100%	Specificity=100%	PPV=100%	NPV=100%
		Achalasia cardia in Endoscopy=4	No Achalasia cardia in Endoscopy =96				
Barium X-ray findings	Achalasia cardia /bird-beak sign in Ba. Xray	4	0				
	Negative for Achalasia cardia in Ba. Xray	0	96				
		Total=4	Total= 96				
		Endoscopic findings		Sensitivity=100 %	Specificity=92.39 %	PPV=53.33%	NPV=100%
		Stricture/ stenosis in Endoscopy=8	No Stricture/ stenosis in Endoscopy =92				
Barium X-ray findings	Positive Stricture/ stenosis in Ba. Xray	8	7				
	Negative Stricture/ stenosis in Ba. Xray	0	85				
		Total=8	Total= 92				
		Endoscopic findings		Sensitivity= cannot determine	Specificity=95.00 %	PPV=0%	NPV=100%
		Presbyesophagus in Endoscopy=0	No Presbyesophagus in Endoscopy =100				
Barium X-ray findings	Positive for Presbyesophagus in Ba. Xray	0	5				
	Negative for Presbyesophagus in Ba. Xray	0	95				
		Total=0	Total= 100				
		Endoscopic findings		Sensitivity=83.33%	Specificity=98.94%	PPV=83.33%	NPV=98.94 %
		Hiatus hernia =6	No Hiatus hernia in Endoscopy =94				
Barium X-ray findings	Hiatus hernia in Ba. Xray	5	1				
	Negative for Hiatus hernia in Ba. Xray	1	93				
		Total=6	Total= 94				
		Endoscopic findings		Sensitivity=50.00%	Specificity=98.89 %	PPV=83.33%	NPV=94.68 %
		Candidiasis in Endoscopy=10	No Candidiasis in Endoscopy =90				
Barium X-ray findings	Positive for Candidiasis in Ba. Xray	5	1				
	Negative for Candidiasis in Ba. Xray	5	89				
		Total=10	Total= 90				
		Endoscopic findings		Sensitivity=100%	Specificity=100%	PPV=100%	NPV=100%
		Schatzki's ring in Endoscopy=1	No Schatzki's ring in Endoscopy =99				
Barium X-ray findings	Schatzki's ring in Ba. Xray	1	0				
	Negative for Schatzki's ring	0	99				
		Total=1	Total= 99				
		Endoscopic findings		Sensitivity= cannot determine	Specificity=98%	PPV=0%	NPV=100%
		Esophageal webs in Endoscopy=0	No Esophageal webs in Endoscopy =100				
Barium X-ray findings	Esophageal webs in Ba. Xray	0	2				
	Negative for Esophageal webs in Ba. Xray	0	98				
		Total=1	Total= 100				
		Endoscopic findings		Sensitivity=100%	Specificity=100%	PPV=100%	NPV=100%
		Zenker's diverticulum in Endoscopy=1	No Zenker's diverticulum in Endoscopy =99				
Barium X-ray	Zenker's diverticulum in Ba. Xray	1	0				
	Negative for Zenker's diverticulum	0	99				

Discussion

In the present study, maximum number of cases were in the age group 30-60 years, the youngest patient was 14 years old and the eldest person was 75 years old. Sixty one of the participants were male and 39 were female (M: F ratio 1.56:1). Sachdeva et al carried out their study with patients in 41–59 years age group.[3] Tongper et al. reported maximum number of cases in 60 years and above age group. Seventy percent cases were males with male to female ratio of 2.33:1.[9] Kishve et al. observed 60.6% patients were males in their study.[10]

In our study we found 20% cases with normal findings in Barium Swallow study and 28% in normal in endoscopy. This patients were actually suffering from Globus sensation. Sachdeva et al. reported 47.2% with no significant finding.[3] Ray S et al. reported 16% normal cases in endoscopy having dysphagia.[11] These patients are theorized to have a sensory disorder in which they sense the normally passing bolus due to augmented afferent esophageal sensation.[12] Whether this is a disorder or peripheral or central sensory processing is unclear. These patients will “feel” the bolus going down. Conversely, they may still sense food or fluid in their esophagus for prolonged periods after the meal, although they may still eat and drink without difficulty.[8]

We found Sensitivity is 60.00% of barium Esophagography in diagnosing Esophageal Carcinoma. Sachdeva et al. reported in their study they found sensitivity of barium Esophagography in diagnosing Esophageal Carcinoma was 54.8%.[3] Ott DJ et al. mentioned radiological method can diagnose Esophageal Carcinoma with 100% sensitivity if performed properly.[1] We also found Barium Xray has sensitivity 100.00%, specificity 100.00 %, for diagnosing Achalasia cardia. Sachdeva et al. reported in their study they found sensitivity of barium Esophagography in diagnosing Achalasia cardia was 100%.[3]

In our study Stricture/ stenosis has been diagnosed by barium Esophagography with 100% sensitive. However, Sachdeva et al. reported that in their study they found sensitivity of barium Esophagography in diagnosing Stricture/ stenosis was75%.[3] In our study we found 5 cases of presbyesophagus out of 100 cases. However, in endoscopic findings all of them were found to be false positive for presbyesophagus. So, the sensitivity was 0%. Whereas, Sachdeva et al. found sensitivity of barium Esophagography was 100% in diagnosing presbyesophagus.

Conclusion

Both the barium swallow Esophagography and endoscopic evaluation of esophagus are good diagnostic tool for evaluation of dysphagia. However, both procedures have some merits and demerits. Barium swallow study is noninvasive, costs less, and may be more convenient for patient and it does not require sedation. It was found that Barium Esophagography has high sensitivity and specificity in diagnosing various conditions causing dysphagia. The barium swallow Esophagography should be the primary diagnostic tool for evaluation of dysphagia.

References

1 Ott DJ, Gelfand DW, Wu WC, Chen YM. Radiological Evaluation of Dysphagia. JAMA. 1986;256(19):2718–21.

2. Castell DO. Dysphagia. Gastroenterology. 1979; 76:1015-24.

3. Sachdeva K, Kaul V. Correlation of Radiological and Endoscopic Findings in Patients Presenting with Dysphagia. Indian J Otolaryngol Head Neck Surg. 2017;69(1):72-6.

4. Dysphagia:01. World Gastroenterology Organisation Practice Guidelines. 2007. http://almacen-gpc.dynalias.org/publico/Dysphagia %20WGO%202004%20Ingles.pdf

5. Ramlan S, Manohar S, Somayaji G. Videofluoroscopy versus upper G.I. endoscopy: A comparative study as a diagnostic tool in patients presenting with dysphagia. Arch Med Health Sci 2015;3:6-11.

6. Altman KW. Understanding dysphagia: A rapidly emerging problem. Otolaryngol Clin North Am 2013;46:13-6.

7. Schechter GL. Systemic causes of dysphagia in adults. Otolaryngol Clin North Am 1998;31:525-35.

8. Jalil AAA, Katzka DA, Castell DO. Approach to Patient with Dysphagia. The Am J Med. 2015;128:1138.e17-23.

9. Tongper D, Naloh M, Hajong R. Clinical and endoscopic study of dysphagia: a prospective cross-sectional study at a tertiary care centre at North-Eastern India. IOSR J Dent Med Sci (IOSR-JDMS). 2015; 14(2):9–11.

10. Kishve SP, Aarif SMM, Kishve PS Kalakoti P. Clinico-pathological profile of oesophageal dysphagia. Indian Med Gaz. 2011;145(10):379–83.

11. Ray S, Patel H, Kotecha J, Parmar H. Analytical Study of Upper Gastrointestinal Endoscopy - 200 cases. IAIM, 2016; 3(9): 98-100.

12. Clouse RE, Richter JE, Heading RC, Janssens J, Wilson JA. Functional esophageal disorders. Gut. 1999;45(2):II31-6.

ORIGINAL ARTICLE

Maternal Factors Affecting Hyperbilirubinaemia Within 28 Days of Life

CHOUDHURY S[1], DAS BK[2], IMTIAZ KS[3], HAYAT SS[4]

Abstract

Introduction: *Neonatal hyperbilirubinaemia resulting in clinical jaundice is a common problem among neonates, particularly during the first weeks of life. The situation of neonatal jaundice in developing countries is relatively same to that of developed countries. In Bangladesh 60% neonates found to be admitted in hospitals due to neonatal jaundice. Effect of hyperbilirubinaemia depends on its cause and the degree of elevation. This study was done to explore the maternal factors related to hyperbilirubinaemia within 28 days of life.*

Methods: *A cross sectional descriptive study was done in Dhaka Medical College and Hospital (DMC&H) among 120 neonates. All the neonates with hyperbilirubinaemia admitted in Paediatric ward having hyperbilirubinaemia from 01.05.11 to 15.05.11 were included. Sample was selected purposively and data were collected by check list using hospital records and face to face interview of mother of those neonates using pretested and revised semi- structured questionnaire.Data were presented as mean ± SD and number (percent) as appropriate. Both descriptive and inferential statistics were considered in data analysis. Statistical analyses were performed using SPSS Software.*

Results: *Among 120 neonates 59.2% were in the age group 2nd to 7th days and 57% were baby boy. Pre-maturity (gestational age <37 wks) was found among 73.3% cases of hyperbilirubinemia. Among the 120 mothers 93.4% were housewives. Of the total mothers 30% were adolescents (<20 years) and 27.5% young adults (20 to 25 years). Majority of them (90%) were Muslim and 56.7% had primary level of education. Normal vaginal delivery was performed in 59.2% of cases. Exclusive breast feeding was done by 43.33% mothers. Of the total cases 33 (27.5%) mothers provided history of jaundice of the previous baby. Maternal co-morbidity GDM was found in 19 (15.8%), hypothyroidism 8 (6.7%) and hepatitis B positive 11 (9.2%) cases. Among the neonates of the mothers with GDM 9.2% had serum bilirubin level above 20mg/dl. The distribution showed significant association (p<0.005).*

Conclusion: *Meticulous clinical practice may be needed to reduce the hyperbilirubinemia related neonatal morbidity and mortality.*

Keywords*: Neonates, Hyperbilirubinaemia, Pre term baby, Gestational Diabetes Mallitus*

Journal of Green Life Med. Col. 2019; 4(1): 39-43

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Received: 1 July 2018 **Accepted:** 24 December 2018

Introduction:

Neonatal hyperbilirubinaemia resulting in clinical jaundice is a common problem among neonates, particularly during the first weeks of life.[1] At birth and early days of life, serum bilirubin more than 7 mg/dl becomes visible as jaundice; rise in bilirubin in newborn remains undetectable for some time until bilirubin rises.[2] Effect of hyperbilirubinaemia depends on its cause and the degree of elevation.[3] The situation of neonatal jaundice in developing countries is relatively same to that of developed countries.[4] In Bangladesh 60% neonates found to be admitted in hospitals due to neonatal jaundice.[5] Major factors related to neonatal jaundice found to be mode of delivery, birth trauma, Gestational DM and breast feeding.[6] The mortality and morbidity picture of neonatal jaundice is markedly different in the developed and developing countries. Neonatal jaundice is a fairly common cause of morbidity in Bangladesh but little information is available on patterns of neonatal jaundice. Attitude and health care pattern of people and early detection of high risk groups are of paramount importance in preventing complications of neonatal jaundice.[4] Considering the realities of multiple risk factors of neonatal hyperbilirubinaemia, the study tried to determine the independent contribution of each risk factor. As a global problem, preventive and control strategies of hyperbilirubinaemia should be based on adequate knowledge and information regarding the incidence and risk factors, which are not available in the developing countries where the vast majority of births occur at home. Identifying infants at risk of severe hyperbilirubinaemia and early intervention may reduce the levels of morbidity and mortality associated with bilirubin encephalopathy.[7] This study was designed to access maternal risk factors related to the hyperbilirubinaemia among the neonates. The study findings may contribute to formulation of guidelines and strategies for better management and prevention of hyperbilirubinaemia among the vulnerable neonates.

Methods:

This descriptive cross-sectional study was conducted to explore the maternal factors related to hyperbilirubinaemia within 28 days of life. A total number of 120 neonates with jaundice admitted in the Department of Neonatology of Dhaka Medical College and Hospital were purposively selected in this study. Data were collected by face-to-face interview of mother of those neonates using pretested and revised semi- structured questionnaire and reviewing medical documents by check list. Maternal factors considered for study were gestational age, mode of delivery, breast-feeding, history of hyperbilirubinaemia in previous baby. Maternal co-morbidities considered were gestational diabetes, hypothyroidism and hepatitis B. Data regarding socio-demographic profile of mothers included age, religion, educational status and occupation. Data analysis was done by using the SPSS software (Version 18.0) for Windows and accordingly descriptive statistics frequency distribution, percentage, and mean ±SD was estimated. Inferential statistics included group comparisons by using Fisher's Exact Test. A two tailed p value less than 0.05 was considered statistical significant. Informed verbal consent was taken from the individual participant prior to inclusion in the study. They were also informed about their right to withdraw from the study at any stage or to restrict their data from analysis. Privacy was maintained during data collection and confidentiality of data was maintained.

