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# GREEN LIFE MEDICAL COLLEGE JOURNAL

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## Journal Committee



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# ABOUT THE JOURNAL


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**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).

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**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:**

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- 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.

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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 and Conclusion:**
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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.

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**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.

# Coronavirus Disease 2019 (COVID-19): A Global Health Emergency

The outbreak of SARS-CoV-2, a novel corona virus previously dubbed 2019-nCoV, that emerged in late 2019 in Wuhan, China, and the resulting Covid-19 disease has taken the world by surprise and confirmed our shared global vulnerability to the appearance of new pathogens. It has now infected more than 9826 people across 20 countries. Nearly 200 people have died, all of them in China where the outbreak began.[1]

Corona viruses are enveloped non-segmented positive sense RNA viruses belonging to the family Corona viridae and the order Nido virales and broadly distributed in humans and other mammals. The name "corona virus" is derived from the Latin *corona*, meaning *crown.* When viewed under electron microscope the virus resembles a royal crown or a solar corona. Six (229E, NL63, OC43, HKU1, MERS-CoV and SARS-CoV) were previously known to infect people; SARS-CoV-2 made it seven. Although most human corona virus infections are mild, the epidemics of the two beta corona viruses, severe acute respiratory syndrome corona virus (SARS-CoV) and Middle East respiratory syndrome corona virus (MERS-CoV), have caused more than 10 000 cumulative cases in the past two decades, with mortality rates of 10% for SARS-CoV and 37% for MERS-CoV.[2,3] The third zoonotic human corona virus of the century emerged in December 2019, with a cluster of patients with connection to Hunan South China Seafood Market in Wuhan, Hubei Province, China. The suspicion of emergence of new virus arose when a cluster of people in a sea-food market at Wuhan City, China developed pneumonia without any clear cause. The WHO was notified of the first suspected cases on 31$^{st}$ December 2019 and decided against declaring the outbreak a public health emergency of international concern on 30$^{th}$ January 2020.[4]

Examining the whole genome, SARS-CoV-2 maintains ~80% nucleotide identity to the original SARS epidemic viruses. Its closest whole genome relatives are two bat SARS-like CoVs (ZC45 and ZXC21) that shared ~89% sequence identity with SARS-CoV-2 so most likely ecological reservoirs for SARS-CoV-2 are bats, but it is believed that the virus jumped the species barrier to humans from another intermediate animal host.[5] The WHO reported that environmental samples taken from the marketplace have come back positive for the novel corona virus, but no specific animal association has been identified. An initial report suggested that snakes might be the possible source based on codon usage,[6] but the assertion has been disputed by others. It is now quite clear that efficient human-to-human transmission by respiratory droplets exists and is a requirement for the large-scale spread of SARS-CoV-2. Emerging evidence suggests that it may also be transmitted through contact and fomites.

Similar to SARS-CoV, a recent study confirmed that Angiotensin Converting Enzyme 2 (ACE 2), a membrane exopeptidase, present in humans in the epithelia of the lung and small intestine is the receptor used by SARS-CoV-2 for entry into the human cells. The asymptomatic incubation period for individuals infected with SARS-CoV-2 is estimated to range from 1 to 14 days. Symptoms may include fever, dry cough, shortness of breath, sputum production, sore throat, chills and diarrhea. Further development can lead to severe pneumonia, acute respiratory distress syndrome, sepsis, septic shock, and death. Among those who died, many had preexisting conditions, including hypertension, diabetes, or cardiovascular disease, and the median time from initial symptoms to death was 14 days (range 6–41 days). Men had a death rate of 2.8% while women had a death rate of 1.7%. In those under the age of 50 the risk of death is less than 0.5% while in those over the age of 70 it is more than 8%. No deaths had occurred under the age of 10.[7] The basic reproduction number has been estimated to be between 1.4 and 3.9 which means each infection from the virus would typically be expected to result in 1.4 to 3.9 new infections. Laboratory testing uses real time reverse transcription polymerase chain reaction (rRT-PCR). The test can be done on respiratory or blood samples and results are generally available within a few hours to days.

There is currently no specific antiviral treatment or vaccine to prevent COVID-19. The best way to prevent illness is to avoid being exposed to this virus. However, as a reminder, CDC always recommends everyday preventive actions to help prevent the spread of respiratory diseases, including 1. avoid close contact with people who are sick, 2. avoid touching your eyes, nose, and mouth, 3. stay

home when you are sick, 4. cover your cough or sneeze with a tissue, then throw the tissue in the trash, 5. clean and disinfect frequently touched objects and surfaces using a regular household cleaning spray or wipe, 6. facemasks should be used only by people who show symptoms of COVID-19 to help prevent the spread of the disease to others. The use of facemasks is also crucial for health workers and people who are taking care of someone in close settings (at home or in a health care facility), 7. Wash your hands often with soap and water for at least 20 seconds before eating; and after blowing your nose, coughing, or sneezing.

No cases have yet been detected in Bangladesh however the dense population has made the country vulnerable to the outbreak. The corona viruses already identified might only be the tip of the iceberg, with potentially more novel and severe zoonotic events to be revealed. Global health experts are warning that the outbreak is a harbinger of things to come. The best strategy for thwarting this epidemic, and for preventing the next, is to help other nations — wherever they are — fight humanity's common enemy over there before we have to fight it over here. We must always remember together we are more powerful and containment starts with you.

***Journal of Green Life Med. Col. 2020; 5(1): 1-2***

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## References

1. World Health Organization. (2020). Novel Coronavirus (2019-nCoV): situation report,11. World Health Organization. Available from: https://apps.who.int/iris/handle/10665/330776 (accessed on January 18, 2020)
2. WHO. Summary of probable SARS cases with onset of illness from 1 November 2002 to 31 July 2003. December 31, 2003. Available from: https://www.who.int/csr/sars/country/table 2004_04_21/en/ (accessed on January 19, 2020).
3. WHO. Middle East respiratory syndrome coronavirus (MERS-CoV). November, 2019. Available from: http://www.who.int/emergencies/mers-cov/en (accessed January 19, 2020).
4. WHO Statement (31 January 2020). "Statement on the second meeting of the International Health Regulations (2005) Emergency Committee regarding the outbreak of novel coronavirus (2019-nCoV)". *World Health Organisation. 31 January 2020*. (accessed on January 31, 2020).
5. Rambaut, A. Preliminary Phylogenetic Analysis of 11 n-CoV2019 Genomes, 2020-01-19. Available from: http: // virological.org/t/preliminary-phylogenetic-analysis-of-11-ncov2019-genomes-2020-01-19/329 (accessed on January 20, 2020).
6. Ji, W.; Wang, W.; Zhao, X.; Zai, J.; Li, X. Homologous recombination within the spike glycoprotein of the newly identified coronavirus may boost cross-species transmission from snake to human. J. Med. Virol. 2020.
7. "Coronavirus Age, Sex, Demographics (COVID-19) - Worldometer". Retrieved from: www.worldometers.info (accessed on January 31, 2020).

*ORIGINAL ARTICLE*

# Changes of Different Parameters during Transversus Abdominis Plane Block as Part of a Multimodal Analgesia at Different Time Interval in Total Abdominal Hysterectomy

HAIDER MA[1], BEGUM R[2], ISLAM MS[3], ISLAM MN[4], MONDOL SK[5], HOSSAIN MG[6], IQBAL MJ[7]

***Abstract***

***Introduction: It is very crucial to see the changes of different parameters during transversus abdominis plane block as part of a multimodal analgesia at different time interval in total abdominal hysterectomy.The purpose of the present study was to observe the changes of different parameters during transversus abdominis plane block as part of a multimodal analgesia at different time interval in total abdominal hysterectomy.***

***Methods: This randomized control trial was conducted in Department of Anaesthesia, Analgesia, Palliative and Intensive Care Medicine of Dhaka Medical College and Hospital, Dhaka, Bangladesh from March 2016 to September 2018 for a period two years and six months. Women planned for an elective total abdominal hysterectomy under general anesthesia were selected as study population. Participants were selected and randomly divided into two groups designed as group I and group II. Patient of both group were given general anesthesia. Group I patient received 20 ml 0.25% bupivacaine and group II patient received 20 ml normal saline as placebo. Then dressing was done. The TAP block was performed after taking all aseptic precaution in the flank palpated between the 12$^{th}$ rib (Costal margin) and the iliac crest. After confirmation of correct position, 20 ml 0.25% bupivacaine was given to group I patient and 20 ml normal saline was given to group II patient within the fascial layer which was confirmed by ultrasound. The presence and severity of pain were assessed using a visual analogue pain scale (VAS). Measurement of Sedation by RASS score was done after given morphine.***

***Results: A total number of 40 patients were recruited for this study and were equally divided into two groups. Thus 20 patients were in the group I and the rest 20 patients were in group II. In 0 to 2 hour group I patient's VAS was less than 6 due to effect of TAP block but group II patients were score greater than 6 ($p<0.05$). Again 8-10 hour VAS was significantly higher in group I patient due to gradually reduce the effect of TAP block which was statistically significant ($p<0.05$). In rest of the time VAS was not statistically significant. In Richmond agitation and sedation score of the study patients, it was observed that majority 16(80.0%) patients had sedation sore 0 at (0h-8h) in group I and 5(25.0%) in group II ($p=0.002$). In next 8 hour (>8h-16h) in group I 8(40%) patient had sedation score 0 and 7(35.0%) in group II ($p=0.935$). The difference of sedation score 0 (0h-8h) was statistically significant between two groups($p<0.05$).***

***Conclusion: In conclusion a statistically significant changes is found in different parameters during transversus abdominis plane block as part of a multimodal analgesia at different time interval in total abdominal hysterectomy.***

***Keywords: Transversus abdominis plane block, Multimodal analgesia, Total abdominal hysterectomy***

***Journal of Green Life Med. Col. 2020; 5(1): 3-7***

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**Introduction:**

Hysterectomy is the second most common operation in gynecology.[1]The pain following abdominal hysterectomy results in a delay in the ambulation, longer hospitalization and increased probability of deep vein thrombosis and dissatisfaction of patients. Multimodal analgesia is effective in handling post-operative pain and in attenuating the side effect of large doses of a single analgesic.[2] The existing alternative drugs that are routinely used as post-operative analgesia for these patients is regular intra-muscular/ intravenous/ PCA combined with other oral agents.

The transversus abdominis plane (TAP) block is a technique of regional anaesthesia that provides analgesia to the parietal peritoneum as well as the skin and muscles of the anterior abdominal wall.[3] There is afascial sheath between the internal oblique and transversus abdominis muscles and the nerves lie deep to this fascia. Ultrasound imaging assists accurate location of the transversus abdominis plane compared with a technique that relies on anatomical landmarks with acknowledged variability.[4]

TAP block done with 0.375% levobupivacaine 20ml (10ml injected each side) before surgical incision and post-operative pain was managed by NSAID and IV PCA morphine . They showed reduced visual analog scale pain scores on emergence and morphine requirements in the first 24 postoperative hours in patients of abdominal surgery under general anaesthesia.[5] The TAP block with 0.75% ropivacaine (1.5 mg/kg to a maximum dose of 150 mg bilaterally) compared with placebo reduced postoperative visual analog scale pain scores and total morphine requirements in the first 48 postoperative hours and also incidence of sedation was reduced in patients of cesarean delivery under neuraxial block.[6] This present study was undertaken to observe the changes of different parameters during transversus abdominis plane block as part of a multimodal analgesia at different time interval in total abdominal hysterectomy.

**Methods:**

**Study Population and Settings:** This was a prospective randomized control trial study. This study was conducted in Department of Anaesthesia, Analgesia, Palliative and Intensive Care Medicine of Dhaka Medical College and Hospital, Dhaka, Bangladesh. This study was carried out from March 2016 to September 2018 for a period two years and six months. All women booked for an elective total abdominal hysterectomy under general anesthesia were selected as study population. Women with the history of allergy to bupivacaine or morphine, history of opioid addiction, patients with coagulation disorders, infection at the needle insertion site or patient refuse to give informed consent to be part of the trial. Finally written informed consent were taken from them if they agreed to take part in the trial.

**Randomization:** A total 40 participants were selected and randomly divided into two groups designed as group I and group II by using fixed number sealed envelope technique. Each group contains 20 patients. This grouping was made by assigned anaesthesiologist and given the code number for every patient.

**Intervention:** Patient of both group were given general anesthesia. A routine general anesthesia was performed using the standard technique. After 3 minute pre-oxygenation fentanyl 2 microgram/kg and thiopental sodium 3 to 5 mg/kg were used for induction. For intubation vecuronium 0.1mg/kg was given. Anesthesia was maintained with halothane 0.6 MAC, $N_2O$ 66%, $O_2$ 33% and incremental dose of vecuronium 0.03 mg/kg when needed. After completion of operation and skin closure TAP block (with the help of USG guidance) was given to all patient. Group I patient received 20 ml 0.25% bupivacaine and group II patient received 20 ml normal saline as placebo. Then dressing was done. Finally 20patient was reversed after fulfilling the reversal criteria with neostigmine 50 microgram/kg & 20 microgram/kg atropine. The TAP block was performed after taking all aseptic precaution in the flank palpated between the 12th rib (Costal margin) and the iliac crest. The neuromuscular plane between the internal oblique muscle and the transversus abdominis muscle was identified with ultrasound guidance. Atraumatic blunt needle for peripheral nerve block (Sono Ned, 21G, 110mm length) was advanced by an ultrasound guided in-plane technique at the anterior axillary line. The first "pop" sensation should be felt as the needle reaches the fascial plane between the external oblique and internal oblique muscles. A second "pop" sensation should be felt as the needle enters in the plane between internal oblique muscle and transversus abdominis muscle. The exact location of the needle tip was confirmed via direct ultrasound visualization. After confirmation of the correct position of the needle and negative aspiration, 1 to 2 ml of normal saline was injected to identify position with water dissection. After confirmation of correct position, 20 ml 0.25% bupivacaine was given to group I patient and 20 ml normal saline was given to group II patient within the fascial layer which was confirmed by ultrasound. Study drug were prepared and labeled properly. In postoperative room all patients were receive Inj paracetamol 1gm IV stat and 6 hourly and

diclofenac sodium 50 mg P/R stat and 8 hourly. Time of first demand of analgesic morphine was then recorded. To all patients ondansetron 8mg IV was given along with 1st dose of Morphine.

**Follow up and Outcomes Measures:** The presence and severity of pain were assessed using a visual analogue pain scale (VAS) and continued it at 2 hour interval up to 24 hours and in between 2 hour whenever patient complains about pain. Morphine 0.15mg/kg intramuscular were given when patient complains pain according to VAS score 6 or above 6 (rest/Movement). Total morphine requirements were documented. Incidences of nausea & vomiting were also being recorded. Measurement of Sedation by RASS score was done after given morphine. VAS scoring and decision of given Morphine were done by a volunteer anesthesiologist who was expert enough about VAS score and was fully unaware of the study.

**Statistical Analysis:** Statistical analyses were carried out by using the Statistical Package for Social Sciences version 22.0 for Windows (SPSS Inc., Chicago, Illinois, USA). Unpaired student t-test was used for continuous variables like age, weight, morphine requirement, frequency of morphine required and first analgesic demand. Chi-Square test was used to analyze the categorical variables like ASA class, occurrence of nausea & vomiting, VAS scale, RASS score. P values <0.05 was considered as statistically significant.

**Results:**

A total number of 40 patients were recruited for this study and were equally divided into two groups. Thus 20 patients were in the group I and the rest 20 patients were in group II. Then TAP block with 20 ml of 0.25% bupivacaine in group I and 20 ml normal saline in group II on each side was given before surgical dressing. Monitoring of pulse, blood pressure, time of first analgesic demand, frequency of morphine, VAS score, sedation score and total morphine consumption was observed in 24 hour post-operative period. The mean age of group I were 53.08 ±4.25 and group II were 51.5±4.97 , which was statistically non-significant (p= 0.286) (Table I).

**Table-I**

*Demographic characteristic of the patients (Mean±SD)*

| Variables | Group I | Group II | P value |
|---|---|---|---|
| Age (in years) | 53.08±4.25 | 51.5±4.97 | 0.286 |

Student t test was performed to see the level of significance.

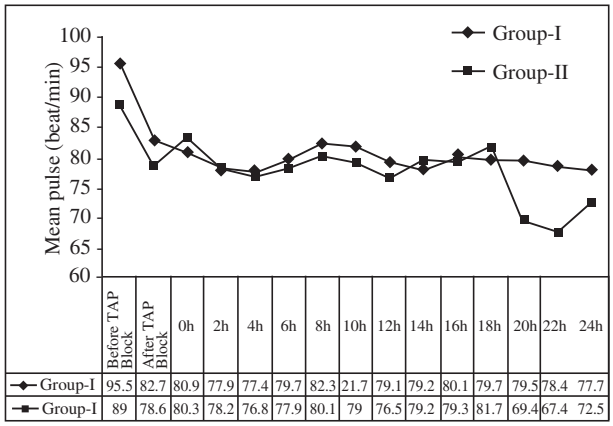


**Fig.-1:** *Comparison of mean Pulse (beat/min) at different time intervals*

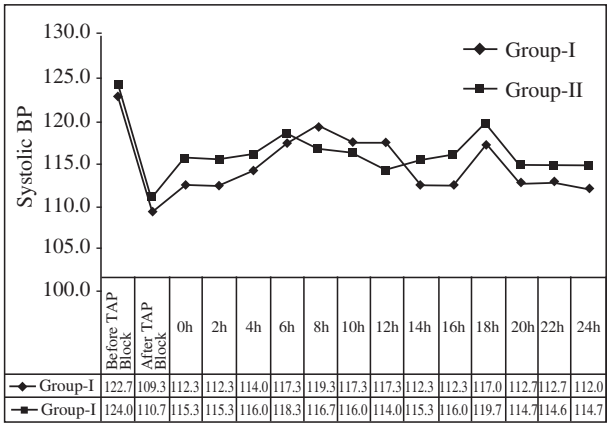


**Fig.-2:** *Comparison of mean Systolic BP at different time intervals*

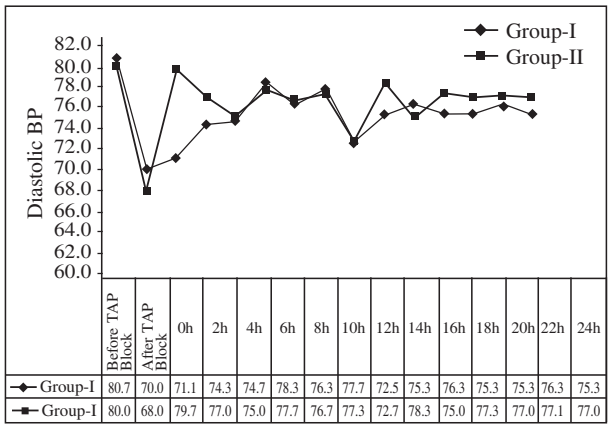


**Fig.-3:** *Comparison of mean Diastolic BP at different time intervals*

In 0 to 2 hour group I patient's VAS was less than 6 due to effect of TAP block but group II patients were score greater than 6, which was statistically significant. After 2 hour VAS score was not significant in between two group due

to group I patient had TAP block and group II patient had received opioid. Again 8-10 hour VAS was significantly higher in group I patient due to gradually reduce the effect of TAP block which was statistically significant. In rest of the time VAS was not statistically significant. *p* value reached from Chi-square test (Table II).

**Table-II**

*Monitoring of VAS at different time interval*

| VAS Score | Group I | Group II | P value |
|---|---|---|---|
| 0 hour to 2hours | | | |
| <6 | 20(100.0%) | 5(25.0%) | 0.001 |
| ≥6 | 0(0.0%) | 15(75.0%) | |
| 4 hours to 6 hours | | | |
| <6 | 16(80.0%) | 15(75.0%) | 0.705 |
| ≥6 | 4(20.0%) | 5(25.0%) | |
| 8 hours to 10 hours | | | |
| <6 | 6(30.0%) | 18(90.0%) | 0.001 |
| ≥6 | 14(70.0%) | 2(10.0%) | |
| 12 hours to 14 hours | | | |
| <6 | 9(45.0%) | 9(45.0%) | 1.00 |
| ≥6 | 11(55.0%) | 11(55.0%) | |
| 16 hoursto 18 hours | | | |
| <6 | 13(65.0%) | 17(85.0%) | 0.144 |
| ≥6 | 7(35.0%) | 3(15.0%) | |
| 20 hours to 22 hours | | | |
| <6 | 20(100.0%) | 20(100.0%) | 1.00 |
| ≥6 | 0(0.0%) | 0(0.0%) | |
| 24 hours | | | |
| <6 | 20(100.0%) | 20(100.0%) | 1.00 |
| ≥6 | 0(0.0%) | 0(0.0%) | |

Regarding Richmond agitation and sedation score of the study patients, it was observedthat majority 16(80.0%) patients had sedation sore 0 at (0h-8h) in group I and5(25.0%) in group II. Here *p* value was 0.002 which was <0.05 that is significant. Innext 8 hour (>8h-16h) in group I 8(40%) patient had sedation score 0 and 7(35.0%) ingroup II. Here *p* value was 0.935 which was not significant. Rest of hour 8(40%)patient had sedation score 0 in group I and group II, which was not statisticallysignificant. The difference of sedation score 0 (0h-8h) was statistically significant($p<0.05$) between two groups (Table III).

**Table-III**

*Richmond Agitation and Sedation Score at Different Time Interval*

| Richmond agitation & SS | Group I | Group II | P value |
|---|---|---|---|
| 0 hour to 8hours | | | |
| -1 | 2(10.0%) | 8(45.0%) | 0.002 |
| 0 | 16(80.0%) | 5(25.0%) | |
| 1 | 2(10.0%) | 7(20.0%) | |
| >8 hours to 16 hours | | | |
| -1 | 7(35.0%) | 8(40.0%) | 0.935 |
| 0 | 8(40.0%) | 7(35.0%) | |
| 1 | 5(25.0%) | 5(25.0%) | |
| 16 hours to 24 hours | | | |
| -1 | 10(50.0%) | 9(45.0%) | 0.881 |
| 0 | 8(40.0%) | 8(40.0%) | |
| 1 | 2(10.0%) | 3(15.0%) | |

SS=sedation score

**Discussion:**

Patients for elective total abdominal hysterectomy under general anesthesia were recruited into this prospective, randomized, controlled study. TAP block was done with 20 ml of 0.25% bupivacaine in trial group and 20 ml normal saline in control group on each side as a part of multimodal analgesia to the anterior abdominal wall.

In this study the different parameters are changes after the application of drugs. In 0 to 2 hour group I patient's VAS was less than 6 due to effect of TAP block but group II patients were score greater than 6, which was statistically significant. After 2 hour VAS score was not significant in between two group due to group I patient had TAP block and group II patient had received opioid. Again 8-10 hour VAS was significantly higher in group I patient due to gradually reduce the effect of TAP block which was statistically significant. In rest of the time VAS was not statistically significant. From this study VAS was used to assess the analgesic demand which was significantly lower in first 2 hour in group I patient than group II. It was due to analgesic effect of TAP block. Belavy et al[7] reported that patient satisfaction with pain relief was significantly higher in TAP block group but there was no significant difference in VAS pain score. Carney et al[3]reported that post-operative VAS pain score at rest or movement were reduced after TAP block at most but not at all time. McDonnell et al[6]also showed that in pain scores were also reduced who received TAP block in the immediate post-operative period and 2, 4, 6 hour postoperatively.

This study showed that sedation score of the study patients were reduced in first 0-8 hour in group I patient. But rest of the time there was no difference with respect to sedation in between two group. McDonnell et al[6]reported that sedation score was reduced in 4 and 6 hour postoperatively. Mrunalini et al[8] and Jadon et al[9] showed that there was no difference with respect to sedation in between two groups. This difference may be due to using different tools to evaluate sedation.

The Richmond agitation and sedation score of the study patients are recorded. It has been observedthat majority 16(80.0%) patients have sedation sore 0 at (0h-8h) in group I and5(25.0%) in group II. Here p-value is 0.002 which is<0.05 that is significant. Innext 8 hour (>8h-16h) in group I 8(40%) patient had sedation score 0 and 7(35.0%) ingroup II. Here pvalue was 0.935 which was not significant. Rest of hour 8(40%)patient had sedation score 0 in group I and group II, which was not statisticallysignificant. The difference of sedation score 0 (0h-8h) was statistically significant($p<0.05$) between two groups. Ra et al[10] also published a randomized controlled trial on the analgesic effect of TAP block after laparoscopic cholecystectomies. They showed that intraoperative use of remifentanil, postoperative pain scores and the postoperative demand for rescue analgesia are significantly reduced in the group that received a TAP bock with Levobupivacaine after induction of anaesthesia. In the above mentioned randomized control studies, the investigators have made use of placebo injections instead of local anesthetics in the control group.

**Conclusion:**

In conclusion a statistically significant changes is found in different parameters during transversus abdominis plane block as part of amultimodal analgesia at different time interval in total abdominal hysterectomy.VAS is significantly higher in group I patient due to gradually reduce the effect of TAP block which is statistically significant. Further multicenter study should be conducted to see the real scenario.