Results:

Figure 1: Distribution of neonates on the basis of duration of age (days)

Of the total 120 neonates most of them that is 59.2% were in 2nd to 7th days. 15.0% and 25.8% on the basis of age 1st day and more than 7 days respectively. Age rage of the 120 neonates was 1-21 days. Mean ± SD age (days) of the neonates was 5.36 ± 4.43.

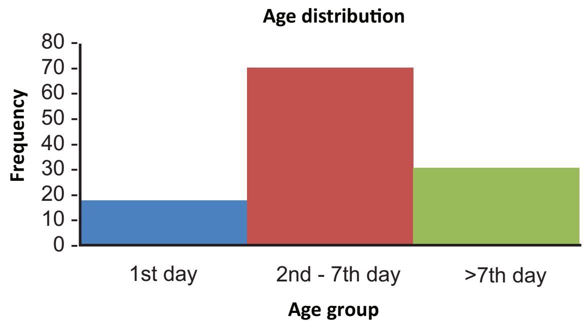

Fig.-2: *Distribution of the neonates by sex (n=120)*

Among 120 neonates majority of them 69 (57 %) were male & 51 (43 %) were female.

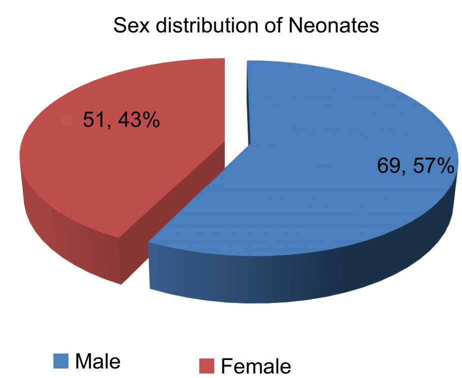

Fig.-3: *Distribution of the neonates by total bilirubin level (n=120)*

Fig shows that the total serum bilirubin level varied from 12 – 23.8 mg/dl. 33 (27.5%) neonates had serum bilirubin level 12-16.99 mg/dl, 76 (63.3%) neonates had serum bilirubin level 17-20 mg/dl and 11 (9.2%) neonates had serum bilirubin level >20 mg/dl.

Socio-demographic characteristics of mothers:

Table 1

Distribution of the neonates mother by age (n=120)

Age of mother	Frequency	Percentage (%)
< 20 yrs	36	30.0
20-25 yrs	33	27.5
25-30 yrs	21	17.5
> 30 yrs	30	25.0
Total	120	100.0

Among 120 neonates mother, 36 (30.0%) were < 20 years age, 33 (27.5%) were between 20-25 years age, 21 (17.5%) were between 25-30 years age, 30 (25.00%) were > 30 years age, mean age of mother was 23.725, SD ± 5.439. Minimum age of the mothers 17 yrs, and maximum age was 35 yrs.

Table-II

Socio-demographic profile of mothers of neonates

Attributes	Findings
Age of mother (mean±SD, yrs)	23.73 ±5.44
Religion	
Islam	90%
Hinduism	10%
Education	
Illiterate	20.8%
Primary	56.7%
Secondary	22.5%
Occupation of mother	
Job	6.6%
Housewife	93.4%

Mean (±SD) age (yrs) of the mothers was 23.73 ±5.44. Of the 120 mothers 30% were adolescent (< 20 yrs) and 27.5% young adult (20 to 25 years). Of the 120 mothers 93.4% were housewife and 90% belonged to Muslim faith and remaining 10% Hinduism. Of the all 56.7% had on the primary level of education and 20% were illiterate.

Maternal risk factors:

Table-III

Distribution of the neonates mother by gestational age during delivery (n=120)

Gestational Age	Frequency	Percentage (%)
<37 weeks	88	73.3
37 weeks or more	32	26.7
Total	120	100.0

88 (73.3%) of the respondents were <37 weeks gestational age during delivery and 32 (26.7 %) were 37 weeks or more, mean gestational age was 35.35 weeks, SD ± 2.286. Minimum gestational age was 31 wks and the maximum gestational age was 40 wks.

Table-IV

Distribution of the neonates mother by Mode of Delivery (n=120)

Mode of Delivery	Frequency	Percentage (%)
NVD	71	59.2
C/S	49	40.8
Total	120	100.0

Among 120 mother 71 (59.2%) of the them had NVD and 49 (40.8 %) had C/S.

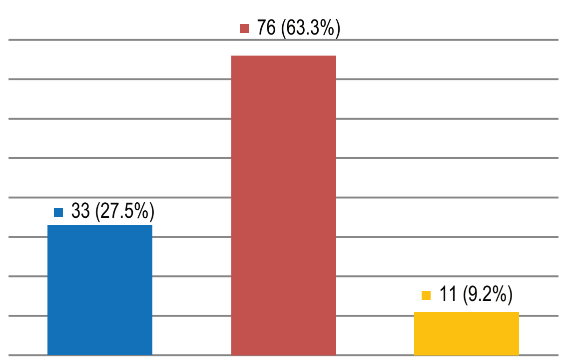

Fig.-4: *Distribution of the neonates mother by history of Exclusive breast-feeding (n=120)*

About half that is 52 (43.33%) of the respondents had history of Exclusive Breast Feeding, 25 (20.84%) had formula feeding and 43 (35.83 %) had mixed feeding.

Table-V

Distribution of the neonates mother by history of Jaundice of Previous Baby (n=120)

Jaundice of Previous Baby	Frequency	Percentage (%)
Yes	33	27.5
No	87	72.5
Total	120	100.0

Among 120 mother, 33 (27.5%) had history of Jaundice of previous baby and 87 (72.5 %) had no history of Jaundice of previous Baby.

Maternal co-morbidity related factors:

Table -VI

Distribution of the neonates mother by developing Gestational Diabetes during pregnancy (n=120)

Gestational Diabetes	Frequency	Percentage (%)
Yes	19	15.8
No	101	84.2
Total	120	100.0

Among 120 mother, 19 (15.8%) of the respondents developed Gestational Diabetes during pregnancy & 101 (84.2 %) did not.

Table-VII

Distribution of the neonates mother by having hypothyroidism (n=120)

Hypothyroidism	Frequency	Percent
Yes	8	6.7
No	112	93.3
Total	120	100.0

Only 8 (6.7%) neonates mother had hypothyroidism and 112 (93.3 %) had no hypothyroidism.

Table-VIII

Distribution of the neonates mother by having Hepatitis (n=120)

Hepatitis Positive	Frequency	Percent
Yes	11	9.2
No	109	90.8
Total	120	100.0

Only 11 (9.2%) neonates mother had hepatitis and 109 (90.8 %) had no hepatitis.

Table -IX

Distribution of Gestational diabetes of mother of the neonates in relation to total bilirubin level

GDM	Total bilirubin level (mg/dl)			Total
	12- 16.	*99*	*17-20*	*> 20*
	N	N	N	
Yes	0	8	11	19
No	33	68	0	101
Total	33	76	11	120

Regarding Gestational diabetes mellitus, whom mother had history of GDM (9.2%), had higher serum bilirubin (>20mg/dl) level. On the contrary, whom mother had no history of GDM, had bilirubin level below 20mg/dl. The association was highly significant (Fisher's Exact Test = 47.833, p value <0.005, CI = 95%)

Discussion:

Of the total 120 neonates most of them i.e. 59.2% were in 2^{nd} to 7^{th} days. Male (57 %) were predominant with ratio of male to female 1.3:1. This result coincided with that of 58.3% from a study conducted in BIEDEM, Dhaka, Bangladesh.[7]

Total serum bilirubin (TSB) ≥ 20 mg/dl occurred in 11 (9.2%) cases. This high level of TSB level was reported in 11.6% of neonates in other study, which was slightly more than this study.[7] The Impact of Breast-Feeding on Early Neonatal Jaundice 88 (73.3%) of the respondents were <37 weeks gestational age during delivery and 32 (26.7 %) were 37 weeks or more. Khatun et al. reported a similar observation in the neonatal unit of a Khulna Medical College Hospital, where 35% of newborns had jaundice and 31% of jaundiced infants were preterm.[14] This finding coincide with the study conducted in United Arab Emirates by Dawodu and his group.[10] The study reconfirmed the prematurity as prominent cause of hyperbilirubinemia in neonates.

In the present study 15.8% of mothers of neonates had gestational diabetes which is lower than the previous study in BIRDEM.[7] The very high (35%) proportion of mother with gestational diabetes may be explained by the fact of selection cases from BIRDEM, a tertiary care hospital of Diabetic Association of Bangladesh. However, the observation in the present study contradicts with those of 3.3% mother of hyperbilirubinemic neonates with

GDM.[10] This may possibly be explained by the inclusion small number of subjects in the present study. Further study involving large number of subjects and adaptation of more stringent inclusion criteria may circumvent the issue. It is important to note that significantly more neonates of GDM (9.2 %) than non-GDM mothers had high bilirubin level ($p<0.005$) which strengthened the notion that neonates of GDM mothers are more likely to suffer from neonatal hyperbilirubinemia.

In this study as like as others, it was detected that most patient had vaginal delivery (59.2%) and rest had caesarian section which is almost similar to that (63.6% vs 36.4% respectively) observed in Iran.[8] Relatively high proportion of in case of those delivered caesarian section might have been compounded by the fact that mother had normal vaginal delivery left hospital much earlier because those had caesarian section were delivered at 6th or 7th postoperative day and those neonates represented relatively higher number.

In the present study about half, 52 (43.33%) of the neonates with hyperbilirubinemia had history of exclusive breast feeding. Higher proportion of neonates with hyperbilirubinemia was also shown in Iran.[8] In United Arab Emirates breast-feeding was significantly associated with hyperbilirubinemia, even in the first three days of life.[13] Another study shows hyperbilirubenaemia occur in 82.7%, were breast-fed v 46.9% in the control group (P less than .0001).[15] However, breast fed babies often shows early onset of jaundice may be due to ineffective lactation in first few days after birth resulting in dehydration or in some instances use of water or glucose in water finally cause aggravation of jaundice.

Meticulous clinical practice may be needed to reduce the hyperbilirubinemia related neonatal morbidity and mortality. Further study is needed involving relatively large number of cases may be carried out to comprehensively comments on the issue and develop a guidelines to circumvent the issue.

References

1. Tikmani SS, Warraich HJ, Abbasi F, et al. Incidence of neonatal hyperbilirubinaemia: a population-based prospective study in Pakistan. Tropical Medicine and International Health 2010; 15: 502-507.
2. Agarwal KN, Pediatrics and neonatology, 2nd edition, New Delhi, CBS Publishers & distributors, 2008; 200-204.
3. Chapman RW, Collier JD, Hayes PC. Liver and biliary tract disease In Boon NA, Colledge NR, Walker BR, et al. Davidson's principles & practice of medicine, 20th edition, Edinburgh, Elsevier limited, 2006; 944-945.
4. Huang MS, Lin MC, Chen HH, et al. Risk factor analysis for late-onset neonatal hyperbilirubinaemia in Taiwanese infants. Pediatr Neonatol 2009; 50: 261-265.
5. Rasul CH, Hasan MA, Yasmin F. Outcome of neonatal hyperbilirubinaemia in a tertiary care hospital in Bangladesh. Malaysian J Med Sci 2010; 17 (2): 40-44.
6. Linn S, Schoenbaum SC, Monson RR, et al. Epidemiology of neonatal hyperbilirubinaemia. Pediatrics 1985; 75: 770-774.
7. Zabeen B, Nahar J, Nabi N, et al. Risk factors and outcome of neonatal jaundice in a tertiary hospital. Ibrahim Med. Coll. J. 2010; 4: 70-73.
8. Heydarian F, Majdi M. Severe neonatal hyperbilirubinaemia; causes and contributing factors leading to exchange transfusion at Ghaem Hosopital in Mashhad. Acta Medica Iranica 2010; 48: 399-402.
9. Wood B, Culley P, Roginski C, et al. Factors affecting neonatal jaundice. Archives of disease in childhood (BMJ) 1979; 54: 111-115.
10. Dawodu A, Qureshi MM, Moustfa IA, et al. Epidemiology of clinical hyperbilirubinaemia in Al Ain, United Arab Emirates. Ann Trop Paediatr 1998; 18: 93-9.
11. Manning D, Todd P, Maxwell M, et al. Prospective surveillance study of severe hyperbilirubinaemia in the newborn in the UK and Ireland. Arch Dis Child Fetal Neonatal Ed 2007; 92: 342-346.
12. Khan MR, Rahman ME, Essence of pediatrics, 3rd edition, Dhaka, 2008; 60-69.
13. Maisels MJ, Gifford K. Normal serum bilirubin levels in the newborn and the effect of breast-feeding. Pediatrics 1986; 78: 837-843.
14. Khatoon S, Islam MN. Neonatal Jaundice—Clinical profile of 140 cases. Bang J Child Health. 1993;17:158–163.
15. Lin Y Y, Tsao N P.The Impact of Breast-Feeding on Early Neonatal Jaundice.Clinical Neonatology 2008 Vol. 15 No.1