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**References:**

1. Scott Urigel CRNA MS. AANA Journal Course: Update for Nurse Anesthetists: Transversus Abdominis Plane (TAP) Blocks. AANA journal. 2014 Feb 1;82(1):73
2. Atim A, Bilgin F, Kilickaya O, Purtuloglu T, Alanbay I, Orhan ME, Kurt E. The efficacy of ultrasound-guided transversus abdominis plane block in patients undergoing hysterectomy. Anaesthesia and intensive care. 2011 Jul;39(4):630-4
3. Carney J, McDonnell JG, Ochana A, Bhinder R, Laffey JG. The transversus abdominis plane block provides effective postoperative analgesia in patients undergoing total abdominal hysterectomy. Anesthesia & Analgesia. 2008 Dec 1;107(6):2056-60
4. Mukhtar KA, Singh S. Transversus abdominis plane block for laparoscopic surgery. British Journal of Anaesthesia. 2009 Jan 1;102(1):143-4
5. Siddiqui MR, Sajid MS, Uncles DR, Cheek L, Baig MK. A meta-analysis on the clinical effectiveness of transversus abdominis plane block. Journal of Clinical Anesthesia. 2011 Feb 1;23(1):7-14
6. McDonnell JG, O'Donnell B, Curley G, Heffernan A, Power C, Laffey JG. The analgesic efficacy of transversus abdominis plane block after abdominal surgery: a prospective randomized controlled trial. Anesthesia & Analgesia. 2007 Jan 1;104(1):193-7
7. Belavy D, Cowlishaw PJ, Howes M, Phillips F. Ultrasound-guided transversus abdominis plane block for analgesia after Caesarean delivery. British Journal of Anaesthesia. 2009 Aug 22;103(5):726-30
8. Mrunalini P, Raju NV, Nath VN, Saheb SM. Efficacy of transversus abdominis plane block in patients undergoing emergency laparotomies. Anesthesia, essays and researches. 2014;8(3):377-82
9. Jadon A, Jain P, Chakraborty S, Motaka M, Parida SS, Sinha N, Agrawal A, Pati AK. Role of ultrasound guided transversus abdominis plane block as a component of multimodal analgesic regimen for lower segment caesarean section: a randomized double blind clinical study. BMC anesthesiology. 2018 Dec;18(1):53.
10. Ra YS, Kim CH, Lee GY, Han JI. The analgesic effect of the ultrasound-guided transverse abdominis plane block after laparoscopic cholecystectomy. Korean journal of anesthesiology. 2010 Apr;58(4):362

*ORIGINAL ARTICLE*

# Effect of Levocarnitine Administration for Management of Dyslipidaemia in Levothyroxine-treated Hypothyroid Patients

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***Abstract***

***Introduction:*** *Hypothyroidism is the one of the common chronic diseases and patients suffering from this disease show hyperlipidaemia inspite of receiving thyroid hormone replacement.Thyroid hormone play an important role in carnitine-dependent long chain fatty acid transport and oxidation. L-carnitine (LC) plays an important physiologic role in lipid metabolism. It appears rational if such patients are treated with l-carnitine in addition to receiving l-$T_4$. The present study was conducted to evaluate the effect of levocarnitine administration on lipid level and thyroid hormone level in hypothyroid patients.*

***Methods:*** *The present randomized control trial was carried out in the Department of Pharmacology and Department of Endocrinology of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka from September 2016 to February, 2018. The study included a total of 71 hypothyroid patients receiving levothyroxine replacement therapy. Patients were randomly divided into Group A (Control group, n = 35) and Group B (Experimental group, n = 36). Blood was collected for baseline measurement of thyroid hormone levels and serum lipid profile. Group A patients were treated with l-$T_4$ and Group B patients were treated with l-carnitine 2g/day for 8 weeks in addition to l-$T_4$ therapy. After 8 weeks, blood was collected from both groups again to measure the same parameters as were measured at baseline.*

***Results:*** *The baseline characteristics of Group A and Group B were almost identical. The total cholesterol levels significantly increased in Group A (106.52 ± 21.79 mg/dL to 132.11 ± 36.26 mg/dL, P = 0.001) and significantly decreased in Group B (141.69 ±54.41 mg/dL to 123.83 ± 32.76 mg/dL, P = 0.017) after 8 weeks. No significant difference (P = 0.317) was observed between two groups. In Group A (Control group), serum TG levels (132.11 ± 72.98 mg/dL to 149.06 ± 58.54 mg/dL, P = 0.129) was not significantly changed but significant reduction (179.12 ± 103.28 mg/dL to 129.51 ± 59.23 mg/dL, P = 0.007) was observed in serum TG levels in Group B (Experimental group). Intergroup difference was not significant (P = 0.166). There was no significant change in the level of plasma HDL-C, which changed from 23.43 ± 5.64 mg/dL to 21.71 ± 7.57 mg/dL (P = 0.250) in Group A and from 22.67 ± 10.09 mg/dL to 26.28 ± 8.43 mg/dL (P = 0.079) in Group B but significant difference (P = 0.019) was observed between the groups after 8 weeks. In Group A, plasma LDL-C level was significantly increased (56.96 ± 23.17 mg/dL to 80.46 ± 34.00 mg/dL, P = 0.001) and decreased (83.78 ± 45.21 mg/dL to 71.67 ± 31.49 mg/dL P = 0.061) in Group B. No significant difference (P = 0.263) was observed between the Control and Experimental group.*

***Conclusion:*** *The results suggest that, administration of l-carnitine in hypothyroid patients being treated with thyroid hormone (l-$T_4$) replacement had produced significant increase of HDL-C level compared to the group of hypothyroid patients who were treated with l-$T_4$ alone without significant changes in levels of other serum lipids.*

***Keywords:*** *Dyslipidaemia, Levocarnitine, Lipid metabolism, Hypothyroidism, Levothyroxine*

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**Introduction:**

Thyroid diseases namely hypothyroidism and hyperthyroidism constitute common endocrine abnormalities in Bangladesh and around the world. Hypothyroidism is more common compared to hyperthyroidism (prevalence rate of 0.3%-5%).[1,2] According to the 6 years duration NHANESIII (National Health and Nutrition Examination Survey-III) study in USA conducted in the years of 1988-1994, the prevalence rate of hypothyroidism was 4.6% (0.3% clinical and 4.3% subclinical).[3] There is no satisfactory data regarding the total number of patients suffering from thyroid dysfunction in Bangladesh. Yet thyroid dysfunction, specially hypothyroidism affects a significant percentage of population throughout Bangladesh. Male and Female both are affected, although the percentage of females affected are higher.[4]Diffuse goitre occupies the highest incidence (7.35%) followed by subclinical hypothyroidism (6.59%) and clinical hypothyroidism(4.97%).[5] The prevalence of subclinical hypothyroidism was 15%.[6] A recent survey in Bangladesh has reported that, the prevalence rate of hypothyroidism was 48% among all the thyroid disorders.[7] All these data suggest that a significant percentage of the population suffer from hypothyroidism who require replacement therapy with thyroid hormone.

Thyroid hormone is involved in fatty acid oxidation.[8] It enhances transfer of free fatty acids for delivery into the mitochondria.[9] l-carnitine, which is synthesized endogenously in the human body from the essential amino acids lysine and methionine[10] appears as an essential carrier of fatty acids to the inside of the cell.[11, 12, 13] l-carnitine transports long chain fatty acids into the mitochondria whereupon the high energy source (ATP) becomes synthesized.

Hypothyroidism is one of chronic diseases and common metabolic disorder patients suffering from this disease shows hyperlipidaemia inspite of receiving thyroid hormone replacement. Due to several changes in the synthesis, metabolism, and mobilization of lipids, total cholesterol and low-density lipoprotein (LDL) cholesterol level remain elevated in hypothyroidism. Thyroid hormones induce hydroxymethylglutaryl coenzymeA (HMG-CoA) reductase expression in liver, which results in increased cholesterol synthesis.[14] Therefore, hepatic cholesterol synthesis is decreased in hypothyroid patients. However, the expression of cell surface LDL cholesterol receptors expressed in fibroblasts, liver, and other tissues also increased by thyroid hormone. Thus the rate of LDL cholesterol clearance from the serum is increased. This thyroid hormone effect on LDL cholesterol receptor expression outweighs the effects of decreased hepatic cholesterol synthesis, leading to a net accumulation of serum LDL cholesterol in hypothyroidism. Thyroid hormones also increases the activity of lipoprotein lipase which lowers triglyceride levels through hydrolysis of triglyceride-enriched lipoproteins and thus facilitate the transfer of cholesterol from these lipoproteins to HDL cholesterol.[15] Therefore, hypertriglyceridemia may develop in hypothyroidism.

Overt hypothyroidism patientswith hyperlipidemiashould be treated with adequate l-T4 therapy following which TSH level become normal usually by 2 to 4 months.[16] Up to 30% to 50% decrease in the ratio oftotal cholesterol to HDL-C can be expectedwith L-T4 treatment.[17] If the hyperlipidemia has notresolved with l-T4 therapy alone, therapeutic lifestylechanges should be instituted and lipid-lowering medicationsshould be added as appropriate. Clinical trials to date have notshown a significant beneficial effect of l-T4 therapy onlipids in patients with subclinical hypothyroidism, mostlikely because theselipid changes are relatively subtle and according to current evidence, specific lipid-loweringtreatment should be instituted in hyperlipidemic patientswith subclinical hypothyroidism regardless of whether ornot they are treated with l-T4.[16]

L-carnitine (LC) plays an important physiologic role in lipid metabolism. In one clinical study, diabetic patients were investigated using a higher dose ofl-carnitine supplementation of 2000 mg/d (2g/day) for 12 weeks. Significant decreases in the levels of TC, TG, LDL-C, oxidized LDL-C, and Apo-B were observed after 12 weeks and increases were observed inthe levels of HDL-C and Apo-A1.[18] Oral l-carnitine supplementation can decrease TG and increase HDL levels, without significant effects on cholesterol or LDL levels in dyslipidemic ESRD (End stage renal disease) patients under continuous hemodialysis.[19] Another study was done in CAD (Coronary atherosclerotic disease) patients which concluded that, l-carnitine supplementation at a dose of 1000 mg/d (1g/day) resulted in significant increases in HDL-C and Apo A1 levels and a slight decrease in TG levels, but no other changes in other lipids.[20] Administration of 1g oral l-carnitine 3 times a week for 16 weeks has been reported to decrease serum TG and serum VLDL levels without significant changes in levels of other serum lipids.[21]

Researches have suggested that, significantly decreased levels of total carnitinein skeletal muscles due to lack of thyroid hormone in hypothyroid patients.[22] This lowering of carnitine may be explained by decreased biosynthesis

of carnitine.[23, 22] Some reports suggestthat, l-carnitine is a peripheral antagonist of thyroid hormone action.[24]

Several lines of evidence have shown an association between thyroid hormone and the l-carnitine system and those strengthen the suggestion that, Thyroid hormone increases carnitine bioavailability[23] followed by activating carnitine dependent fatty acid import into mitochondria.When hypothyroid patients receive l-$T_4$, thyroid hormone would promote carnitine synthesis but would also accelerate mitochondrial fatty acid oxidation which uses carnitine may future lead to relative carnitine deficiency.

The present study is an attempt to investigate lipid lowering effect of l-carnitine in hypothyroid patients by administering l-carnitine. A large number of patients in this country are suffering from hypothyroidism related hyperlipidaemia although getting adequate l-$T_4$ therapy. So far as the research is informed, adequate study has not been carried out in this country to detect the claimed benefit of l-carnitine on lipid level of hypothyroid patients. So, the present study has been designed to asses ameliorating effects of l-carnitine in the management of hyperlipidaemia in levothyroxine treated hypothyroid patients.

**Methods:**

Being a randomized controlled trial, the present study was carried out in the Department of Pharmacology and Department of Endocrinology of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from September 2016 to February, 2018. The research protocol was reviewed and approved by the IRB of BSMMU on 15th March, 2017. An approval number was collected (No. BSMMU/2017/2668). This study was also registered in Clinical Trial.gov and the ID number was (NCT03372772). All participants (patients) included in the study were informed about the nature and purpose of the study. A written consent was obtained from each person included in the study.

Enrolment of patients were performed by following specific inclusion criteria: clinical diagnosis of primary hypothyroid patients, Age: 20-50 years, both sexes,levothyroxine treatment receiving for last 6 months,serum $FT_4$ level (0.80-1.80 ng/dL)and serum TSH level (0.35-5.50 μIU/mL) were within normal reference range. Patients with following characteristics were excluded from the study: acute or chronic liver diseases, anaemia, clinical diagnosis of diabetes mellitus, cardiovascular disease (such as heart failure, arrhythmia and uncontrolled hypertention), patients with psychological disorders (such as depression, anxiety disorder, schizophrenia, alchoholism or fatigue disorder due other systemic diseases), patients having serious infections or terminal illness (such as tuberculosis, HIV or malignant tumour), autoimmune diseases (such as rheumatoid arthritis, SLE or multiple sclerosis), patients with impaired renal function, pregnant or expecting mothers,nursing mothers, patients receiving drugs eg. corticosteroid, iron, calcium, amantadine, lithium, carbamazepine, phenobarbiton, beta-blocker.

92 patients were enrolled and were randomized into Control group (Group A, n=49) and Experimental group (Group B, n=43). 14 patients from Group A and 7 patients from Group B had dropped out from the experiment due to personal reasons, delaying in follow up or not willing to continue the treatment. Therefore, 35 patients from the Group A and 36 patients from Group B remained to complete the study. Patients body weight, blood pressure and pulse rate were obtained at the time of enrolment in the study (baseline). 6 mL blood were collected following overnight fasting for the baseline measurement of serum TSH and serum $T_4$ level and lipid levels (serum cholesterol, triglyceride, HDL-C, LDL-C level). Group A patients were treated with L-$T_4$ only at appropriate dose orally once daily for 8 weeks and Group B patients were treated with L-carnitine at a dose of 2 gram oral solution daily in two divided dose for 8 weeks in addition to L-$T_4$ therapy. Compliance sheets were provided for each patients. Consumption of medicine was ensured by telephone and return of empty vial and also from the patient's compliance sheet. After 8 weeks, blood was collected again from both groups to measure the same parameters as were measured at baseline. Patients were asked to report for any adverse effects (if observed) of the medication given during the period of study.

Estimation of plasma cholesterol by enzymatic (CHOD-PAP) method;[25] plasma triglycerides by enzymatic (GPO-PAP) method;[26]plasma high density cholesterol (HDL-C) by enzymatic (CHOD-PAP) method, after precipitation;[27, 28] plasma low density lipoprotein cholesterol (LDL-C) by Friedewald equation.

Estimation of serum TSH and free$T_4$ level: Was estimated by automated analyser (Unicel DXI-600).

The data was analysed using SPSS (Statistical Package for Social Sciences) software, version: 19.0. The quantitative variables were expressed as mean ± SD. The 'P' value less than 0.05 considered statistically significant.

**Results:**

All participants were non-smoker, non-hypertensive and non-diabetic. In Group A, the age was 33.51 ± 8.08 years (mean ± SD) and in Group B, the age was 35.42 ± 7.52 years

(mean ± SD). No significant difference between the two groups could be obtained statistically. In Group A, one out of 35 patients was male and 34 were female while in Group B, two out of 36 patients were male and 34 were female. The body weight was 61.54 ± 9.58 kg in Group A and in Group B the body weight was 62.50 ± 8.18 kg. Again there was no significant difference between the two groups.

The serum TSH level in Group A and Group B were2.43 ± 1.56μIU/ml and 2.52 ± 1.38 repectively. After 8 weeks, the same parameter were 2.36 ± 1.56 (Group A) and 3.00 ± 1.49 (Group B) respectively. The change was not statistically significant (P = 0.841 and P = 0.128 respectively). The mean decrease (%) in TSH level in the Group A was 2.88% and the mean increase (%) in Group B was 19.05%. At baseline, the serum $T_4$ level in Group A and Group B were1.29 ± 0.21 ng/dL and 1.29 ± 0.16 ng/dLrespectively. After 8 weeks, these were 1.39 ± 0.30 (Group A) and 1.31 ± 0.18 ng/dL (Group B) respectively. The mean increase (%) in $T_4$ level in the Group A was 7.75% and in Group B was 1.55%. This was not significantly (P = 0.051 and P = 0.597 respectively) different.

At baseline the serum TC levelin Group A patients was 106.52± 21.79 mg/dL(mean ± SD). After 8 weeks of treatment, the serum TC level was increased to 132.11 ± 36.26mg/dL(mean ± SD). The mean increase (%) in TC level was 24.02% which was statistically significant (P = 0.001) than the previous value. On the other hand, baseline serum TC level in Group B patients was 141.69 ± 54.41 mg/dL (mean ± SD). After 8 weeks of treatment, the serum TC level was decreased to 123.83 ± 32.76 mg/dL (mean ± SD). The mean decrease (%) in TC level was 12.60% which was significantly (P = 0.017) more than the previous value. At baseline, the serumTG level in Group A patients was 132.11± 72.97 mg/dL (mean ± SD). After 8 weeks of treatment, serum TG level was increased to 149.06 ± 58.54mg/dL (mean ± SD). The mean increase (%) in TG level was 12.83%. This was not statistically significant (P = 0.129) than the previous value.On the other hand, baseline serum TG level in Group B patients was 179.12 ± 103.28 mg/dL(mean ± SD). After 8 weeks of treatment, serum TG level was significantly (P = 0.007) decreased to 129.51 ± 59.23mg/dL(mean ± SD). The mean decrease (%) in TG level was 27.69%.At the onset of study, serum HDL-C level in Group A patients was 23.43± 5.64 mg/dL(mean ± SD). After 8 weeks of treatment, serum HDL-C level was decreased to 21.71 ± 7.57mg/dL(mean ± SD). The mean decrease (%) in HDL-C level was 7.34%. This was not significantly (P = 0.250) differ from the previous value.The baseline serum HDL-C level in Group B patients was 22.67± 10.09 mg/dL (mean ± SD). After 8 weeks of treatment, plasma HDL-C level was increased to 26.28 ± 8.43mg/dL (mean ±

**Serum TSH and serum FT4 levels in Group A and Group B shown in table 1at baseline and after 8 weeks**

**Table-I**

*Serum TSH and $FT_4$ levels at baseline and after 8 weeks*

| Variables | Group A Control group (L-$T_4$)(n=35) (mean ± SD) | | | | Group B Experimental group (L-$T_4$ + L-carnitine) (n=36) (mean ± SD | | | |
|---|---|---|---|---|---|---|---|---|
| | Atbaseline | After8 weeks | Pvalue | %change | Atbaseline | After8 weeks | Pvalue | %change |
| Serum TSHμIU/Ml | 2.43 ± 1.56 | 2.36 ± 1.56 | 0.841 | ↓2.88% | 2.52± 1.38 | 3.00 ± 1.49 | 0.128 | ↑19.05% |
| Serum$FT_4$ ng/dl | 1.29±0.21 | 1.39±0.30 | 0.051 | ↑7.75% | 1.29±0.16 | 1.31±0.18 | 0.597 | ↑1.55% |

TSH = Thyroid stimulating hormone, $T_4$ =Thyroxine, ↑ = Indicates increase of level,
↓ = Indicates decrease of level, Data was analysed by using **paired t-test**

**Table-II**

*Blood lipid profile in Group A and Group B at baseline and after 8 week*

| Variables | Group A Control group (L-$T_4$ )(n=35) (mean ± SD) | | | | Group B Experimental group (L-$T_4$ + L-carnitine)(n=36) (mean ± SD | | | |
|---|---|---|---|---|---|---|---|---|
| | Atbaseline | After8 weeks | Pvalue | %Change | Atbaseline | After8 weeks | Pvalue | %change |
| Serum TC(mg/dL) | 106.52±21.79 | 132.11±36.26 | 0.001 | ↑24.02% | 141.69±54.41 | 123.83±32.76 | 0.017 | ↓12.60% |
| Serum TG(mg/dL) | 132.11 ±72.97 | 149.06±58.54 | 0.129 | ↑12.83% | 179.12±103.28 | 129.51±59.23 | 0.007 | ↓27.69% |
| Serum HDL-C (mg/dL) | 23.43±5.64 | 21.71±7.57 | 0.250 | ↓7.34% | 22.67±10.09 | 26.28±8.43 | 0.079 | ↑15.92% |
| Serum LDL-C (mg/dL) | 56.96±23.17 | 80.46±34.00 | 0.001 | ↑43.65% | 83.78±45.21 | 71.69±31.49 | 0.061 | ↓14.43% |

TC = Total cholesterol, TG = Triglyceride, HDL-C = High density lipoprotein cholesterol, LDL-C = Low density lipoprotein cholesterol, ↑ = Indicates increase of level, ↓ = Indicates decrease of level, Data was analysed by using paired t-test

SD)which was not statistically significant (P = 0.079) than the previous value. The mean increase (%) in HDL-C was 15.92%. At baseline, the plasma LDL-C level in Group A and Group B were56.96 ± 23.17mg/dL and 83.78 ± 45.21 mg/dLrespectively. After 8 weeks, the same parameter were 80.46 ± 34.00 mg/dL Group A and 71.69 ± 31.49mg/dL in Group B respectively. The mean increase (%) in LDL-C level in the Group A was 41.25% and the mean decrease (%) in LDL-C in the Group B was 14.43%. The change was statistically significant (P = 0.001) in Group A but not significant (P = 0.061) in Group B.

Baseline Comparisons of lipid profile in between Group A and Group B (shown in table 3) were statistically insignificant and Serum TSH level and serum $T_4$ level were within normal range.

**Table-III**

*Distribution of thyroid function status and serum lipid levels in Control and Experimental groups at baseline*

| Groups Variables | Group A (n = 35) | Group B (n = 36) | P-value |
|---|---|---|---|
| Serum TSH () | 2.43±1.56 | 2.52±1.38 | >0.05 |
| Serum Free $T_4$ (ng/dL) | 1.29±0.21 | 1.29±0.16 | >0.05 |
| Serum TC (mg/dL) | 106.52±21.79 | 141.69±54.41 | >0.05 |
| Serum TG (mg/dL) | 132.11±72.98 | 179.12±103.28 | >0.05 |
| Serum HDL-C (mg/dL) | 3.43±5.64 | 22.67±10.09 | >0.05 |
| Serum LDL-C (mg/dL) | 56.96±23.17 | 83.78±45.21 | >0.05 |

P value = statistically significant, Data was analysed by using independent t-test

In comparing between Group A with Group B, it appeared that HDL-C in Group B significantly (P) improved compared to those in Group A.Comparisons between Group A and Group B after 8 weeks shown in table[4].

**Table-IV**

*Distribution of thyroid function status and serum lipid levels in Control and Experimental groups after 8 weeks*

| Groups Variables | Group A (n = 35) | Group B (n = 36) | P-value |
|---|---|---|---|
| Serum TSH () | 2.36±1.56 | 3.00±1.49 | >0.05 |
| Serum Free $T_4$ (ng/dL) | 1.39±0.30 | 1.39±0.30 | >0.05 |
| Serum TC (mg/dL) | 132.11±36.26 | 123.83±32.76 | >0.05 |
| Serum TG (mg/dL) | 149.06±58.54 | 129.51±59.23 | >0.05 |
| Serum HDL-C (mg/dL) | 21.71±7.57 | 26.28±8.43 | >0.05 |
| Serum LDL-C (m3g/dL) | 80.46±34.00 | 71.69±31.49 | >0.05 |

P value = statistically significant, Data was analysed by using independent t-test

**Safety and tolerability assessment during the 8 weeks trial of l-carnitine administration in hypothyroid patients treated with l-$T_4$**

The incidence of adverse events was mild. L-carnitine was generally well tolerated and produced no severe drug-related adverse events.In the group treated with l-carnitine (Group B = 36 patients), 3 patients complained of nausea, 1 patient complained of diarrhoea and 2 patients complained of epigastric discomfort but were not so severe to stop the drug. No serious adverse effects were seen in this group that needed dose adjustment or withdrawal of drug.

**Discussion:**

A total of 71 patients without other co-morbidities and presented with symptoms of fatigue had been included in this study.The patients were on l-$T_4$ therapy for last 6 month or more and had became euthyroid as a result of l-$T_4$ administration. The efficacy of l-carnitine on their thyroid hormone and lipid levels were investigated.

Such patients were allocated l-carnitine administration in additional to their l-$T_4$ therapy (Group B, Experimental group) and was compared after 8 weeks of therapy with theother group of patients who were kept only on l-$T_4$ therapy (Group A, Control group). Parameters for serum lipid levels (TC, TG, HDL-C and LDL-C) and serum thyroid hormone levels (TSH and $T_4$) were measured.

Hypothyroidism is a more or less genetically transmitted endocrine disease where hyperlipidaemia is significantly important complain of patients even after receiving replacement therapy.

In the present study, serum TSH and $T_4$ levelswere within normal reference range at baseline and after 8 weeks in both Group A (P = 0.841, and P = 0.051 respectively and in Group B (P =0.128, and P = 0.597 respectively). No statistically significant difference were observed between the two groups in case of serum TSH and $T_4$ level (P = 0.080 and P = 0.162 respectively) after 8 weeks.This result suggests that there may be no effect of l-carnitine on serum thyroid hormone levels.