ORIGINAL ARTICLE

Effect of Smoking on Cardiac Autonomic Function in Cigarette Smokers Assessed by Frequency Domain Analysis of Heart Rate Variability

Ferdous M

Abstract

***Introduction:**Cigarette smoking is associated with various forms of acute cardiac events such as myocardial infarction, ventricular fibrillation etc.**Objective:**To assess HRV by frequency domain methods of HRV in healthy cigarette smokers in current regular healthy male cigarette smoker.*

***Methods:**This cross sectional study was conducted in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka from July 2011 to June2012. One hundred twenty apparently healthy male current regular cigarette smokers (Group B) aged 20-55 years were enrolled in the study group. Seventy apparently healthy male non smoker subjects (Group A) were taken ascontrol. Frequency domain measures of HRV were recorded by a RMS digital Polyrite. Statistical analysis was done by independent sample t test.*

***Results:** Resting Pulse, SBP, DBP, mean heart rate was significantly higher (p<0.001) in all smokers compared to control. Moreover, Low frequency (LF) component,LF power expressed in normalized unit (LFnu) and LF-HF ratio were significantly (P < 0.001) higher in all smokers and total power, high frequency (HF) component and HF power expressed in normalized unit (HFnu) were found significantly (P < 0.001) lower in all smokers compared to control.*

***Conclusion:**Increased sympathetic activity with concurrent suppression of cardiac vagal modulation occurs in regular cigarette smokers which are further exaggerated immediately after smoking a cigarette.*

***Keywords:** Cardiac autonomic nerve function, Heart rate variability, Nicotine.*

Journal of Green Life Med. Col. 2019; 4(1): 44-47

Introduction:

Cigarette smoking is one of the major modifiable risk factors for cardiovascular disease.[1, 2, 3]It is a major cause of atherosclerotic disease and is one of the major risk factor for coronary heart disease along with hypertension and lipid disorders. Cigarette smoking has a harmful effect on various parts of neurocardiovascular regulation system including afferent and efferent division of autonomic nervous system and central nervous system.[2]

Various researchers reported that cigarette smoking increases the risk of sudden death more than tenfold in men and five fold in women.[4]Several mechanisms are suggested in the literature for the harmful effects of smoking on cardiovascular events. Among them, smoking induced increased sympathetic activity is the most important one.[5]

Long term cigarette smoking is a major and independent risk factor for cardiovascular morbidity and mortality which has been highlighted by various investigators. Chronic smokers have a higher resting pulse rate and resting blood pressure compared to non-smokers indicating sympathetic hyperactivity in smokers.[5,6,7]

In various literatures, smoking induced increased sympathetic activity is the most important mechanism for harmful effects of smoking on cardiovascular events.[12]

Heart rate variability (HRV) is the most sensitive and quanitative marker for individual assessment of sympathetic and parasympathetic activity. In clinical practice, HRV has been shown to be a valuable non-invasive tool for the assessment of autonomic regulation

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Received: 27 November 2018 **Accepted:** 24 December 2018

of cardiovascular function. Frequency domain parameters have been recommended for HRV analysis with 5-minute recordings. Several frequency domain measures including total power, LF power,LF nu, HF power, HF nu, LF/HF ratio are commonly used for HRV measurement.[8]The vagal activity is a major contributor to the HF component. The LF component is a marker of sympathetic modulation, LF/HF ratio is considered as a mirror of sympatho-vagal balance.[9, 10]

Among the different form of tobacco consumption, cigarette smoking is the most common and frequent one and is a key variable in assessing the current and ongoing health risk of a population.Bangladesh was the first country to sign the WHO Framework Convention on Tobacco Control (FCTC). In 2005, Bangladesh enacted the Tobacco Control Act (TCA). Despite the enactment of the TCA, Bangladesh has experienced an alarming increase in cigarette use over last five year.[11]

The results of this study would provide guideline to clinician and to identify risk factors for autonomic dysregulation related cardiovascular disease. So the output of the study will act as a source of background information and guideline for clinician for better management of cardiovascular events in the smokers.

Methods:

This comparative analytical study was carried out in the department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka from July 2011 toJune 2012. A total number of 190 healthy male subjects aged 20-55 years were included for this study. Age, sex and BMI matched 70 non smokers were taken as control (Group A)and 120 healthy current regular male smokerswere selected as study group (Group B) from them. All of the smokers were selected from hospital staff, patient's attendants, motor vehicles drivers, medical colleagues from BSMMU campus and by personal contact. Study subjects were selected by following simple random sampling procedureand the protocol of this study was approved by central ethical review committee of BSMMU. Non smoker subjects were selected by personal contact. Smokers with history of coronary artery disease, active respiratory infection, diabetes mellitus, consumption of other tobacco products, thyroiddisorders, renal or hepatic dysfunction, taking drugs affecting autonomic nervous system or any psychiatric illness were excluded from the study. Selected subjects were informed about the risk and benefit and detail procedure of the study before enrollment and written consent was obtained from the willing volunteers.

For recording HRV parameters subjects wereprepared from the preceeding day of examination. They were advised to complete their meal by 9:00pm on the previous night, to remain free from any physical or mental stress, refrain from smoking atleast 12 hours before the study, they should not take any sedatives or drugs affecting nervous system and should have a sound sleep at night. The subjects were also asked to have light breakfast without tea or coffee on the next day. All examination were done in the autonomic nerve function test laboratory in the department of physiology, BSMMU between 9:00am to 2:00pm. In order to make Autonomic nerve function test, all subjects were done by frequency domain measures of HRV. The subject was lied on a bed in supine position and allowed to take rest for 15-20 minutes. 5 minutes baseline ECG recording for HRV was taken by polygraph. Frequency domain measures of HRV like total power, LF power, LF nu, HF power, HF nu, LF/HF ratio were analyzed. Data were expressed as mean and ±SEM. Comparison of data between groups were done by unpaired't' tests. P value<0.05 was taken as level of significance.

Results:

The anthropometric parameters & baseline characteristics of the subjects are presented in

Table I. The mean values of mean heart rate, pulse, SBP and DBP were significantly higher in group B than that of group A (Table I). Among the HRV parameters, Total power, HF power and HFnu were significantly reduced and LF power, LFnu and LF/HF ratio were significantly increased in all smokers compared to the control group(Table II).

TableI

Baseline characteristics in smokers & non smokers (n=190)

Parameters	Non smoker (n=70) Group A	Smoker (n=120) Group B
Age (years)	32.3±0.97 (22-52)	32.56±0.66 (22-49)
BMI(kg/m^2)	26.67±0.43 (19.88-34.24)	24.99±0.31 (17.51-33.78)
Pulse(beat/min)	72±0.58 (60-80)	78±0.5*** (62-99)
SBP(mm of hg)	112±0.98 (100-126)	121.63±1.01** (100-140)
DBP(mm of hg)	70±0.91 (60-85)	75.37±0.72*** (60-90)

Data expressed mean±SE. Figure in parenthesis indicate ranges. Data were analyzed by unpaired t test ***P<0.001 SBP=Systolic blood pressure, DBP=Diastolic blood pressure, BMI=Body mass index

Table II

Frequencydomain measures of HRV in different groups (n=190)

Parameters	Non smoker (n=70) Group A	Smoker (n=120) Group B
Total power (ms^2)	3347.97±101.03	2774.45±142.85**
	(897.4-5782.7)	(1034.5-5123.4)
LF power(ms^2)	671.70±38.36	870.23±51.97***
	(300.63-1342.35)	(232.8-1907.8)
HF power(ms^2)	584.46±29.42	452.05±31.99***
	(203.71-1195.28)	(127.3-1082.5)
LF norm(nu)	54.4±0.96	72.99±1.25***
	(42.5-79.2)	(58.4-88.9)
HF norm(nu)	37.95±0.88	31.28±1.74***
	(22.3-58.7)	(12.5-52.4)
LF/HF ratio	1.22±0.05	2.13±0.12***
	(0.58-2.63)	(0.95-5.08)

Data were expressed as Mean ± SE. Figures in parentheses indicate ranges. Statistical analysis were done by Independent sample t-test

Group A: Apparently healthy non smoker (control)

Group B: Apparently healthy male smoker

*** = p<0.001, ns = non significant (p>0.05), ** = p<0.01

Discussion:

The present study evaluated cardiac autonomic nerve function activity in apparently healthy current regular male cigarette smokers by analysis of frequency domain measures of heart rate variability and compared in age and BMI matched apparently healthy non smoker adults.

The current study evaluated cardiac autonomic status by analyzing HRV and CV risks in apparently healthycurrent regular male cigarettesmokers. Decreased vagal tone and HRV suggested by lower HF, HFnu, TP and increased cardiac sympathetic drive reflected by higher LF, LF nu and significantly higher LF/HF ratio were noted in allsmokers.[13]

In the present study, mean HR, SBP and DBP were found significantly higher in all smokers than non smokers. Similar results were also found by Andrikopoulas et al. and Cagirci et al. in heavy smokers.[3, 14]

In the present study, LF power, LF nu and LF/HF ratio were significantly higher in all groups of smokers compared to non smokers which were similar to some researchers.[1,2,15]Also, some researchers compared percentile change of LF power and LF nu between habitual and past smoker which were increased in both groups.[14]

In the present study, total power, HF power and HF nu were significantly lower in all smokers compared to non smoker. Similar trend was reported by some investigators in heavy smoker.[2,3] On the contrary, some researchers found decreased HF power which was not significant.[1]

Increased sympathetic activity or decreased vagal modulation and impaired sympathovagal balance assessed by heart rate variability analysis in cigarette smoker has been associated with an increased risk of coronary heart disease and arrhythmia and sudden cardiac death.[2,3,5]

The reduced cardiac vagal modulaion and the simultaneous sympathetic hyperactivity in the smokers of this study may be the effect of consumption of nicotine and other substances contained in cigarette smoke and this view is shared by several groups of researchers.[1, 2, 3, 14, 15, 16] This cardiac sympathoexcitatory effect of nicotine is the major reason for various hazardous effects of smoking.[2, 3, 5]

Nicotine is considered as a major component of cigarette smoke & it stimulates nicotinic receptors of autonomic ganglia increasing the post ganglionic sympathetic fibers leading to increase catecholamine release, muscle sympathetic nerve excitation and increase peripheral chemoreceptor sensitivity.[18,19,20]

It has been suggested that short term changes observed in LF/HF & HF immediately after smoking are due to increased release and/or a reduced clearance of catecholamine at neuroeffector junctions.[2,17]

Conclusion:

Increased sympathetic activity with concomitant suppression of cardiac vagal modulation occurs in regular cigarette smokers which are further exaggerated immediately after smoking a cigarette. In addition to the individual changes in sympathetic or parasympathetic function, their balance at any instant is also affected in smokers. Above all, the results of this study would provide guideline to clinician and to identify risk factors for autonomic dysregulation related cardiovascular disease. So the output of the study will act as a source of background information and guideline for clinician for better management of cardiovascular events in the smokers

References:

1. Barutcu I, Esen AM, Kaya D, Turkmen M, Karakaya O, Melek M, *et al*. Cigarette smoking and heart rate variability: Dynamic influence of parasympathetic and sympathetic maneuvers. Ann Noninvasive Electrocardiol 2005;10:324-9.
2. Alyan O, Kacmaz F, Ozdemir O, Maden O, Topaloglu S, Ozbakir C, Metin F, Karadede A, Iikay E. Effects of cigarette smoking on heart rate variability and plasma N-terminal pro-B type natriuretic peptide in healthy subjects: is there the relationship between both markers? Ann Noninvasive Electrocardiol.2008;13(2): 137-144

3. Cagirci G, CAY S, Karakurt O, Eryasar N, Kaya V, Canga A, Yesilay AB, Kilic H, Topaloglu S, Aras D, Demir AD, Akdemir R. Influence of heavy cigarette smoking on heart rate variability and heart rate turbulence parameters. Ann Noninvasive Electrocardiol.2009;14(4):327-332

4. Kannel WB, Mcgee DL, Castelli WP. Latest perspectives on cigarette smoking and cardiovascular disease: the Framingham study. J cardiac rehabil.1984;4: 267-277

4. HeringD, Somers VK, Kara T, Kucharska W, Jurak P, Bieniaszewski L, Narkiewicz K. Symapthetic neural responses to smoking are age dependent.Journal of hypertension. 2006;24: 691-695

6. Cryer PE, Haymond MW, Santiago JV, Shah SD.(1976) Norepinephrine and epinephrine release and adrenergic mediation of smoking associated and hemodynamic and metabolic events. N Engl J Med. 295(11):573-577

7. Gidding SS, Xie X, Lui K, Manolio T, Flack JM, Gardin JM. (1995) Cardiac function in smokers and non-smokers: The CARDIA Study.J Am Coll Cardiol.26:211-216

8. Task Force of the European society of cardiology and the North American society of pacing and electrophysiology. Heart Rate Variability.Standards of measurement, physiological interpretation and clinical use.Circulation.1996; 93: 1043-1065.