Hypothyroidism is a common cause of secondary dyslipidemia. Perhaps the unutilized fat in hypothyroid patients which would have led to ATP formation if patients were not deprived of thyroid hormones had become accumulated. Following l-$T_4$ administration the accumulated body fat including cholesterol, TG and LDL underwent a process of being utilized and this would lead for the body lipid to become normalized at the physiological levels. In a study[29] this was observed that

after restoration of euthyroidism with l-$T_4$therapy serum levels of TC and LDL-C were significantly decreased but serum levels of HDL-C and serum TG levels remained as before.It should be mention that, in the present study serum TC levels of 64 patients out of 71 including both groups (Group A and Group B) were within normal level and serum LDL-C levels of 63 patients were also within normal level at baseline and after 8weeks. Surprisingly and unfortunately serum HDL-C level of 68 patients were below normal reference range both at baseline and after 8 weeks of intervention in Group B even. Serum TG levels were above thenormal range in 32 patients at baseline and in 24 patients after 8 weeks.These observations suggest that, perhaps the serum lipid levels were under the process of being at the physiological range following l-$T_4$ administration andPerhaps a longer time would produce better observations.

According to the statistical analysis of the results of the present study,it was observed that significant decreases of serum TC levels and serum TG levels (P = 0.017 and P = 0.007 respectively) were observed in Group B after 8 weeks. But there was no significant increase of HDL-C level (P = 0.079) or decrease of LDL-C level (P = 0.061). No statistically significant difference was observed among the Group A and Group B in case ofserum cholesterol level, triglyceride level and LDL-C level(P = 0.361, P = 0.166 and P = 0.263 respectively) but significant difference was observed in case of serum HDL-C level (P = 0.019) after 8 weeks. These results would perhaps suggest alleviating effects of l-carnitine upon serum lipid profiles. The observations mentioned above in the present study perhaps corresponds with the studywhere a randomized placebo-cotrolled trial conducted upon patients of chronic kidney disease and has shown that, l-carnitine administration had decreased serum TG concentrations and had significantly increased serum HDL-C concentrations. No significant effect of l-carnitine to decrease serum TC and LDL-C level could be observed in their study.[19]

Hypothyroidism is associated with increased oxidative stress[30] and plasma malondialdehyde (MDA) level which is a marker of oxidative stress are still higher in hypothyroid patients receiving replacement therapy and reached to euthyroid state.[31]Increased oxidative stress underlies the pathophysiology of hypertension[32] and atherosclerosis[33] by directly affecting vascular wall cells.l-carnitine administration has been observed to be associated with significant reduction in oxidative stress and an increased in antioxidant enzymes activities in coronary artery disease patients.[34]

But that at 8 weeks the lipids did not become completely physiological in Group A patients indicate that the alleviation did not occur at full phase. Administration of l-carnitine for 8 weeks in patients of group B has shown a improved status only regarding the serum HDL level. These observations indicate that perhaps the alleviating processes are not yet completed. Perhaps a longer time would produce better observations.

Again wide age range of the study population is a limitation of present study. The sample size was small and therefore the findings cannot be generalized to the total reference population as a whole. The serum carnitine level before and after 8 weeks of intervention were not estimated. Again different confounding factors like menopausal oestrogen level of women aged over 40 years were overlooked.In future larger-scale clinical trials with various dosage forms of L-carnitine would provide better indications about the effects of L-carnitine on lipid profileof hypothyroidism

**Conclusion:**

The results suggest that, administration of l-carnitine in hypothyroid patients being treated with thyroid hormone (l-$T_4$) replacement had produced significant improvement of levels of HDL-C, compared to the group of hypothyroid patients who were treated with l-$T_4$ alone. It is being concluded that thyroid hormone replacement alone in hypothyroid patients cannot significantly lower the lipid level which the patients suffer from. These could bachieved by concurrent administration of l-carnitine.

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**References:**

1. Wang C, Crapo LM. The epidemiology of thyroid disease and implications for screening . Endocrinol Metab Clin North Am, 1997 Vol. 26, pp. 189-218.
2. Bjoro T, Holmen J, Kruger O, Midthjell K, Hunstab K, SchreinerT, et al. Prevalence of thyroid disease, thyroid dysfunction and thyroid peroxidise antibodies in a large unselected population. The Health study of Nord-Trondelag (HUNT). European Journal of Endocrinology and Metabolism, 2000; vol. 87, pp. 489-499.
3. Hollowell JG, Staehling NW, Flander WD, Hannon WH, Gunter EW, Spencer CA, et al. Serum TSH, $T_4$ and thyroid antibodies in the united states population (1988-1994): National Health and Nutrition Examination Survey (NHANES III). Journal of Clinical Endocrinology and Metabolism, 2002;vol. 87, no. 2, pp. 489-499.
4. Allam M. Detection of Prevalence and Characterization of Thyroid Disorder Patients in Dhaka: A Survey based study. 2012

5. Paul AK, Miah SR, Mamun AA, Islam S. Thyroid Disorder in Khulna District: A community based study. Bangladesh Medical Research, 2006;vol. 32 (3), pp. 66-71.
6. Akhter N, Hasan S. Subclinical hypothyroidism and hyperprolectinemia in infertile women: Bangladesh perspective after universal salt iodination. The internet journal of Endocrinology, 2008; Vol. 5(1), pp. 357-563.
7. Rafa MR. Awareness and Prevalence of Hyperthyroidism and Hypothyroidism in Endocrine Disordered patients of Bangladesh. EWU Institutional Repository, 2016.
8. Pucci E, Chiovato L, Pinchera A. Thyroid and lipid metabolism International Journal of Obesity. 2000; 24:109-112.
9. Liu YY, Brent GA. Thyroid hormone crosstalk with nuclear receptor signalling in metabolic regulation.Trends in Endocrinology and Metabolism, 2010 ;Vol. 21, pp. 166-173.
10. Cave MC, Hurt RT, Frazire TH, Matheson PJ, Garrison RN, Mcclain CJ, Mcclave SA. Obesity inflammation and the potential application of pharmaconutrition. Nultrition in Clinical practice, 2008; vol. 23, pp. 1634.
11. Bremer J, Hokland B. Role of carnitine-dependent metabolic pathways in heart disease without primary ischemia. Zeitschrift fur Kardiologie, 1987; vol. 76, pp.
12. Borum PR. Carnitine and lipid metabolism. Bol Asoc Med PR,1991;vol. 83, pp. 134-135.
13. Flanagan JL, Simmons PA, Vehige J, Willcox MDP, Garrettn Q. Role of carnitine in disease.Nutrition and Metabolism. 2010; 7:30.
14. Choi JW, Choi HS. The regulatory effects of thyroid hormoneon the activity of 3-hydroxy-3-methylglutaryl coemzyme A reductase.Endocrine Research, 2000; vol. 26**,** pp. 1–21.
15. Lam KSL, Chan MK, Yeung RTT. High-density lipoprotein cholesterol, hepatic lipase and lipoprotein lipase activities in thyroid dysfunction – effects of treatment.Quarterly journal of medicine.International Journal of Medicine, 1986; vol. 59, pp**.** 513–521.
16. Pearce EN. Hypothyroidism and Dyslipidemia: Modern concepts and approaches . Current Cardiology Report. Current Science Inc, 2004; vol. 6, pp. 451-456.
17. Brien T, Sean F, Dineen, Peter C, Brien, Palumbo PJ. Hyperlipidemia in patients with primary and secondary hypothyroidism. Mayo Clinic Proceeding, 1993; vol. 68 (9), pp.860–866.
18. Malaguamera M, Vacante M, AvitableT, Cammalleri L, Motta M. L-carnitine supplementation reduces oxidized LDL, Cholesterol in patients with diabetes.American journal of Clinical Nutrition, 2009; vol. 89, pp. 71-6.
19. Naini AE, Moradi M, Mortazavi M, Harandi AA, Hadizadeh M, Shirani F, et al. Effects of Oral L-Carnitine Supplementation on Lipid Profile, Anemia, and Quality of Life in Chronic Renal Disease Patients under Hemodialysis: A Randomized, Double-Blinded, Placebo-Controlled. Trial Journal of Nutrition and Metabolism,2012 Article ID : 510483, 6 pages.
20. Lee B, Lin J, Lin Y, Lin. Effect of L-carnitine supplementation on lipid profiles in patients with coronary artery disease.Lipid in health and disease, 2016; vol. 15, pp. 107.
21. Eshghinia IS, Marjani A, Kor BEZ. Effects of Oral L-carnitine Supplementation on Lipid Profiles and Anemia in Patients under Hemodialysis in Gonbadkavoos.Annual Research & Review in Biology, 2014; vol. 4(7), pp. 1092-1098.
22. Sinclair C, Gilchrist JM, Hennessey JV, Kandala M. Muscle carnitine in hypo and hyperthyroidism.Muscle Nerve, 2005; vol. 32, pp. 357-359.
23. Galland S, Georges B, Borgne FL, Conductier G, Dias JV, Demarquoy J. Thyroid hormone control carnitine status through modifications of gamma-butyrobetaine hydroxylase activity and gene expression. Cellular and Molecular Life Science, 2002; vol. 59, pp. 540-545.
24. Benvenga S. Effects of L-carnitine on thyroid hormone metabolism and on physical exercise tolerance.Hormone and Metabolic Research, 2005;vol. 37 ( 9 ), pp. 566-571.
25. Richmond W. Preparation and properties of a cholesterol oxidase from Nocardia sp. And its application to the enzymatic assay of total cholesterol in serum.Clinical Chemistry, 1973; vol. 19, pp. 1350-1356.
26. Fossati P, Prencipe L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clinical Chemistry, 1982; vol. 28(10), pp. 2077-2080.
27. Lopes VMF, Stone P, Elliss S, Colwell JA. Cholesterol determination in high-density-lipoproteins separated by three different methods.Clinical Chemistry, 1977; vol. 23, pp. 882-884.
28. Assmann G, Schriewer H, Schmitz G, Hagele E. Quantification of high density lipoprotein cholesterol by precipitation with phosphotungstic acid/$MgCL_2$. Clinical Chemistry, 1983; vol. 29, pp. 2026-2030.
29. Tsimihodimos V, Bairaktari E, Tzallas C, Miltiadus G, Liberopoulos E, Elisuf M. The incidence of thyroid function abnormalities in patients attending an outpatient lipid clinic. Thyroid, 1999; vol. 9, pp. 365-8.
30. Nanda N, Bobby Z, Hamide A. Inflammation and oxidative stress in hypothyroids: additive effects on cardiovascular risk. Indian Journal of Physiology and Pharmacology, 2011; vol. 55 (4), pp. 351-6.
31. Baskol G, Atmaca H, Tanrivardi F, Baskol M, Kocer D, Bayram F. Oxidative stress and enzymatic antioxidant status in patients with hypothyroidism before and after treatment.Experimental Clinical Endocrinology and Diabetes, 2007; vol. 5 (8), pp. 522-6.
32. Nakazono K,Watanabe N,MatsunoK,Sasaki J,Sato T,Inoue M. Does superoxide underlie the pathogenesis of hypertension? Proc. Natl. Acad. Sci. U. S. A. 1991. **88**: 10045-10048.
33. Ohara Y, Peterson TE, Harrison DG. Hypercholesterolemia increases endothelial superoxide anion production. J. Clin. Invest. 1993; **91**:2546-2551.
34. Lee B, Lin J, Lin Y, Lin P. Effect of L-carnitine supplementation on oxidative stress and antioxidant enzymes activities in patients with coronary artery diseases: a randomized, placebo-controlled trial. Nutrition Journal, 2014; vol. 13, pp. 79.

*ORIGINAL ARTICLE*

# Status of Renal Function in Perinatal Asphyxiated Newborn in a Tertiary Care Hospital

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***Abstract***

***Introduction: Perinatal asphyxia is a perennial problem that has come to stay as one of the single most important cause of neonatal mortality and morbidity. The immediate and long term outcome depends to a large extent on the early recognition and appropriate management of complications. The objective of this study was to assess the renal function in newborn suffering from asphyxia in a tertiary care hospital of Bangladesh.***

***Methods: This cross sectional descriptive study was conducted in Neonatology department of Bangabandhu Sheikh Mujib Medical University from June 2009 to May 2010. Renal function was assessed by urine output, serum creatinine, and fractional excretion of sodium (FENa) in 35 neonates suffering from perinatal asphyxia.***

***Results: Thirty five asphyxiated newborn were studied to find out renal impairment. Mean age of the study babies was 3±2 days. Male female ratio of the newborn was 4:3. Fetal distress was more observed in those delivered by caesarean delivery (57.1%). The commonest history related to birth was premature rupture of membrane (57.1%) and the commonest manifestation of fetal distress was in the form of respiratory distress (82.9%). In the current study 2 babies presented with oliguria and 6 babies had creatinine level above normal limit. According to FENa level renal function was normal in 26 (74.3%) asphyxiated babies while 6 (17.1%) had pre renal failure and 3 (8.6%) had renal failure. Among 35 cases 34 (97.1%) had moderate asphyxia while 1 (2.9%) had severe asphyxia. Renal failures were observed in 2 out of 34 moderately asphyxiated babies and 1 out of 1 severely asphyxiated baby by FENa level. Among 9 asphyxiated babies having renal impairment by FENa level,4 presented with higher creatinine level and 2 cases presented with oliguria.Eight asphyxiated babies died during the course of treatment and all who died had renal impairment.***

***Conclusion: Renal impairment occurs in significant number of asphyxiated neonates. More severely asphyxiated neonates are more likely to experience renal impairmentand death than those with moderate asphyxia. FENa can be recommendedas more sensitive tests for renal tubular functions like urinary beta-2 microglobulin and N-acetyl glucosaminidase (NAG) and cystatin C.***

***Key words: Perinatal asphyxia, Renal impairment, Fractional excretion of sodium***

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**Introduction:**

Asphyxia is an important cause of morbidity and mortality among neonates. It can lead to multiorgan dysfunction. In response to stressful condition of perinatal asphyxia there is redistribution of blood away from organs such as the kidney, bowel, lungs, and skeletal muscle to preserve perfusion of vital organs such as the brain, heart, and adrenal glands.[1] As a consequence, the under perfused organs become vulnerable to hypoxic injury.

Perinatal asphyxia is a Greek word which means stopping of pulse. Perinatal asphyxia is defined as delay of more than 1 minute in onset of spontaneous respiration at birth. Perinatal asphyxia is also an oxygen deficit from the 28

week of gestation to the first 7 days following delivery. As a result an insult to the fetus or newborn occur due to lack of oxygen (hypoxia) and/or circulation (ischemia) to various organs.

It is estimated that about 7 million perinatal deaths occur each year, mostly in developing countries.[2] Nearly 3 -6 million newborn suffer from moderate to severe birth asphyxia with a minimum toll of 800,000 lives and at least equal number develop sequel.[3]

In Bangladesh, 7 – 8 % of rural and 9 – 10% of urban newborn are born with moderate to severe birth asphyxia and more than 150,000 newborn are born with asphyxia every year.[4] About 30% – 50% of infants born with moderate to severe asphyxia ultimately suffer from some form of mental and physical disabilities.

Birth asphyxia constitutes 21% of total neonatal deaths in Bangladesh.[5] But significant data on renal function status in these sick newborn are lacking. Because diagnosis of renal impairment in the neonate and its differentiation from functional oliguria is often difficult and is frequently delayed. Moreover diagnosis of acute renal failure is difficult in neonates as many of the established clinical and biochemical parameters are unreliable in this age group. But it can be assessed by urinary beta-2 microglobulin and N-acetyl glucosaminidase (NAG) and cystatin C.

There are only a few studies done on renal function in newborn with perinatal asphyxia in Bangladesh. The objective of this study was to assess the renal function in newborn suffering from asphyxia in a tertiary care hospital of Bangladesh. The study findings may help to increase awareness of the possibility of renal insufficiency in asphyxiated newborn and thereby progressive increase in diagnosis of acute renal failure.

**Methods:**

This cross sectional study was conducted in the Neonatology department of Bangabandhu Sheikh Mujib Medical University after obtaining ethical clearance from the institute from June 2009 to May 2010. Thirty five diagnosed cases of perinatal asphyxia with given consent were included in the study. Babies were excluded if he or she was premature or post mature, large or small for gestational age, malformed, had significant illness, receiving renal suppressant drugs or whose mothers had significant illness.

A preformed history sheet filled up at enrollment into study containing relevant information such as age of the child, sex of the child, maternal age, maternal antenatal history etc. A careful physical examination was performed.On the basis of apgar score at 5 minutes the asphyxiated babies were further grouped into moderate (score 4-6) and severe asphyxia (score ≤3). Then 2ml of venous blood sample was collected for the estimation of creatinine level and electrolytes. Urine samples were simultaneously collected using commercially available pediatric urine bags. Care was taken to prevent leakage and contamination of urine with stool. The blood and urine sample thus collected were sent for estimation of creatinine, sodium and potassium using computerized auto-analyzer.

Following renal indices was calculated:

Fractional excretion of Na (%) = $U_{Na}/S_{Na} \div U_{Cr}/S_{Cr} \times 100$

Where $U_{Na}$ = Urine Sodium (mmol/L), $S_{Na}$ = Serum Sodium (mmol/L), $U_{Cr}$=Urine creatinine (µmol/L), $S_{Cr}$ = Serum Creatinine (µmol/L) .

An asphyxiated neonate was considered to have renal impairment if any of the following criteria was noted: urine output<0.5 ml/kg/h, serum creatinine >0.9 mg/dl. Acute renal failure was considered if FENa >0.72. Statistical analysis was carried out manually.[6]

**Results:**

Thirty five asphyxiated newborn were studied to see renal impairment. Mean age of the study babies was 3 days with a standard deviation of ± 2 days. Male female ratio of the newborn was 4:3. More than half (52%) of the mothers were primigravida and median age of the mothers was 23 years. Fetal distress was more observed in caesarean delivery (57.1%).

The commonest history related to birth was premature rupture of membrane (57.1%) (Table I) and the commonest manifestation of fetal distress was in the form of respiratory distress (82.9%) (Table II).

**Table I**

*Distribution of asphyxiated babies according to their history related to birth*

| History related to birth | Frequency | Percentage |
|---|---|---|
| Premature rupture of membrane | 20 | 57.1 |
| Prolong delivery | 6 | 17.2 |
| Obstructed labor | 6 | 17.1 |
| Knotting of umbilical cord around the neck | 1 | 2.9 |
| Respiratory failure of mother | 2 | 5.7 |
| Total | 35 | 100 |

**Table II**

*Distribution of asphyxiated babies according to their clinical features*

| Clinical features | Frequency | Percentage |
|---|---|---|
| Respiratory distress | 29 | 82.9 |
| Poor feeding | 17 | 48.6 |
| Cyanosis | 11 | 31.4 |

* Multiple response presents

Oliguria was found in 2 (5.7%) of the study children. Serum creatinine level above 0.9 mg/dl was present in 6 (17.1%) of the newborn. The current study observed 33asphyxiated babies were not suffered from renal impairment accordingto presence of oliguria, out of which 4 were suffered from renal impairment according to serum creatinine level (Table III).

**Table III**

*Distribution of asphyxiated babies according to presence of oliguria and levels of serum creatinine*

| Serum creatinine | Oliguria present | Oliguria absent | Total |
|---|---|---|---|
| Up to 0.9 mg/dl | 0 (00.0%) | 29 (82.9%) | 29 (82.9%) |
| Above 0.9 mg/dl | 2 (5.7%) | 4 (11.4%) | 6 (17.1%) |
| Total | 2 (5.7%) | 33 (94.3%) | 35 (100%) |

But about 25.7% of the asphyxiated newborn had some sorts of renal impairment according to FENa (Table IV).

**Table IV**

*Distribution of asphyxiated babies according to renal impairment by level of Fractional Excretion of Sodium (FENa)*

| FENa level | Frequency | Percentage |
|---|---|---|
| Normal (up to 0.72%) | 26 | 74.3 |
| Pre renal failure (>0.72% - <2.5%) | 6 | 17.1 |
| Renal failure (>2.5%) | 3 | 8.6 |
| Total | 35 | 100 |

Among 35 cases 34 (97.1%) had moderate hypoxia while 1 (2.9%) had severe asphyxia. Renal function was normal in 26 (74.3%) asphyxiated babies while 6 (17.1%) develop pre renal failure and 3 (8.6%) developed renal failure. Renal failures were observed in 2 out of 34 moderately asphyxiated babies and 1 out of 1 severely asphyxiated baby (Table V).

Among 9 asphyxiated babies having renal impairment by FENa level 4 presented with higher creatinine level and 2 cases presented with oliguria.

Twenty seven cases were discharged while 8 died during the course of treatment. All the babies who died had renal impairment (Table VI).

**Table V**

*Distribution of asphyxiated babies according to renal impairment in grades of asphyxia*

| Grading of asphyxia (apgar score) | Normal (%) | Renal impairment (%) | | | Total (%) |
|---|---|---|---|---|---|
| | | Pre renal (% | Renal (%) | Total (%) | |
| Moderate asphyxia ( 4 - 6) | 26 (74.3) | 6 (17.1) | 2 (5.7) | 8 (22.8) | 34 (97.1) |
| Severe asphyxia ( 3 or less) | 0 (00.0) | 0 (00.0) | 1 (2.9) | 1 (2.9) | 1 (2.9) |
| Total | 26 (74.3) | 6 (17.1) | 3 (8.6) | 9 (25.7) | 35 (100) |

**Table VI**

*Distribution of the asphyxiated babies according to their outcome at discharge*

| Outcome at discharge | Frequency | Percentage |
|---|---|---|
| Alive | 27 | 77.1 |
| Dead | 8 | 22.9 |
| Total | 35 | 100 |

**Discussion:**

This descriptive study was carried out with an objective to assess the impairment of renal function in newborns suffering from perinatal asphyxia by doing fractional excretion of sodium (FENa), in addition to serum electrolytes and serum creatinine. A total of 35 newborn were included in the study.

The present study found male female ratio in the newborn suffering from perinatal asphyxia was almost 4:3. In a study found that male was predominant and male female ratio was 3.3:1.[7]

The current study observed 33 asphyxiated babies were not suffered from renal impairment accordingto presence of oliguria out of which 4 were suffered from renal impairment according to serum creatinine level (Table III).In a study it was observed that 28 neonates presented with Oliguria > 24 hours, out of which 8 had renal impairment according to serum creatinine level which was different from present study.[8]

Among cases 34 (97.1%) had moderate hypoxia while 1 (2.9%) had severe asphyxia according to level of FENa. Renal function was normal in 26 (74.3%) asphyxiated babies while 6 (17.1%) develop pre renal failure and 3 (8.6%) developed renal failure. Renal failure were observed in 2 out of 34 moderately asphyxiated babies and 1 out of 1 severely asphyxiated baby (Table V). Impairment of renal function as noted in 25.7% of the cases correlates well with the observation by Goodwin[9] and Nouri[10] but much lower when compared to studies by Perlman[11] and Aldana[12].

In the present study, 8 died during the course of treatment and all the babies who died had renal impairment (Table VI).

The presence of multiorgan dysfunction certainly seems to predict a worse outcome in infants with acute renal failure from any cause, including those of perinatal asphyxia.

**Conclusion:**

Renal impairment occurs in significant number of asphyxiated newborns. More severely asphyxiated newborns are more likely to experience renal failure than those with moderate asphyxia. Renal failure can be anticipated in perinatal asphyxia if present with increased fractional excretion of sodium in addition to oliguria or increased serum creatinine level. FENa can be recommended as more sensitive tests for renal tubular functions like urinary beta-2 microglobulin and N-acetyl glucosaminidase (NAG) and cystatin C.

**References:**


1. Eric DT, Jeffrey M, Periman MB, et al. Renal Injury in Sick Newborn Infants: A Prospective Evaluation Using Urinary â2-microglobulin Concentrations . Pediatrics 1988; 81: 432-440.
2. Costello AM de L. Perinatal health in developing countries. Trans R Soc Trop Med Hyg 1993; 87:1-2.
3. World Health organization Child health and development: health of the newborn, Geneva; WHO 1991; 29:205-9.
4. Joseph R. Resuscitation at birth. Singapore Med J 1990; 31: 166-70.
5. Bangladesh Demographic Health Survey 2004.
6. Mondol N, Bhat BV, Banuoriya C, Koner BC. Oxidative stress in perinatal asphyxia in relation to outcome. Indian J Padiatr.2010; 77:515-7.
7. Airede AL. Birth asphyxia and hypoxic-ischaemia encephalopathy: incidence and severity. Ann Trop Paediatr 1988; 11:331-335.
8. Tack ED, Perlman JM and Robson AM. Renal Injury in Sick Newborn Infants: A Prospective Evaluation Using Urinary $b_2$-Microglobulin Concentrations. Pediatrics 1988; 81: 432-440.
9. Goodwin TM, Belai I, Hernandez P, Durand M, Paul RH. Asphyxial complications in term neo born with severe umbilical acidemia. Am J Obstet Gynecol.1992;167:1506-12.
10. Nouri S, Mahdhaoui N, Beizig S, Zakhama R, Salem N, Ben Dhafer S, et al. Acute renal failure in full term neonates with perinatal asphyxia. Arch Padiatr. 2008;5:229-35.
11. Perlman JM, Tack ED,Martin T, Shackelford G, Amon E. acute systemic organic injury in term infants after asphyxia. Am J Dis Child. 1989;143:617-20.
12. Aladana VC, Romaro MS, Vagas OA, Hermamdez AJ. Acute complications in full term neonates with severe neonatal asphyxia. GinecolObstet Mex. 1995;63:123-7.