9. Akselrod S, Gordon D, Ubel FA, Shanon DC, Barger AC, Cohen RJ.(1981) Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat to beat cardiovascular control.Science.213:220-222

10. Pomeranz B, Macaulay RJ, Caudill MA, Kutz I, Adam D, Gordon D, Kilborn KM, Barger AC, Shanon DC, Cohen RJ. (1985) Assessment of autonomic function in humans by heart rate spectral analysis. Am J Physiol Heart Circ Physiol. 248: H151-H153

11. Anti-tobacco 2009. [Internet] Bangladesh tobacco facts. [cited on November2102]. Availablefrom:http://anti-tobacco bd.net/index.php?option=com_content& view

12. Hering D, Somers VK, Kara T, Kucharska W, Jurak P, Bieniaszewski L, Narkiewicz K. Symapthetic neural responses to smoking are age dependent.(2006).Journal of hypertension. 24:691-695

13. Lutfi MF, Sukkar MY. (2011) The effect of gender on heart rate variability in asthmatic and normal healthy adults. International Journal of health sciences.5:146-154

14. Andrikopoulos GK, Dilaveris PE, Richter DJ, Gialafos EJ, Lazaki EA, Avgeropoulou CK. Influence of cigarette smoking on heart rate variability in young healthy subjects. A.N.E.1999;4(2). 204-211

15. Lucini,D, Bertocchi F, Malliani A, Pagani M. A controlled study of the autonomic changes produced by habitual cigarette smoking in healthy subjects. Cardiovascular research.1996;31:633-639

16. Eryonucu B, Bilge M, Guler N, Uzun K, Gencer M.(2000) Effects of cigarette smoking on the circadian rhythm of heart rate variability.Acta cardiol.55(5):301-5

17. Grassi G, Seravalle G, Calhoun DA, Bolla GB, Giannattaiso C, Marabini M, Bo AD, Mancia G.(1994) Mechanisms responsible for sympathetic activation by cigarette smoking in humans.Circulation.90:248-253

18. Adamopoulas D, Borne PV, Argacha JF.(2008) New insights into the sympathetic endothelial and coronary effects of nicotine. Clinexppharmacol physiol. 35:458-463

19. Katzung GB. (2009) Basic and Clinical Pharmacology .11th ed. New York: The McGraw-Hill Companies. P94-108

20. Seth SD, Seth V. (2009) Text Book of Pharmacology. 3rd ed. New Delhi: Reed Elsevier India Private Limited: pII33-II36

Irreversible pulpitis- An update

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Abstract:

Irreversible pulpitis is a stage of pulpal inflammation and degeneration marked by clinical symptoms resulting from significant injury to the pulpal tissues. The damage may have resulted from current irritation from caries or the cumulative effects of past injury from restorative procedures, trauma, or other insults. In either case, the magnitude of the damage and the ability of the pulp to repair itself depend on physiologic changes within the pulp and the health of the tissue at the time of injury Similar to reversible pulpal inflammation, the term irreversible pulpitis is based on history and clinical signs that do not necessarily reflect the severity of damage in the pulpal tissues accurately. Although the origin is similar to that of reversible pulpal inflammation, an irreversibly inflamed pulp is not expected to improve, even if the causative agent is removed. Therefore, when irreversible pulpitis is diagnosed, the treatment of choice is root canal therapy or extraction. This review article briefly describes on how to diagnose the pulpal condition, etiology sign symptoms and what is its treatment option.

Key Words: Irreversible pulpitis, reversible pulpitis, differential diagnosis, treatment, prevention.

Journal of Green Life Med. Col. 2019; 4(1): 48-52

Introduction:

The dental pulp is a connective tissue consisting of nerves, blood vessels, ground substrates, interstitial fluid, odontobasts, fibroblasts, and other cellular components, historically, there have been a variety of diagnostic classification systems advocated for determining endodontic disease.[1]

Inside the innermost part of each tooth is an area called the pulp. The pulp contains the blood, supply, and nerves for the tooth. Pulpitis is a condition that causes painful inflammation of the pulp. It can occur in one or more teeth, and is caused by bacteria that invade the tooth's pulp, causing it to swell.

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Received: 12 November 2018 **Accepted:** 21 December 2018

There are two forms of pulpities: reversible and irreversible. Reversible pulpitis refer to instances where the inflammation is mild and the tooth pulp remains healthy enough to save. Irreversible pulpities occurs when inflammation and other symptoms, such as pain, are severe, and the pulp cannot be saved. Irreversible pulpities may lead to a type of infection called periapical abscess. This infection develops at the root of the tooth, where it causes a pocket of pus to form. If not treated, this infection can spread to other parts of the body, such as the sinuses, jaw, or brain.[2]

Irreversible pulpitis at an advanced stage of development is characterized by acute and intense pain that is difficult to control with painkillers.[14,15]; hence the patient needs urgent endodontic help. In such cases a quick and precise diagnosis is necessary, as well as immediate action to treat the symptoms.[16,17,18,19]

Etiology

According to the Marck Manual, deep cavities close to the pulp, trauma to a tooth, crown preparations, repeated invasive procedures, a **creacked tooth** and grinding and clenching your teeth are all scenarios that can inflame the blood vessels in your pulp. These vessels then press on the nerves, causing discomfort. If the pain occurs with temperature extremes but goes away quickly, you may have a reversible condition. But if the pain is intense, lingers after temperature changes, occurs unprompted or is scattered, making it hard to determine ther exact location, it may have irreversible pulpitis.[3]

Pulpitis may be caused by dental caries that penetrate through the enamel and dentin to reach the pulp, or it may be a result of trauma, such as physical abuse of the tooth or thermal insults, including overheating from insufficiently cooled dental drills and use of dental curing lights More often it is from physical trauma rather than dental treatments.

Inflammation is commonly associated with a bacterial infection but can also be due to other insults such as repetitive trauma or in rare cases periodontitis. In the case of penetrating decay, the pulp chamber is no longer sealed off from the environment of the oral cavity.[8]

When the pulp becomes inflamed, pressure begins to build up in the pulp cavity, exerting pressure on the nerve of the tooth and the surrounding tissues. Pressure from inflammation can cause mild to extreme pain, depending upon the severity of the inflammation and the body's response. Unlike other parts of the body where pressure can dissipate through the surrounding soft tissues, the pulp cavity is very different. It is surrounded by dentin, a hard tissue that does not allow for pressure dissipation, so increased blood flow, a hallmark of inflammation, will cause pain.[9]

Pulpitis can often create so much pressure on the tooth nerve that the individual will have trouble locating the source of the pain, confusing it with neighboring teeth, called referred pain. The pulp cavity inherently provides the body with an immune system response challenge, which makes it very difficult for a bacterial infection to be eliminated.[10]

If the teeth are denervated, this can lead to irreversible pulpitis, depending on the area, rate of infection, and length of injury. This is why people who have lost their dental innervation have a reduced healing ability and increased rate of tooth injury. Thus, as people age, their gradual loss of innervation leads to pulpitis.[11]

Symptoms:

Both types of pulpitis cause pain, though the pain caused by reversible pulpitis may be milder and occur only while eating. The pain associated with irreversible pulpitis may be more severe, and occur throughout the day and night.

Other symptoms of both forms of pulpitis include:

- inflammation
- sensitivity to hot and cold food
- sensitivity to very sweet food

Irreversible pulpitis may include additional symptoms of infection, such as:

- running a fever
- swollen lymph nodes
- bad breath

bad taste in the mouth

In a healthy tooth, the enamel and dentin layers protect the pulp from infection. Pulpitis occurs when these protective layers are compromised, allowing bacteria to get into the pulp, causing swelling. The pulp remains trapped inside the tooth's walls, so the swelling causes pressure and pain, as well as infection.

The enamel and dentin layers can become damaged by several conditions, including:

- cavities or tooth decay, which causes erosion to the tooth
- injury, such as an impact to the tooth
- having a fractured tooth, which exposes the pulp
- repetitive trauma caused by dental issues, such as jaw misalignment or bruxism (tooth grinding).[4]

Irreversible pulpitis:

Symptomatic Irreversible Pulpitis is based on subjective and objective findings that the vital inflamed pulp is incapable of healing and that root canal treatment is indicated. Characteristics may include sharp pain upon thermal stimulus, lingering pain (often 30 seconds or longer after stimulus removal), spontaneity (unprovoked pain) and referred pain. Sometimes the pain may be accentuated by postural changes such as lying down or bending over and over-the-counter analgesics are typically ineffective. Teeth with symptomatic irreversible pulpitis may be difficult to diagnose because the inflammation has not yet reached the periapical tissues, thus resulting in no pain or discomfort to percussion. In such cases, dental history and thermal testing are the primary tools for assessing pulpal status.[5]

Asymptomatic Irreversible Pulpitis is a clinical diagnosis based on subjective and objective findings indicating that the vital inflamed pulp is incapable of healing and that root canal treatment is indicated. These cases have no clinical symptoms and usually respond normally to thermal testing but may have had trauma or deep caries that would likely result in exposure following removal.[5]

Fig : *Following the placement of a full gold crown on the maxillary right second molar, the patient complained of sensitivity to both hot and cold liquids; now the discomfort is spontaneous. Upon application of Endo-Ice on this tooth, the patient experienced pain and upon removal of the stimulus, the discomfort lingered for 12 seconds. Responses to both percussion and palpation were normal; radiographically, there was no evidence of osseous changes. Diagnosis: Symptomatic irreversible pulpitis normal apical tissues.*

Symptoms:

1. The pain often continues when the cause has been removed, and it may come and go spontaneously, without apparent cause.
2. The patient may describe the pain as sharp piercing, or shooting, and it is generally severe.
3. The patient may also state that bending over or lying down, that is change of position, exacerbates the pain; changes in intrapulpal pressure may be the cause.
4. When no outlet is present, whether because of a covering of decay or a filling or because of food packed into a small exposure in the dentin, pain can be most intense.
5. The patient may also have pain referred to adjacent teeth, to the temple or sinuses when an upper posterior tooth is involved, or to the ear when a lower posterior tooth is affected.
6. Pain is increased by heat and is sometimes relieved by cold, although continued cold may intensify the pain.
7. Apical periodontitis is absent, except in the later stages, when inflammation or infection extends to the periodontal ligament.

Diagnosis:

1. Inspection generally discloses a deep cavity extending to the pulp.
2. The surface of the pulp is eroded. An odor of decomposition is frequently present in this area.
3. Probing into the area is not painful to the patient until the deeper areas of the pulp are reached.
4. A radiograph may also show exposure of the pulp.

Differential diagnosis:

1. One must distinguish between reversible and irreversible pulpitis.

Reversible Pulpitis	Irreversibile Pulpitis
Pain lasts for a moment.	Pain is severe & last longer.
Stimulus requires to elicit the pain	Spontaneous pain
Sharp pain	• In later stages – Boring, Gnawing or throbbing • Sharp, piercing or shooting
EPT uses less current than on a controlled tooth	Asymptomatic stage : Early Symptomatic stage : LessMore : current is required than a control tooth.