*ORIGINAL ARTICLE*

# An Assessment of Hand Hygiene Practice among Adolescent People of Dhamrai Upazilla, Bangladesh

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*Abstract*

***Introduction:*** *Hygiene is essential to the public health mission of reducing the transmission and consequences of infectious diseases. Hand washing is very important hygiene practice, especially for children and adolescents. The objective of the study was to find out the habit of hand hygiene practice in daily life of adolescent people.*

***Methods:*** *A descriptive type of cross sectional study was conducted by non-probable purposive sampling by using a semi structured questionnaire among 418 adolescent people in several villages of Dhamrai Upazilla of Bangladesh from 1st January to 31st March, 2019 through face to face interview. The collected data were analysed manually and some portion by using calculator and computer based software Microsoft office, Excel worksheet.*

***Results:*** *Out of the 418 respondents 212 (50.7%) were male and 206 (49.3%) were female. Most of the respondents were students (93.8%) and 57.4% had completed primary level of education. Among 418 respondents 96.9% always washed their hand before meal and most (98.1%) of them always washed their hand after coming from toilet. Majority (54.8%) used tap water for hand washing and 33.3% washed hands for 1 minute. Among them, 79.9% washed their hands with bar soap. During hand washing 89.9% respondents removed hand accessories and 60.0% washed their hands up to wrist. They (59.09%) dried their hands after hand washing by using separate towels (42.6%). About 68.9% knew about hand washing and they (61.6%) learnt this from their parents. About half (50.9%) of the respondents who reported illness in last 2 weeks of interview with most common morbidities were RTI (31.9%) and GIT (24.8%). Due to these morbidities about 59.1% were absent from school. Among them 43.1% were absent for 1-2 days.*

***Conclusion:*** *Majority of the respondents always washed their hands after coming from toilet but yet it's not satisfactory. Considering the findings of the study it is recommended for arrangement of awareness program and campaigning about hand washing and more educational TV programs and cultural show like puppet show, street drama show should be organized regarding this important issue.*

***Key words:*** *Adolescent, Hand hygiene, Respiratory tract Infection, Gastrointestinal tract infection*

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**Introduction:**

Proper hand washing is one of the simplest, most affordable and effective means of stopping the spread of infection via feces, body secretion , and inanimate objects.[1] Hand hygiene involves any action of hand cleansing, rubbing hands with an alcohol made hand rub or washing hands with soap and water to avoid the growth of microorganisms on hands.[2] Normal hand washing with soap and water is the best component of a hand hygiene program to reduce the risk of contracting infection through contact with hands, however, there is difficulty of

maintaining compliance to basic hand washing practices and this are difficulties to conquer, especially being in a school environment.[3] Hand washing is especially important for adolescent, as this group is more susceptible to infections gained from unwashed hands.[4] The practice of hand hygiene is a simple yet effective way to prevent infections.[5] In developing countries, 80% of the diseases are associated with poor domestic and personal hygiene and about 2.2 million people die; mostly children die annually due to diarrhoea; the same number again die from acute respiratory infections.[6] Hand washing is the most important way to reduce the spread of infection. When hand washing is done correctly by children and adults there can be a 17% reduction in respiratory infections for young children. This translates to prevent more than 100,000 cases of common cold per year.[7] So the general objective of the study was to assess the hand hygiene practice among the adolescent people and their socio demographic status, preference of hand washing, materials used for hand washing, timing of hand washing, drying of hand after wash, materials used for drying hands ,source of water for hand washing.Types of morbidities due to inadequate and improper hand wash and their duration of absence from work place for this reason were also considered.

**Methods:**

It was a cross sectional type of descriptive study.The study was conducted in several villages of Dhamrai Upazilla during the period of 1st January 2019 to 31st March 2019 among 418 adolescent boys and girls with age range of 10-18 years. Non probability convenient sampling was done to select the sample. Semi structured questionnaire was developed, pretested and then finalized. Data were collected by face to face interviewing of adolescent boys and girls residing in DhamraiUpazilla by using semi structured questionnaire. After collection of data, each questionnaire was checked for inconsistency. Then data were analyzed manually and some portion by using calculator and using computer based software, Microsoft Office Excel worksheet.

**Results:**

The age group of 10 to 12 years constituted the highest proportion (57.4%) of the respondents. Fifty one(51%) percent of the respondents were male and 49% female.More than half (57.4%) of the respondents had completed primary level of education & the rest of the respondents had Junior School Certificate level (27.5%), SSC (10.0%), HSC (02%) and others (2.6%) respectively. Most (93.8%) of the respondents were students & rest were day laborers (3.6%), shopkeeper (0.5%) & household worker (1.9%) individually (Table-I).

**Table-I**

*Distribution of the respondents by sociodemographic characteristics (n=418)*

| Sociodemographic characteristics | Frequency (n) | Percentage ( %) |
|---|---|---|
| Age of respondents in year | | |
| 10-12 years | 240 | 57.4 |
| 13-15 years | 116 | 27.8 |
| 16-18 years | 62 | 14.8 |
| Gender of the respondents | | |
| Male | 213 | 51 |
| Female | 205 | 49 |
| Religion of the respondents | | |
| Islam | 373 | 89.2 |
| Hinduism | 45 | 10.8 |
| Educational status | | |
| Illiterate | 02 | 0.5 |
| Primary Education Certificate | 240 | 57.4 |
| Junior school Certificate | 115 | 27.5 |
| SSC | 42 | 10 |
| HSC | 08 | 02 |
| Others | 11 | 2.6 |
| Occupational Status | | |
| Student | 392 | 93.8 |
| Day labourer | 15 | 3.6 |
| Shopkeeper | 02 | 0.5 |
| Household work | 08 | 1.9 |
| Others | 01 | 0.2 |

It has been shown in Table-II, that 86.2% of the respondents had habit of washing hand always before meal and 84.2% had habit of washing hand after coming from toilet always. Only 33.3% washed their hand for one minute that is ideal.

**Table-II**

*Knowledge of respondents about hand washing (n=418)*

| Variable | Frequency (n) | Percentage (%) |
|---|---|---|
| Preference of hand washing | | |
| Before meal | 405 | 97 |
| After coming from out side | 410 | 98.1 |
| After using toilet | 247 | 59.1 |
| After playing | 167 | 40 |
| After handling the animal | 105 | 25.1 |
| After any cleaning | 111 | 26.5 |
| After handling the sick people | 60 | 34.3 |
| After handling the garbage | 153 | 37 |
| After blowing nose | 71 | 17 |
| Whenever hands look dirty | 91 | 22 |
| Habit of hand washing before meal | | |
| Always | 360 | 86.2 |
| Sometimes | 52 | 12.4 |
| Never | 06 | 1.4 |
| Habit of hand washing after coming from toilet | | |
| Always | 352 | 84.2 |
| Sometimes | 56 | 13.4 |
| Never | 10 | 2.4 |
| Time duration of hand washing | | |
| 5 second | 80 | 19.1 |
| 10 second | 56 | 13.4 |
| 15 second | 65 | 15.6 |
| 30 second | 78 | 18.6 |
| 1 minute | 139 | 33.3 |

* Mulltiple response was present

In Table-III shown, more than half (54.8%) of the respondents used tap water whereas 45.2% people used tube well water. Majority (78.7%) of the respondents used bar soap to wash their hands. Surprisingly among 418 respondents about 46.2% male respondents are more conscious about removing hand accessories than female (43.8%). About 59.09% respondents dried their hands after washing hands. Proportion of using separate towel was much more than using common towel after washing hands.

**Table-III**

*Accessories used by the respondents related to Hand Washing (n=418)*

| Variable | Frequency (n) | Percentage (%) |
|---|---|---|
| Source of water for hand washing | | |
| Tap water | 229 | 54.8 |
| Tube well | 189 | 45.2 |
| Material used by the respondents after Hand Washing | | |
| Ashes | 4 | 0.9 |
| Detergent | 12.1 | 2.9 |
| Bar soap | 328.9 | 78.7 |
| Liquid Soap | 43 | 10.3 |
| Only water | 30 | 7.2 |
| Removal of hand accessories | | |
| Yes | 376 | 89.9 |
| No | 28 | 6.7 |
| Do not know | 04 | 3.4 |
| D Drying hands after washing | | |
| Yes | 247 | 59.64 |
| No | 117 | 27.44 |
| Do not know | 54 | 12.92 |
| Way of drying hand after completing the Hand washing | | |
| Using common towel | 145 | 34.7 |
| Using separate towel | 178 | 42.6 |
| Tissue paper | 64 | 15.3 |
| Air dry | 16 | 3.8 |
| Clothing | 15 | 3.6 |

It is shown in figure 01, out of all 418 respondents, about 61.6% learnt about hand washing from their parents, 22% got to know from their teachers and 9% from the media.

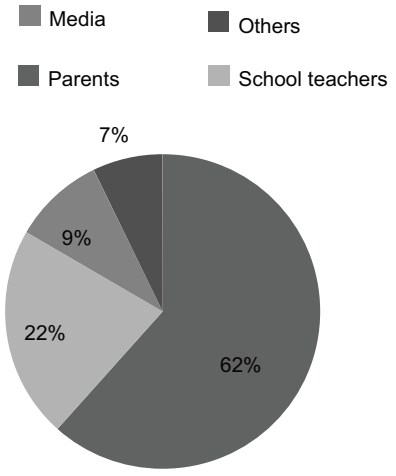


**Figure-01:** Distribution of the respondents according to concerned persons from whom the respondents learn about hand washing. (n=418)

In Figure-02, it is shown that out of 213 respondents about 51% were ill in the last 2 weeks and 49% were not ill.

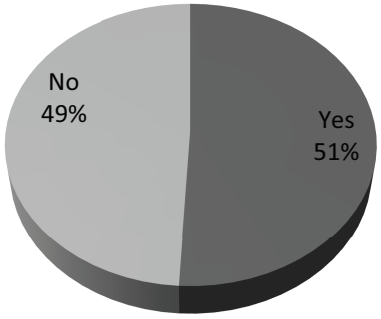


**Fig-2**: *Distribution of the respondents according to their illness in last two weeks.(n=213)*

It is revealed in the figure 03, the type of infection distribution of the study population. Out of 213 samples, majority (44.2%) of the respondents suffered from cold, fever, etc. whereas 31.9% suffered from respiratory tract infection and 24.9% suffered from gastrointestinal tract infection.

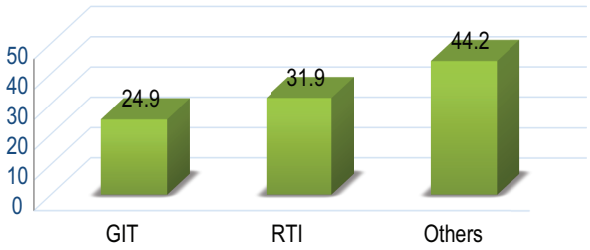


**Fig.-3:** *Distribution of the study population by infection they suffered.(n=213)*

**Discussion:**

The objective of the study was to assess the hand hygiene practice among the adolescent people and their socio demographic status, preference of hand washing, time and materials used for hand washing,drying of hand after wash, materials used for drying hands, source of water for hand washing. Types of morbidities due to inadequate and improper hand wash and their duration of absence from work place for this reason were also considered.