2. In later stage of irreversible pulpits, the symptoms may simulate those of an acute alveolar abscess.
3. Abscess has following symptoms which helps in differentiating it from pulpitis:
   - Tenderness on percussion.
   - Tenderness on palpation.
   - Swelling.
   - Mobility.
   - Lack of response to pulp vitality testing

Before making a diagnosis, it is necessary to determine whether dental pain is of nonodontogenic or odonto-genic origin. Among the nonodontogenic pathological conditions, referred pain presents more difficulties in differential diagnosis,[12] while majority of the nonodonto-genic diseases (eg,temporomandibular joint syndrome, pericoronitis, mouth ulcers, sinusitis, sialolithiasis) do not present difficulties in differential diagnosis.

Clinically, it is possible to determine the degree of pulp pathology by asking the patient about the history of pain of the involved tooth. This history adds a useful dimension in the diagnosis for the clinician as to whether the pulpitis is reversible or irreversible.[12,13]

Treatment:

1. Complete removal of pulp / Pulpectomy.
2. In posterior teeth – removal of coronal pulp / Pulpotomy should be performed as an emergency procedure.

Prognosis:

The prognosis of the tooth is favorable if the pulp is removed and if the tooth undergoes proper endodontic therapy and restoration.

Special considerations:

When irreversible pulpitis is present, the teeth that are most difficult to anesthetize are mandibular molars, followed by mandibular premolars, the maxillary molars and premolars, and the mandibular anterior teeth. The fewest problem arise in the maxillary anterior teeth. In some teeth, irreversible pulpitis is the condition in the apical portion of the canals; the tissue in the chamber is necrotic and does not respond to pulp testing. The pulp chamber can be entered easily, but when attempts are made to place a file to length, severe pain results. In such cases supplemental injections are of great help.[6]

Risk Factors :

Anything that increases the risk of tooth decay, such as living in an area without fluoridated water or having certain medical conditions, such as diabetes, may increase the risk of pulpitis.

Children and older adults may also be at increased risk, but this is largely determined by quality of dental care and oral hygiene habits.

Lifestyle habits may also increase the risk for pulpitis, including:

- poor oral hygiene habits, such as not brushing teeth after meals and not seeing a dentist for regular checkups
- eating a diet high in sugar, or consuming foods and drinks which promote tooth decay, such as refined carbohydrates
- having a profession or hobby that increases your risk of impact to the mouth, such as boxing or hockey
- chronic bruxism[7]

Pain management :

Pain management, both before and after treatment, is usually done with nonsteroidal anti-inflammatory (NSAIDs) drugs. These provide relief from pain and inflammation.[7]

In reversible pulpitis, pulp vitality can be maintained if the tooth is treated, usually by caries removal, and then restored. Generally, no treatment is necessary, other than the healing period for the pulp. Irreversible pulpitis and its sequelae require endodontic therapy, nevertheless, preoperative administration of ibuprofen one hour before local anesthesia injection is an effective method for achieving a deep anesthesia during endodontic treatment of patients with irreversible pul pitis.[20] Moreover, anti-inflammatory treatment improves the postoperative pain.[21]

Prevention

Pulpitis can often be avoided by practicing good oral hygiene and visiting a dentist regularly. Reducing or eliminating sweets, such as sugary colas, cake, and candy, can also help.

If you have bruxism, a tooth guard may help protect your teeth.[7]

References :

1. Pulpitis: A review, Dr. Syed Gufaran Ali, IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), Volume 14, Issure 8 Ver.VI(Aug.2015), PP92-97.
2. What is pulpitis? Written by Corey Whelan, https://www.healthline.com/health/pulpitis

3. Reversible and Irreversible pulpitis : Causes And Treatment by Donna Pleis, https://www.colgate.com/enus/oralhealth/ conditions/mouthsores-and-infections-and-reversible-and - irreversible pulpitis-causes- and - treatment.

4. Pulpitis: A review, Dr. Syed Gufaran Ali, IOSR Journal of Dental and Medical Sciences (IOSR- JDMS), e-ISSN: 2279-0853, p-ISSN:2279-0861. Volume 14, Issue 8 Ver.VI(Aug.2015), PP92-97.https://www.healthline.com/ health/pulpitis

5. Dabuleanu M. Pulpitis reversible/irreversible. J Can Dent Assoc 2013;79:90-94

6. Al Reader, Nusstein J, Hargreaves KM. Local anesthesia in ndodontics. Pathways of the pulp. 9th ed. St. Louis, Missouri: Mosby/Elsevier 2006

7. Pulpitis: A review, Dr. Syed Gufaran Ali, IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), e-ISSN: 2279-0853, p-ISSN:2279-0861. Volume 14, Issue 8 Ver.VI(Aug.2015), PP92-97. https://www.healthline.com/ health/pulpitis

8. Kakehashi S, Stanley HR, Fitzgerald RJ. The effects of surgical exposures of dental pulps ingerm-free and conventional laboratory rats. Oral Surg Oral Med Oral Pathol 1965:20:340-9

9. Hargreaves, KM. Goodis, HE. Seltzer and Bender's Dental Pulp. Quintessence, 2002.

10. Torabinejad, M. Walton, RE. Endodontics: Principles and Practice. 4th Edition. Elsevier Health Sciences, March 2008.

11. Byers, M. R., Suzuki, H. and Maeda, T. (2003), Dental neuroplasticity, neuro-pulpal interactions, and nerve regeneration. Microsc. Res. Tech., 60: 503–515.

12. Diagnosis and managing pulpitis: reversible or irreversible? Adriano Piattelli, MD, DDS*, Tonino Traini, DDS,PhD, Bender IB. Pulpal pain diagnosis—A review. J Endod 2000;26(3): 175-179

13. Diagnosis and managing pulpitis: reversible or irreversible? Adriano Piattelli, MD, DDS*, Tonino Traini, DDS, PhD, Bender IB. Reversible and irreversible painful pulpitis: Diagnosis and treatment. Aust Endod J 2000;26(1):10-14

14. Emergency treatment of irreversible pulpitis : Elka Radeva, Department of Conservative Dentistry,Faculty of Dental Medicine, Medical University - Sofia, Bulgaria. Journal of IMAB- Annual Proceeding (Scientific Papers) 2008, Book-2. Claffey E. et al. Anesthetic efficacy of articaine for inferior alveolar nerve blocks in patients with irreversible pulpitis. J.Endod. 2004, Aug; 30(8):568-71

15. Emergency treatment of irreversible pulpitis : Elka Radeva, Department of Conservative Dentistry,Faculty of Dental Medicine, Medical University - Sofia, Bulgaria. Journal of IMAB- Annual Proceeding (Scientific Papers) 2008, Book-2. Mc Dougal et al. Succes of an alter-native for interim management of irreversible pulpitis. J. Am. Dent.Assoc. 2004, Dec;135 (12): 1707-12

16. Emergency treatment of irreversible pulpitis : Elka Radeva, Department of Conservative Dentistry,Faculty of Dental Medicine, Medical University - Sofia, Bulgaria. Journal of IMAB- Annual Proceeding (Scientific Papers) 2008, Book-2. Bender IB. Reversible and irreversible painful pulpitides: diagnosis and treatment.Aust. Endod. J., 2000, Apr; 26(1):10-4

17. Emergency treatment of irreversible pulpitis : Elka Radeva, Department of Conservative Dentistry,Faculty of Dental Medicine, Medical University - Sofia, Bulgaria. Journal of IMAB- Annual Proceeding (Scientific Papers) 2008, Book-2. Lopez-Marcos JF. Aetiology, classi-fication and pathogenesis of pulp and periapical disease. Med Oral Pathol Oral Cir Bucal. 2004, 9, 58-62

18. Emergency treatment of irreversible pulpitis : Elka Radeva, Department of Conservative Dentistry,Faculty of Dental Medicine, Medical University - Sofia, Bulgaria. Journal of IMAB- Annual Proceeding (Scientific Papers) 2008, Book-2. Ranali et al. Perception of Pain and Anxiety during Emergency Dental Care.Dent. Anesth. Res., 2006, March, 78-9

19. Emergency treatment of irreversible pulpitis : Elka Radeva, Department of Conservative Dentistry,Faculty of Dental Medicine, Medical University - Sofia, Bulgaria. Journal of IMAB- Annual Proceeding (Scientific Papers) 2008, Book-2. Vohra F. et al. An evaluation of etiologic factors for root canal therapy. J.Pak Dent Assoc Sep 2005; 14(3): 154-7

20. Diagnosis and managing pulpitis: reversible or irreversible? Adriano Piattelli, MD, DDS*, Tonino Traini, DDS, PhD, Modaresi J, Dianat O, Mozayeni MA. The efficacy comparison of ibuprofen, acetaminophen-codeine, and placebo premedication therapy on the depth of anesthesia during treatment of inflamed teeth. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102(3):399-403.

21. Diagnosis and managing pulpitis: reversibleor irreversible? Adriano Piattelli, MD, DDS*, Tonino Traini, DDS, PhD, Ianiro SR, Jeansonne BG, McNeal SF, Eleazer PD. The effect of preoperative acetaminophen or a combination of acetaminophen and Ibuprofen on the success of inferior alveolar nerve block for teeth with irreversible pulpitis. J Endod 2007;33(1):11-14.

CASE REPORT

A Serus Cyst Adenocarcinoma - A Common Ovarian Malignancy Presenting with A Very Uncommon Presentation

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Abstract

The various malignant form of the epithelial tumor of the ovary are in practice, usually grouped together as ovarian adenocarcinoma. These tumors tends to occur in women aged 45-60 years and are commonly asymptomatic until they are achieved a considerable bulk: most common complaints are of increasing abdominal girth, lower abdominal pain or discomfort and the presence of pelvic mass. Urinary symptoms due to pressure on bladder are quite frequent while disturbance of menstrual cycle or post-menopausal bleeding are unusual. A 60 years old postmenopausal lady, from Ahmedbag, Dhaka was presented with the complaints of post-menopousal bleeding, and heaviness & lump in the lower abdomen for 3 months. She was clinically diagnosed as a case of ovarian tumor (ovarian malignancy) & went through laparotomy followed by surgical staging. Then TAH with BSO with infra-colic omentectomy with pelvic lymphadenectomy was done. Histopathology revealed serus cyst adenocarcinoma (left ovary) without any metastasis to the opposite ovary or any other organs. In our case one of the most common ovarian neoplasm presented with the rarest symptom made the case interesting to report.

Key words: Serus Cyst Adenocarcinoma, Ovarian Malignancy, Epithelial tumour

Journal of Green Life Med. Col. 2019; 4(1): 53-56

Introduction:

Epithelial ovarian cancer is the second leading cause of death from gynaecological cancer, and the 5th most common cancer in women overall.[1] And malignant serous tumors account for 75% of epithelial cancers.[2] It is a serious disease, particularly in advanced stages with a course that is punctuated by frequent tumor recurrence, and has a negative impact on quality and length of life. Disease progression typically occurs via loco-regional peritoneal dissemination and its consequence rather than via visceral metastatic disease; patients commonly develop recurrent ascites and bowel obstruction. Some patients are cured and have high 5year survival even of advanced ovarian cancer with first-line multimodality therapy and therefore research interest in reducing the incidence of recurrence and improving the prognosis of disease is intense.

Case Presentation:

A 60 years old postmenopausal lady, was presented with the complaints of per vaginal bleeding for 3 episode. Bleeding was scanty, blackish in colour and only once for 1st two episode, but in 3rd time it was fresh bleeding & stay for a day but not heavy. It was not associated with pain, per vaginal discharge, itching & also not related with coitus. She also complains for heaviness in the lower abdomen but no pain. Her heaviness was associated with feeling of abdominal distension & vague discomfort along with some weight loss. She also complaints of dyspepsia, anorexia & flatulence. Her bowel & bladder habit was

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Received: 06 December 2018 **Accepted:** 24 December 2018

normal. With all these complaints she consult with a gynaecologist. On examination the consultant found a mildly tender lump of about 8x6 cm occupying left iliac &hypogastric region. It was dull on percussion with areas of resonance in the flanks. Then she was advised USG of whole abdomen. USG revealed partly solid &partly cystic lump. Then she was referred to Green Life Medical College Hospital for further management. On admission, a partly solid & partly cystic lump about 10x8 cm was found per abdominally which was mobile and mildly tender. On Per speculum examination cervix was healthy. On by manual examination, Uterus was bulky (6 weeks) which was deviated to right, a moderate size well defined & freely mobile lump was felt through the left & anterior fornix & a cleft was felt between uterus & the mass. Transvaginalsonography showed bulky uterus with heterogeneous myometrium. Endometrium is ill-defined about 9.6 mm thickened & heterogeneous in echotecture with moderate endometrial fluid collection. For better evaluation MRI was done which revealed: uterus was bulky with multiple fluid collection is seen in posterior myometrium with loss of endo-myometrial differentiation. No mass lesion or fluid collection is seen in endometrium. A large lobulated mass (measuring about 12x10 cm) having solid & cystic component in left adnexa of the lower abdomen. There was no ascites and no lymphadenopathy present. And no sign of metastasis was found in liver or omentum. Endometrial curettage and cervical biopsy was done, which shows atrophic change and chronic cervicitis respectively. Then decision of laparotomy was taken with joint consultation with a general surgeon, anesthetist and oncologist. During laparotomy surgical staging & systemic exploration of abdominal cavity was done, there was no sign of liver, GIT, sub diaphragmatic or omental metastasis. Para aortic lymph nodes were also not enlarged & no ascites was present. There was a solid tumor in the left ovary, which was 12x10 cm in size, surface was smooth. Uterus was 6 weeks in size. Right ovary was atrophied. Then TAH with BSO with infracolicomentectomy with pelvic lymphadenectomy was done. There was no metastasis to the opposite ovary & other organs & without any LVSI (Lympho-vascular Space Invasion).Her histopathology report revealed chronic cervicitis, atrophic change in endometrium and myometrium and moderately differentiated high grade serous cyst adenocarcinoma (left ovary). Lymph node shows reactive changes and omentum was free of metastasis. For her further treatment consultation with oncologist was done and he advised for chemotherapy with paclitaxel and carboplatin.

Discussion:

Cystadenocarcinoma is a malignant neoplasm derived from glandular epithelium, in which cystic accumulations of retained secretions are formed. The neoplastic cells manifest varying degrees of anaplasia and invasiveness, and local extension and metastases occur. It is the most common malignant ovarian tumor contains complex multi-loculated cyst but with exuberant solid areas in places. It usually presents with omental metastases which cause ascites. The current life time risk is 1 per 70 women and the incidence peaking at the age of 67 years.[3] Repeated ovulation thought to be an important factor which favor ovarian cancer and use of oral contraceptive pill has been linked with 40% reduction in risk in some study.[4] Other protective factors include early menarche, late menopause, pregnancy, childbirth, and breast feeding. These are balanced against life style factors that increase risk of the disease, such as women who conceive later in life, having smaller family, obesity and hormone replacement therapy. Approximately 5-10% of ovarian cancer are associated with as autosomal dominant syndrome where there in an inherited defect in one of the three genes: BRCA1 and BRCA2 (site specific ovarian cancer syndrome) and the mismatch repair genes [identify in type ll Lynch syndrome or hereditary non polyposis colorectal cancer syndrome (HNPCC)].[5]

Epithelial ovarian cancer has often been described as a ‘silent killer’, with 75% patients being diagnosed with late stage (stage lll/lV) disease with a 5 year survival is 30-40%, rather than stage l when survival is 84-94%.[6] This is largely because the symptoms of early stage ovarian cancer are thought to be subtle or absent. However, Goff et al. demonstrated that symptoms of EOC are present in 90% of affected women, even with early stage disease, and that these symptoms are often dismissed, so that for 37% of women with the disease there is a delay of at least 6 months from presentation until a diagnosis is made. A ‘symptom index’ includes pelvic or abdominal pain, urinary urge or frequency, or difficulty with eating/early satiety present for less than a year, which has been shown to accurately predict the presence of ovarian cancer when used in combination with serum tumor marker.[7] Ovarian can also cause pelvic pain, constipation or diarrhea, menstrual disturbance or post- menopausal bleeding. Sign include gaseous abdominal distension, pelvic mass, abnormal bowel sound, ascites, palpable abdominal mass, lymphadenopathy, pleural effusion, an umbilical mass

(Sister joseph's nodule), and rarely intra-abdominal organomegaly. Ovarian cancer has extensive heterogeneity within and between histologic subtypes. High-grade serous carcinoma is the most aggressive subtype and accounts for the majority of advanced stage cases. Ten-year survival for all ovarian cancer is approximately 30–40% according to the SEER registry and other studies. Long-term (LT) survival of women with high-grade serous carcinoma (HGSC) is low and often associated with completely resected disease (no gross residual). The diagnosis of ovarian cancer is histopathological and it is important to manage the patient rationally. Histopathological type, tumor grade, and FIGO stageare all determined by biopsy obtained using radiological or laparoscopic guidance or during formal laparotomy. Most large centers require a detailed histopathological analysis in order to manage the patient rationally. Other markers such as oestrogen receptor status can provide useful information for the later management of the patient. CA-125 as stand-alone test, is neither adequate for diagnosis nor for screening as it is elevated in a variety of benign and malignant conditions. In addition, CA-125 is elevated in only 80% of known ovarian cancers and in 50% of those with early-stage disease; In patients whose CA-125 is elevated at diagnosis, serial CA-125 measurement provides a means of assessing response to subsequent chemotherapeutic treatment. Unfortunately, the majority of patients with ovarian cancer will relapse and ultimately die from their disease. While the prognosis in stage I ovarian cancer is excellent, with earlier lower-grade stages having a cure rate of greater than 90%, prognosis, despite evidence of recent improvement, overall leaves much room for improvement, with 1-year survival of 71%, 5-year survival of 40% and 10-year survival of 33% (CRUK website).[8] The main factors that predict for survival include FIGO stage of disease, tumour grade, surgical debulking status, histological subtype and sensitivity of disease to platinum-based chemotherapy. The standard management of stage IC-IV EOC is to perform primary debulking surgery with the explicit aim of total macroscopic clearance and to enable complete surgical staging. This is followed by adjuvant carboplatin-containing chemotherapy for all patients other than those with FIGO stage Ia and lb lower-grade tumours for whom surgery is sufficient and chemotherapy can be omitted.[9] Recent data suggest that in the context of high surgical quality, there is no disadvantage to neo-adjuvant chemotherapy followed by delayed primary surgery.[10] First-line chemotherapy comprises either carboplatin alone or carboplatin in combination with paclitaxel. These standards of care have been defined by international randomized clinical trials Gynecologic Oncology Group (GOG) 111[11], OV-10[12], GOG132[13] and ICON3[14]. Ovarian cancer is best managed by centralized integrated multidisciplinary teams. This has been shown to improve outcomes in this disease. In general, the team consists of a surgical oncologist, a non-surgical oncologist, a radiologist and a pathologist specialized in ovarian cancer management. Palliative care specialist input may be required in all phases of the disease.

Conclusion:

While many factors have been reported to have prognostic value for HGSC beyond the current FIGO staging system, most have limited value for patients with advanced stage disease.[6] Nonetheless, the use of intraperitoneal therapy in patients with microscopic and small volume residual disease after cytoreductive surgery has been associated with long-termsurvival.[15] HGSC and are likely to have had optimal surgicalcytoreduction, defined at the time of this study as <1cm of residual disease. Though studies have shown that survival of advanced stage HGSC patients is poor, there is a dearth of collective data on 10-year survivors of HGSC with sufficient clinical annotations to determine which modifiable and predictive characteristics are reliably associated withoutcome. The few studies that have been conducted generally examined multiple histologic types together. For example, in a study of mixed histologicsubtypes from Sweden, age, stage, residual disease and postoperative CA-125 were all associated with LT survival There are intrinsic biologic factors associated with platinum sensitivity that are generally associated with long-term survival, but a surprising yet small fraction of patients who had either suboptimal cytoreduction or primary platinum resistance achieved LT survival. A SEER registry study found that advanced stage patients and those who did not receive primary surgical resection had worse LT outcomes.[16]Clinical evidence has suggested that low-grade serous carcinoma is not as responsive to conventional (taxane and platinum-based) chemotherapy as high-grade serous carcinoma.[17] Recent studies suggest that a significant number originate from intraepithelial carcinoma in the fallopian tube. Their development is rapid, and they have a better response to conventional chemotherapy compared with low-grade tumors.

Thus, it is clear that low- and high-grade serous carcinomas are distinctly different neoplasms with different pathogenesis, behavior, and response to treatment from high grade. By taking into account the pathogenesis of ovarian cancer as outlined in this dualistic model, a more rational approach to early detection can be undertaken and novel types of treatment can be developed.

References:

1. Dewhurt's textbook of Obstetrics & Gynaecology eight edition, by d. Keith Edmonds, 760-773.
2. Jeffcoate's Principles of gynaecology eight edition by Narendra Malhotra and Protap Kumar,490-527.
3. Boyle P, Ferlay J. Cancer incidence and mortality in Europe, 2004. Ann Oncol 2005;16:481-488.
4. Berchuck A, Schildkraut J. Oral contraceptive pills. Prevention of ovarian cancer and other benefits. N C Med J 1997;58: 404-407.
5. Sogaard M, Kjaer SK, Gayther S. Ovarian cancer and genetic susceptibility in relation to the BRCA1 and BRCA2 genes. Occurrence, clinical importance and intervention. ActaObstetGynecolScand 2006;85:93-105.
6. R.S. Suidan, P.T. Ramirez, D.M. Sarasohn, J.B. Teitcher, S. Mironov, R.B. Iyer, et al., A multicenter prospective trial evaluating the ability of preoperative computed tomography scan and serum CA-125 to predict suboptimal cytoreduction at primary debulking surgery for advanced ovarian, fallopian tube, and peritoneal cancer, Gynecol. Oncol. 134 (2014) 455–461.
7. Andersen MR, Goff BA, Lowe KA et al. Use of a Symptom Index, CAl25:and HE4 to predict ovarian cancer. GynecolOncol 2010;116:378-383.
8. CRUK website 2010; Cancer Stats: Key Facts Ovarian Cancer. Available at: http: / /info. cancerresearchuk. Org/cancer stats/ types/ovary/.
9. Junor EL Hole DJ, McNulty L, Mason M, Young J. Specialist gynaecologists and survival outcome in ovarian cancer: a Scottish national study of 1866 patients. Br J ObstetGynaecol 1999;106:1130-1136.
10. Van Gorp T, Amant F, Neven P, Berteloot P, Leunen K, Vergote I. The position of neoadjuvant chemotherapy within the treat-ment of ovarian cancer. Minerva Ginecol 2006;58:393-403.
11. McGuire WP, Hoskins WJ, Brady MF et al. Cyclophosphamide and cisplatin compared with paclitaxel and cisplatin in patients with stage III and stage IV ovarian cancer. N Engl J Med 1996;334:1-6.
12. Piccart MJ, Bertelsen K, James K et al. Randomized intergroup trial of cisplatin-paclitaxel versus cisplatin-cyclophosphamide in women with advanced epithelial ovarian cancer: three-year results. J Natl Cancer Inst 2000;92:699-708.
13. Muggia FM, Braly PS, Brady MF et al. Phase III randomized study of cisplatin versus paclitaxel versus cisplatin and paclitaxel in patients with suboptimal stage III or IV ovarian cancer: a Gynecologic Oncology Group study. J ClinOncol 2000;18:106-115.
14. The International Collaborative Ovarian Neoplasm (ICON) Group. Paclitaxel plus carboplatin versus standard chemotherapy with either single-agent carboplatin or cyclophospha-mide, doxorubicin, and cisplatin in women with ovarian cancer: the ICON3 randomized trial. Lancet 2002;360:505-515.
15. D. Tewari, J.J. Java, R. Salani, D.K. Armstrong, M. Markman, T. Herzog, et al., Longterm survival advantage and prognostic factors associated with intraperitoneal chemotherapy treatment in advanced ovarian cancer: a gynecologic oncology group study, J. Clin. Oncol. 33 (2015) 1460–1466.
16. L.A. Baldwin, B. Huang, R.W. Miller, T. Tucker, S.T. Goodrich, I. Podzielinski, et al., Ten-year relative survival for epithelial ovarian cancer, Obstet. Gynecol. 120 (2012) 612–618.
17. R.D. Cress, Y.S. Chen, C.R. Morris, M. Petersen, G.S. Leiserowitz, Characteristics of long-term survivors of epithelial ovarian cancer, Obstet. Gynecol. 126 (2015) 491–497.

CASE REPORT

Celiac Disease in Association with Leucocytoclastic Vasculitis And Constipation: An Uncommon Atypical Clinical Manifestation

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Abstract

Celiac disease is an immune mediated disorder precipitated by gluten in genetically susceptible people. Its prevalence is not known in Bangladesh because of easy unavailability of its screening test. Its presentation is diverse; hence diagnosis needs strong suspicion along with clinical expertise. We are reporting here a case of celiac disease with an unusual presentation with leucocytoclastic vasculitis and constipation. After diagnosis, patient was on strict gluten free diet and tablet dapsone for 6 months. The patient improved both clinically and biochemically afterwards. Celiac disease is not that uncommon in Bangladesh and screening test should be done whenever there is any suspicion to rule out celiac disease.

Key words: Bangladesh, Celiac disease, Constipation, Leucocytoclastic vasculitis

Journal of Green Life Med. Col. 2019; 4(1): 57-60

Introduction:

Celiac disease (CD) is an immune-mediated disorder elicited by gluten in genetically susceptible individuals and characterized by the presence of a variable combination of gluten dependent clinical manifestations, celiac disease specific antibodies, HLA- DQ2 or HLA- DQ8 haplotypes and enteropathy.[1] In USA and Europe, its prevalence ranges from 1 in 100 to 1 in 200.[2] Prevalence is not known in Bangladesh due to widespread unavailability of appropriate screening test. Celiac disease is caused by an inflammatory T-cell response to gluten and characterized by the presence of auto-antibodies in blood and histological alterations of the small bowel mucosa. Auto-antibodies are anti-gliadin, anti-reticulin, anti-endomysial and anti-tissue transglutaminase.[3] Histological examination of small intestine shows villous atrophy, crypt hyperplasia and damage to the surface epithelium. The injury is greatest in the proximal small bowel and extends distally for a variable distance.[4] In Bangladesh after the introduction of IgA tTG, as a screening test, few cases of CD have been identified. Here we present a case of celiac disease who presented with malnutrition, constipation and itchy skin rash.

Case presentation:

A 14-year-old female student, hailing from Keraniganj got herself admitted into Shaheed Suhrawardy Medical College Hospital with the complaint of rashes over limbs for 10 days. At first she noticed multiple, painless, itchy, purplish-red rashes over both legs, sparing the soles. On the day of admission rashes flared up and extended to the thighs, buttocks and forearms. She was afebrile. During hospital stay, successive flares occurred at 5 to 7 days' interval that would partly fade out, leaving no scars or pigmentations.

Following 3rd day of admission she developed, pain in the epigastric, umbilical and left lumbar regions that would aggravate after meal & relieve occasionally by vomiting. She gave a history of chronic constipation for about 1

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Received: 08 December 2018 **Accepted:** 24 December 2018

year which would incompletely relieve by usual laxatives. She had normal bladder habit.

She was lethargic, fatigable and had exertional palpitation. She gave no history of haematuria, per-rectal bleeding, facial swelling, sore throat, joint pain or swelling, difficulty in swallowing, oral ulceration, jaundice or tingling, numbness in limbs.

On examination she was ill looking, anxious, anaemic. Body built was normal with low BMI (11.07 Kg/m^2). Koilonychia was present. Skin examination revealed multiple, palpable, non-tender, non-blanching, maculo-papular, purplish-red rash over both legs and forearms. These rashes were occasionally confluent and ecchymotic. Abdomen examination revealed mild abdominal distension with slightly tender epigastric and umbilical regions with no organomegaly or ascites. Bowel sound was sluggish. All other system examinations revealed no abnormalities.

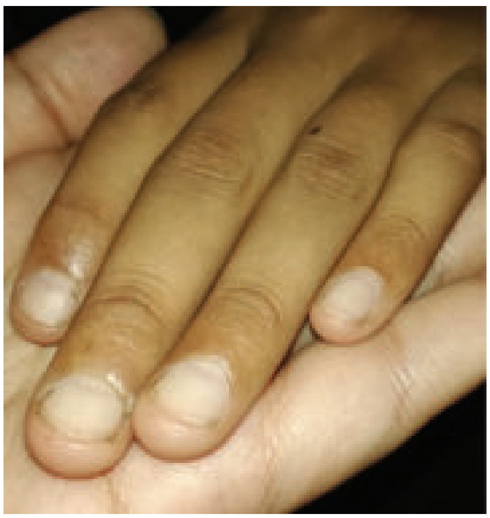

Fig.-1: *Koilonychia*

Fig.- 2: *Skin rash*

Investigation profile:

Investigation	Value	Reference range
Hb (g/dl)	6.2	12-15.5
PBF	Microcytic hypochromic anaemia with frequent target cell with neutrophilic leukocytosis	-
CRP (mg/l)	17.45	<5
ALT	17	10-50
TSH (mIU/mL)	1.80	0.2-4.5
FT_4 (ng/dL)	0.83	0.7-1.63
Iron Profile		
S. Iron (µg/dl)	12	37-145
TIBC (µg/dl)	518	274-494
S. Ferritin (ng/dl)	7.9	15-120
Stool for OBT	Positive	-
S. Creatinine (mg/dl)	0.89	0.57-1.11
S. Electrolytes (mmol/L)		
Na^+	136	135-145
K^+	3.9	3.6-5.0
Cl^-	103	95-107
HCO_3^-	24	21-29
Bleeding time	2 min 45 sec	2-9 min
Clotting time	6 min 30 sec	8-15 min
Chest x-ray P/A view	Normal findings	-
Plain x-ray abdomen	Huge gaseous distension	-
USG of whole abdomen	Normal study	-
S. Amylase (U/L)	26	30-110
S. Lipase (U/L)	69	23-300
HBsAg	Non-reactive	-
Anti HCV	Non-reactive	-
ANA (U/ml)	6.10	<10
p-ANCA (U/ml)	3.10	<6
c-ANCA (U/ml)	2.92	<6
Upper GI Endoscopy	Peptic Ulcer Disease	-
Colonoscopy	Normal up to terminal ileum	-
Tissue biopsy from duodenum	Chronic duodenitis	-
Skin Biopsy	Leucocytoclastic vasculitis	-
Tissue Transglutaminase – IgA (U/ml)	69.2	50

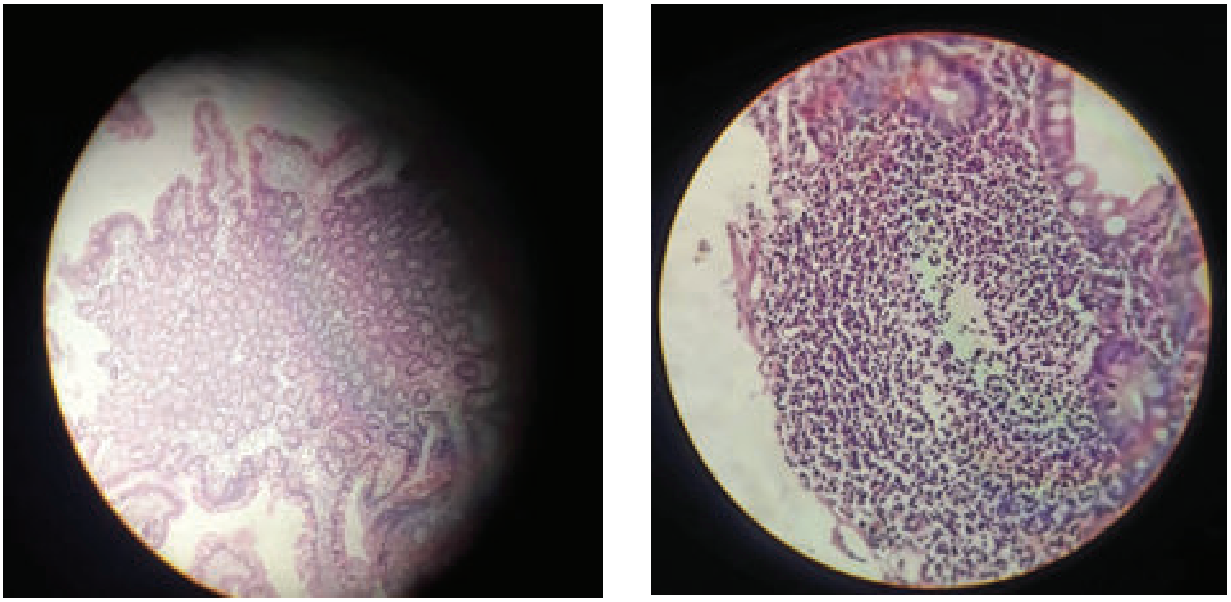

Fig.-3: *Chronic duodenitis*

Discussion:

Celiac Disease (CD) is an immune-mediated intolerance to gluten which is permanent.[2] The activity of gluten resides in the gliadin fraction, which contains certain repetitious amino acid sequences that lead to sensitization of lamina propria lymphocytes. Gluten consists of a complex mixture of many gliadin and glutenin polypeptides. Gliadins are monomers, whereas glutenins form large polymeric structures.[5]

In addition to the classic form of CD, the other recognized forms of this disease are: 1) asymptomatic or clinically silent – with no clinical manifestations and some with gluten sensitive enteropathy. Most of these patients are first-degree relatives of persons with CD;[2] 2) non-classical or atypical – with onset in late childhood; patients presenting with isolated manifestations such as short stature, treatment-refractory iron deficiency anemia, arthritis or arthralgia, epileptic enamel hypoplasia, dermatitis herpetiformis, elevated transaminases, precocious puberty, delayed menarche, recurring abdominal pain, and constipation.[2] Constipation is consistently cited as one of the presenting symptoms of atypical CD, particularly in review articles; however, it has only rarely been described (with supporting evidence) as a symptom of CD in the literature.

We tried to review the literature extensively yielding very few articles with constipation as a symptom of CD in children.[6,7] CD is increasingly recognized in adults without complaints of diarrhea who are often diagnosed during screening. Atypical forms are becoming more common.[8] The North American Society for Pediatric Gastroenterology, Hepatology and Nutrition already recommended to consider CD as an important differential diagnosis of children with persistent gastrointestinal symptoms, including recurrent abdominal pain, constipation, diarrhea and vomiting.[9] A multicenter U.S. study in 2007 diagnosed 22 cases of CD among 976 subjects, four of whom reported constipation.[10]

Patients of CD may also present with various skin manifestations as only presentation, Dermatitis Herpetiformis (DH) being the most common. Other presentations are Cutaneous Vasculitis- Leucocytoclastic Vasculitis, Atopic Dermatitis, Prurigo nodularis, Psoriasis - Palmoplantar pustulosis.[11]

The patient described in this case report had chronic constipation, recurrent abdominal pain and skin rashes with a very low BMI. It is common knowledge that chronic constipation is highly prevalent among pediatric patients and that the vast majority of these cases are of functional etiology. However, when constipation presents alongside a variety of red flags, including compromised nutritional status, it should be regarded as an indication of in-depth diagnostic studies. In the patient described herein, constipation and signs of under nutrition persisted during outpatient follow-ups, with new symptoms of itchy rashes. These prompted the assessment of digestive-absorptive function, leading to a diagnosis of CD. After diagnosis, the patient was on gluten free diet and tablet dapsone for leucocytoclastic vasculitis. After 6 months of treatment, she became symptom free, gained weight [27 kg to 40 Kg;

BMI: 11.07 to 16.4 Kg/m^2], attained menarche and started her school again with good performance.

Conclusion:

To conclude, CD may present in a variety of forms, namely: classical or symptomatic, asymptomatic, and atypical. Although there have been few descriptions of chronic constipation as a symptom of CD, the prevalence of this atypical manifestation does not appear to be negligible. It is assumed that celiac disease is present in Bangladesh. Therefore, it is essential that healthcare providers be aware of it as a possible atypical presenting symptom of CD and should pursue relevant investigations when the disease is suspected.

Reference:

1. Husby S, Koletzko S, Korponay- Szabo IR, Mearin ML, Phillips A Shamir R,etal.European Society for Pediatric Gastroenterology and Nutrition Guidelines for the Diagnosis of Coeliac Disease. JPGN 2012; 54: 136- 160.
2. Fasano A. Clinical presentation of celiac disease in the pediatric population. Gastroenterology 2005; 128:S68-73.
3. Kelly CP, Lamont JT, Bonis PA. Diagnosis of celiac disease. WWW.uptodate.com 2018, accessed on 21.10.2018.
4. Webb C, Halvarsson B, Norstrom F, Myleus A, Carlsson A, Danielsson L et al. Accuracy in Celiac Disease Diagnostics by Controlling the Small bowel Biopsy Process. JPGN 2011; 52: 549-553.
5. Maki M, Lohi O. Celiac Disease. In: Kleinman RE, Sanderson IR, Goulet O, Sherman PM, Mieli-Vergan G, eds. Pediatric Gastrointestinal Disease, 4th edition, Pennysylvania: BC Decker Inc; 2008. P 932- 943
6. Egan-Mitchell B, McNicholl B. Constipation in childhood coeliac disease. Arch Dis Child 1972; 47: 238-40.
7. Sharma A, Poddar U, Yachha SK. Time to recognize atypical celiac disease in Indian children. Indian J Gastroenterol 2007; 26: 269-73.
8. Rampertab SD, Pooran N, Singh P, Green PH. Trends in clinical presentation of celiac disease in the US over the last fifty years. Gastroenterology 2003; 124 (Suppl 1): A659.
9. Hill ID, Dirks MH, Liptak GS, Colletti RB, Fasano A, Guandalini S et al. Guideline for the diagnosis and treatment of celiac disease in children: recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. J Pediatr Gastroenterol Nutr 2005; 40: 1-19.
10. Catassi C, Kryszak D, Louis-Jacques O, Duerksen DR, Hill I, Crowe SE et al. Detection of celiac disease in primary care: a multicenter case-finding study in North America. Am J Gastroenterol 2007; 102: 1454-60.
11. Marzia Caproni, Veronica Bonciolini, Antonietta D'Errico, Emiliano Antiga and Paolo Fabbri.Celiac Disease and Dermatologic Manifestations: Many Skin Clue to Unfold Gluten-Sensitive Enteropathy. Gastroenterology Research and Practice. Volume 2012, Article ID 952753, 12 pages. doi:10.1155/2012/952753

CASE REPORT

Struma Ovarii: A Rare Tumour of Ovary

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Abstract

Struma ovarii is the presence of thyroid tissue as a majore celluler component in an ovarian teratoma. It is rare ovarian tumour with little clinical and imaging features. Most of the cases are benign and often associated with mature cystic teratoma. Increase in the CA-125 level in the serum can potentially lead to a misdiagnosis of a malignant ovarian carcinoma preoperation. About 5-10% of all cases of struma ovarii are reported to be malignant. The diagnosis of a cystic struma ovarii is usually made on histopathology. Prognosis of benign struma ovarii is good following treatment with laparotomy.

Keywords: Ovarian teratoma, Struma ovarii, Thyroid tissue

Journal of Green Life Med. Col. 2019; 4(1): 61-63

Introduction:

Struma ovarii is a rare histological diagnosis, a variant of dermoid in which thyroid tissue constitute > 50 % of the component[1] also called as monodermal ovarian teratoma where thyroid tissue predominantes. It is mostly common in between the ages of 40 and 60 years and accounts for approximately 3% of ovarian teratoma and 0.3% of ovarian tumours.[2] 5-20% of mature teratoma has the component of thyroid tissue,[3] and only 2% of these cases were diagnosed struma ovarii. According to the world health organization (WHO) classification, this disease was diagnosed as ovarian goiter which comprises thyroid tissue either entirely or predominantly (>50%). Malignant struma ovarii is even rarer as occurring in less than 5% of cases and metastases were seldom Struma ovarii rarely produces sufficient thyroid hormone to cause hyperthyroidism. Woman with struma ovarii usually presents with a pelvic mass or less frequently with hyperthyroidism or ascities. Due to its ultrasound morphology, which is quite similar to that of malignant carcinoma , most struma ovarii cases are often operated on with laparotomy.[2] The physician should be very cautious consulting the patient since the proper treatment and the treatment of this rare neoplasm is not well defined.

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Received: 25 November 2018 **Accepted:** 24 December 2018

We present a case of struma ovarii with hypothyroidism managed with laparotomy.

Case Presentation:

A 40 year-old woman presented with vague mass per abdomen and pain for the last 4 months. She was non diabetic and normotensive. But she complains of pain which was dull aching in nature and had history of vomiting before admission. She had no features of thyrotoxicosis, anemia, or fever. She was admitted to our medical college for further workup and management. She had no positive family history of ovarian tumors. On examination, she had no pallor, icterus, or lymph node enlargement. Mild tachycardia (96bpm) respiratory and cardiovascular examination was normal. On abdominal examination, 12 to 10 cm mass was felt on deep palpation on suprapubic region arising from the pelvis, smooth surface and tender. Per speculum findings were suggestive of normal reproductive changes of vagina and cervix. On bimanual pelvic examination revealed a large mass 14 to 12cm felt adherent posterior to the uterus, uterus could not be separated from the lump. Ultrasound showed a large ovarian complex mass (15.8 × 5cm) mainly cystic with marginal irregular soft tissue & septation in the lower abdomen, more in the right probably right ovarian mass. Left Ovary was healthy with tiny follicles. Uterus was bulky with normal endometrial echo. Normal kidney, urinary bladder, no features of obstructions, no ascites and no lymph adenopathy was detected. Blood investigations including ovarian tumors marker was, CA-125, 55u/ml, LDH; 120 u/L. Her thyroid profile showed TSH=8.73µiu/ml; T_3=1.35µg/ml, T4=10.4µg/dl. Then she was treated with Thyroxin 50 mg and was diagnosed as a case of ovarian tumors with hypothyroidism. After consultation with endocrinologist she was advised to come after 2 months for her definitive treatment of laparotomy. The patient was again evaluated after 2 months and her TSH level was 6.65µiu/ml, the ovarian tumors remained unchanged. The patient underwent surgical treatment total abdominal hysterectomy with bilateral salpingo ophorectomy with adhesiolysis. During the procedure , a cystic tumour with a diameter of 14×5 cm arising from the right ovary with the intact capsule , mobile and well circumscribed was visualized.& removed. The pouch of douglas was obliterated, whole of the posterior surface was adherent with sigmoid colon. There was a chocolate cyst in the left ovary. The post-operative period was uneventful and the patient was discharged on 7th post-operative day. She was advised to continue the thyroxin (50µgm) and to repeat the TSH level. After one month her TSH level became 0.01u.i.u/ml without medicine. She became fully cured without any complains.

According to the pathology report the tumors of the rt ovary measured 10×6 cm, which was multiloculated containing straw coloured fluid. Wall of cyst was lined by columnar epithelial cells. The wall contains many variable sizes and shapes of thyroid follicles filled with colloid material No evidence of malignancy was seen. Because of the presence of mature cystic teratoma and thyroid component the definitive diagnosis of thyroid component was struma ovarii.

Discussion:

Adnexal masses are the most common gynecological findings. Ovarian tumors, as a part of adnexal tumor masses, represent two third of these cases. Histopathological diagnosis after adequate surgical treatment is the golden standard for a final diagnosis of an adnexal mass. According to the WHO classification, they represent a group of ovarian monodrama teratomas.[5] Those composed predominantly of thyroid tissue are termed Struma ovarii.[6] This tumor was first mentioned in 1889 by Bottlin. It comprises 1% of all ovarian tumors and 2.7% of all dermoid tumor.[7] Most tumor's present in peri or postmenopausal women with symptoms of enlarging mass or are just incidental findings.[8]They most commonly appear as a unilateral mass but in one fifth of cases a contralateral tumor can also be found. Dermoid cyst may contain hair, teeth, bone and fatty material. Thyroid tissue is rarely found on histological examination. It is not common to see hormonally active Struma ovary. A teratoma having thyroid tissue may converted to hyperplasia, either diffuse or nodular, thyroiditis, lymphoma or carcinoma. It is a rare benign tumors that could present symptoms of hyperthyroidism in 5-8% of cases. Struma ovarii is a rare ovarian tumour with no specific clinical menifestations (Table 1). Raised Cancer antigen (CA) levels can also be found in few cases. [9] This may result in misdiagnosis of malignant ovarian cancer in elderly patient which lead to unnecessary extensive surgery; which means this clinical phenomenon of little value in struma ovarii patients. The gold standard treatment is surgery and prognosis is excellent. During the operation, cysts should be removed totally, so as to avoid the omission and recurrence of the disease. Flushing the abdominal cavity and absorb all the fat / hair may help to recover the intraperitoneal environment. Conservative surgery (cystectomy and oophorectomy) is recommended for struma ovarii, specially if they have fertility potential. Laparoscopic approach may be preferable. Benign struma ovarii and malignant forms without metastasis has good prognosis.

Clinical and pathological features of struma ovarii:

Signs	Macroscopic examination	Scraps smear/frozen section
Pelvic mass	Mostly< 10cm in size	Multicystic mass with lobulated surface.
Hyperthyroidism in 5% cases.	Mixed cystic and solid nodular mass	Sometimes thickened septa
Pseudo-meig's syndrome in 17% cases	Vascular networks	Occasionally has diluted thyroid follicle
Raised CA-125 in a few cases	Hemorrhage, necrosis, fibrosis	Normal appearing thyroid tissue composed of thyroid follicles
Tumor rupture in few cases	Green or brown gelatinous liquid	May associated with hyper/ hypoactivity of thyroid tissue/ nodular goiter.
Non-specific symptoms	Dermoid background in non . cystic tumour	Non specific appearance of epithelial cells with flattened to cuboidal.

However because of the difficulty of setting an accurate preoperative diagnosis, most cases have been diagnosed based on postoperative pathology findings.

With this case report the correlation between the preoperative clinical assessment and intro-operative assessment and the histological diagnosis can be made.

Conclusion:

Struma ovarii is a rare benign ovarian tumour which could mimic an ovarian cancer because the results pre-operatory (ultrasound, CT scan, and CA-125) .Treatment of these tumor includes cystectomy or salpingo-oophrectomy and does not require prolonged follow-up periods or excessive postoperative investigations unless woman are symptomatic. Very few cases of struma-ovarii become malignant but careful evaluation of malignancy is necessary by histopathological examination.

References:

1. Willemse PH, Oosterhuis JW, Aalders JG, Piers DA, Sleijfer DT, Vermey A, *et al*. Malignant struma ovarii treated by ovariectomy, thyroidectomy, and 1311 administration. Cancer 1987;60;178-82.
2. Nurliza Binti Md Nor, Kusumoto T, Inoue S, Nakamura K, Seki N, Hongo A, et al. Three cases of struma ovarii underwent laparoscopic surgery with definite preoperative diagnosis. Acta Med Okayama. 2013;67;191-195.
3. Devaney K, Snyder R, Norris HJ and Tavassoli FA. Proliferative and histologically malignant struma Ovarii; a clinicopathological study of 54 cases. Int J Gynecol Pathol 1993; 12: 333-334.
4. Bocker W. WHO classification of breast tumors and tumors of female genital organs: pathology and genetics. Verh Dtsch Ges Pathol 2002; 86, 116-119.
5. Tovassoli A, Devilee P, editors. World Health Organization classification of tumours of the breast and female genital organs. Lyon: IARC Press, 2003: 171. PMCid: PMC3957561.
6. Hoffman B, Schorge J, Schaffer J, Halvorson L, Bradshaw K, Cunningham G editors. Williams Gynecology 2nd edition. New York McGraw-Hill, 2012; 267.
7. Kim SJ, Pak K; Lim HJ Seong SJ. Clinical diversity of struma ovarii. Korean J Obstet Gynocol 2002;45:748-52.
8. Salhi H, Loamouri B, Boujelbene N, Hassoura JB, Dhiab T, Hechiche M, Rahal K. Primary ovarian Carcinoid Tumor: a report of 4 cases. Int Surg J. 2017; 4(8): 2826-2828.
9. Loizziv, Cappuccini F and Berman ML. An unusual presentation of struma ovarii mimicking a malignant Process. Obstet Gynecol 2002; l00: 1111-1112.

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Date	Topics	Department
08.07.18	Overview of study design	Department of CME
11.07.18	Free radical & anti-oxidants	Department of Biochemistry
18.07.18	Childhood headache	Department of Pediatric
22.07.18	Skills of preparation of presentation	Guest Lecture(Prof. Ayenu)
25.07.18	The Corpus Callosum	Department of Anatomy
29.08.18	Labor Analgesic	Department of Gynae and Obs
05.09.18	Destructions of protective functions of respiratory passage by smoking	Department of Physiology
12.09.18	Procedure of medico legal Autopsy	Department of Forensic Medicine
16.09.18	World Rabies day	Department of Microbiology
19.09.18	Growth failure	Department of Endocrinology
26.09.18	Voriconazole	Department of Dermatology
03.10.18	MDR- TB treatment Bangladesh regimen	Department of Community Medicine
04.10.18	Health hazards with mobile phone	Prof. Pran Gopal Dutta
07.10.18	Fall in elderly population	Department of Medicine
10.10.18	Vit-D deficiency and its effect	Department of Medicine
21.10.18	Young people and mental health in a changing world	Department of Psychiatry
24.10.18	Pain and it's management	Department of Pharmacology
31.10.18	Otitis Media	Department of ENT
04.11.18	Essential pain management	Department of Anesthesia
07.11.18	OCD	Department of Psychiatry
14.11.18	Phantom limb pain	Department of Orthopaedics
18.11.18	Universal Children Day, 2018	Department of Pediatric
02.12.18	AIDS Day: Overview of HIV/ AIDS in Bangladesh	Department of Medicine
05.12.18	Study of Abdominal lump	Department of Surgery
12.12.18	Role of expectant treatment on obstetrics	Department of Gynae and Obs.
19.12.18	Paediatric fracture-how to diagnose	Department of Orthopaedics