Most of the respondents (57.4%) were in the age group of 10-12 years. Among the total respondents 50.7% were male and 49.3% were female and more than half (57.4%) of them were primarily educated. But a study which was done among Indian students in Puducherry where only 40.19% were in age group of 10-12 years and 48.1% were male students and 51.9% were female students and most of them were in middle school which is quite different from present study.[8]

Regarding economical status most of the respondents (46.9%) belonged to lower middle class family where monthly income was below twenty thousand taka. But in the study conducted in India has depicted a quite astonishing scenario where 66% of the respondents had very low monthly income below three thousand six hundred taka.[9]

In the present study, primary education of the mother and father of the respondents were 25.6% and 16.3% respectively. But primary education of mother and father were almost double in the study of Puducherry which were 38.8% and 28.9% consequently.[9]

Very few (3.2%) of the respondents did not their hand before meal and only 1.9% did not wash hands after coming from toilet which was similar to the study conducted in Chennaiamong school going adolescent where 94.4% washed hands before meal and 89.8% after coming from toilet.[10]

In this study among adolescent respondents 78.7% use bar soap, 7.2% used only water for hand washing. Another study conducted by Md. Abdur Razzak in Bangladesh published in World Journal of Nutrition and Health in 2017, where about 88% adolescent washed their hands with soap and water, less than 9% used soil to wash their hands which is higher than the present study.[11] But a study by Borchgrevnik showed washing hands with soap was 67% which was less in frequency than present study.[12]

About 54.8% respondents used tap water for hand washing in this study which is quite similar to the study done in Chennai where 65.3% had ideal water source for hand washing that was running water from tap.[10]

In this study 61.6% respondents learned about hand washing from their parents, 21.8% from school teachers and 9.4% from media. But In another study conducted in slum area of Kolkatawhere 54.2% from parents, nearly 40.9% of the students learned about hand washing through teachers, 3% from media.[13]

This study reveals that about 24.9% suffered from gastrointestinal infection and 31.9% from respiratory tract infection. It is not unlikely of high prevalence of respiratory tract infection than gastrointestinal infection as because of severe air pollution of Bangladesh, especially in Dhaka. Whereas in America 40 % suffered from GIT infection and 20% from other infections.[7, 14,15,16]

In this study absence of respondent due to illness 43.2% for 1-2 days, 36.6% for 2-3 days and 20.2% for more than 3 days. This finding is almost same with the study conducted in China where absenteeism due to illness 54% and in Egypt 40%. But in Kenya it is 35%, in Philippines 27% and in Columbia 20%.[9,17]

**Conclusion:**

Male respondents were little more in numbers than the female. Most of the respondents always washed their hands before meal & only few of them never washed their hands before meal. Majority of them always washed their hands after coming from toilet but yet it's not satisfactory. It was revealed that there were no female respondents who never washed their hands after coming from toilet. Among the respondents, most of them dried their hands after washing hand but the rate was not up to the mark. The number of the respondents was high who used separate towel to dry their hands after washing and more male used separate towels than female. Hand washing is very important for reducing mortality and morbidity of many diseases. Awareness program should be undertaken among the adult members of the family because a child gets his primary education from his family.Emphasis should be given on school health program as it the crucial period for a person to develop hygiene practice. By extending the quality school health program it will be easier to disseminate healthy life style among the population in a country. In addition, the government should support both private sector and NGOs to conduct more surveys on hand washing.

**References:**


1. Center for Disease Control and Prevention. Department of Human and Services. 2009.
2. Pittet D, Allegranzi B, Boyce J The WHO guidelines on hand hygiene in health care and their consensus recommendations. Infect Control HospEpidemiol. 2009 Jul; 30(7):611-22.
3. Monsma M, Day R, St Arnaud S. Handwashing makes a difference. Journal of School Health. March 1992;62 (3):109-11.
4. Afolabi OO, Adewumi EO, Medavarapu S, et al. A Study to acertain the Practice of Hand Hygiene among Medical Students in Commonwealth of Dominica. Arch Med. 2016, 8:5.
5. Anand D, Prakash S; Assessment of the hygiene and sanitation practices of students of class VI to IX in urban government inter college at Allahabad district, India. Int J Community Med Public Health. 2018 Sep;5(9):3870-75.
6. Beth S, Curtis V, Rabie T, Garbrah-Aidoo N (2007) Health in our hands, but not in our heads: understanding hygiene motivation. Health Policy Plan. 2007 Jul;22(4):225-33.
7. Niffenegger JP. Proper handwashing promotes wellness in child care. J Pediatr Health Care 1997 Jan-Feb; 11(1): 26-31.
8. Behera BK, Jena SK et al. Hygiene practice in rural school children in Puducherry. Journal of Evolution of medical and dental sciences 2013; 2(24):4363-72.
9. Besha B, Guche H, Chare D et al. Assessment of Hand Washing Practice and it's Associated Factors among First Cycle Primary School Children in Arba Minch Town, Ethiopia, 2015. Epidemiology (Sunnyvale) 2016;6(3): 247-57.
10. Tamilarasi R, Arunmozhi R, Raja VK, Rajajeyakumar M. A Study to Assess the Knowledge and Practice of Hand Washing among School Going Adolescents in Chennai.International Journal of Health Sciences & Research 2016 August; 6(8): 147-55.
11. Md. Abdur Razzak, Md. Asaduzzaman, FarhaMatin Juliana, UmmaySadia, Syed Mahfuz Al Hasan, and Md. SabirHossain, "Nutrition Education Effects on Better Hand Hygiene Practice Among Adolescent Girls." World Journal of Nutrition and Health, vol. 5, no. 2 (2017): 30-32.
12. Borchgrevink CP, Cha J, Kim S. Hand Washing Practices in a College Town Environment, carl P. Borchgrevink, Jaemin cha, PhdSeunghyun Kim J Environ Health 2013 Apr;75(8):18-24.
13. Sarkar M. Personal hygiene among primary school children living in a slum of Kolkata, India. J Prev Med Hyg 2013 Sep; 54(3): 153–58.
14. Curtis V, Cairncross S. Effect of washing hands with soap on diarrhoea risk in the community: A systematic review. Lancet Infect Dis 2003; 3:275-81.
15. Nasreen S, Azziz-Baumgartner E, Gurley ES. Prevalent high-risk respiratory hygiene practices in urban and rural Bangladesh. Trop Med Int Health, 2010 Jun;15(6);762-71.
16. Ryan MA, Christian RS, Wohlrabe J. Handwashing and respiratory illness among young adults in military training. Am J Prev Med 2001;
17. Zhou G, Jiang T, Knoll N, Schwarzer R. Improving hand hygiene behaviour among adolescents by a planning intervention. Psychol Health Med, 2015; 20 (7):824-31.

*ORIGINAL ARTICLE*

# A Study of Child Sexual Abuse Recorded in Dhaka Medical College Hospital

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***Abstract***

***Introduction: Child sexual abuse (CSA) is a global problem that has significant consequences for public health; it has been a prominent topic of public concern for more than a decade, but many basic facts about the problem remain unclear or in dispute.***

***Methods: This retrospective cross-sectional study was conducted among victims of CSA at Forensic Medicine department and one stop crisis center of Dhaka Medical College, during the period of January 2017-December 2017.***

***Results: We conducted the study of 311 cases of CSA in order to highlight the epidemiological features and negative impact on victims' well-being and to emphasize the need for a multidisciplinary approach to the primary prevention and management of CSA. We noted an increase in cases number with male predominance. Most of our patients came from lower socioeconomic classes. The perpetrators were male in 100% of cases; acquaintances in 70% of cases and family members in 22 cases. Physical examinations were normal in 61% of cases, however, a range of psychological and physical effects were identified with dramatic health consequences: three cases of attempted suicide, five pregnancies and one case of HIV virus infection.***

***Conclusion: From the findings, conclusion seems children are probably the mostneglected members of our society. As a result, they are consistently becoming easy victims of all sortsof abuses.Violence against children must be stoppedand the judiciary, law enforcing agents, parents andguardians of the children themselves must besensitized to the provisions of the convention on theRights of the child and the laws protecting childrenin Bangladesh.***

***Key words: Child sexual abuse, Rape, incest, Child suicide***

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**Introduction:**

Childhood sexual abuse (CSA) is a complex life experience that has become the subject of great community concern and the focus of many legislative and professional initiatives.[1] This is evidenced by the expanding body of literature on sexual abuse, public declarations by adult survivors and increased media coverage of sexual abuse issues. However, in Bangladesh, because sexual abuse is usually a hidden offense, there are no statistics on how many cases actually occur each year.[2] Statistics cover only the cases that are disclosed, to one stop crisis center, to child protection associations, to children´s hospitals or to law enforcement.[3] The purpose of our study is to highlight the epidemiological features and negative physical and mental health effects on CSA victims; and

emphasize the need for a multidisciplinary approach to the primary prevention and management of CSA.

**Methods:**

We conducted a 1year (January 2017 - December 2017) retrospective study of CSA victims consulting at the department of Forensic Medicine of Dhaka Medical College and One Stop Crisis Center (OCC) of Dhaka Medical College Hospital. The clinical records of 311 patients were reviewed; we identified demographic data, CSA characteristics, clinical and psychological features and therapeutic and follow-up data. The study design and data collection that is reviewed from clinical record of one stop crisis center. The data collection technique and approval was taken from DMC ethical clearance committee.

**Results:**

Before the late 2008s, CSA cases were sporadic. In the following decades, the number of cases reported annually increased with a peak in 2017, since that year a little decline was recorded (Figure 1).

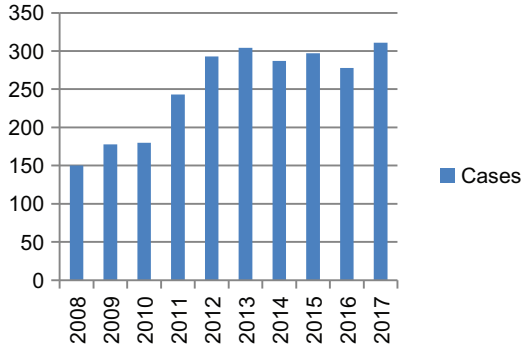


**Fig.-1:** *Frequency of Cases*

**Demographic characteristics**

We noted that approximately 15% of victims were between ages 0 and 5 years. Between ages 6 and 10 years, the percentage almost tripled (48%). Ages 11 to 15 years accounted for a quarter (26%) of cases, with children 16 years and older accounting for the remaining 11 % of cases (Table 1)

**Table-I**

*Age distribution of the victims*

| Age (Years) | No. of Cases | Percentage % |
|---|---|---|
| 0-5 | 47 | 15% |
| 6-10 | 149 | 48% |
| 11-15 | 81 | 26% |
| 16-18 | 34 | 11% |
| Total | 311 | 100% |

Before the age of 16 years boys were at about two times higher risk than girls, with a percentage of 68 %. Victims 16 years and older were female in 82% of cases (Figure 2).

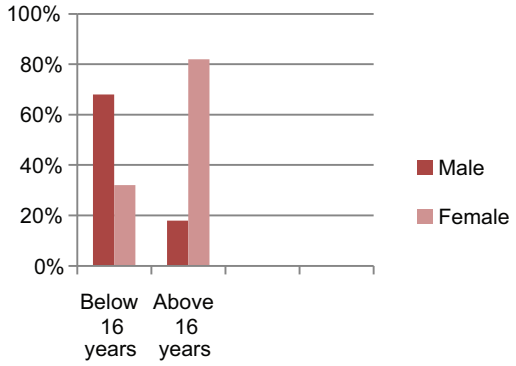


**Figure 2:** *Gender distribution of the victimsWe identified 3 cases of CSA with mental retardation*

Reported cases came from all socioeconomic classes; however, almost 72% of cases had a low socioeconomic status, the majority was living in sub-rural areas.

**Table-II**

*Socioeconomic status of the victims*

| Socioeconomic status | No. of Cases | Percentage % |
|---|---|---|
| Low | 224 | 72% |
| Middle | 59 | 19% |
| High | 28 | 9% |
| Total | 311 | 100% |

**Family structure:** We observed some cases of familial impairments, in fact parental substance abuse was noted in 11% of cases, the absence of one/both parents was identified in 17% of cases, as well as the presence of a stepfather in the home (8%) and parental conflicts (45%).

**Table-III**

*Family structureof the victims*

| Family structure | No. of cases | Percentage % |
|---|---|---|
| Parental substance abuse | 34 | 11% |
| Absence of one/both parents | 53 | 17% |
| Presence of a stepfather | 25 | 08% |
| Parental conflicts | 140 | 45% |
| Normal Family structure | 59 | 19% |
| Total | 311 | 100% |

**Offender:** 100% of child sexual abuse perpetrators were men.

**Offender's relationship to victim:**81% of victims were sexually abused by a non-relative(figure:3); offenders outside the family were casual acquaintances of the victim in 70% of cases and strangers in 30% of cases (figure: 4). Employers were the offenders in just 3% of victims. We identified 16% cases of incest; of these, two thirds were abused by their biological fathers. 7% of victims were abused by multiple offenders.

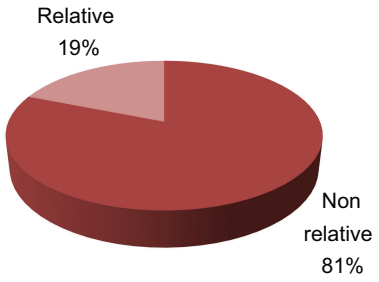


**Fig.3 :**

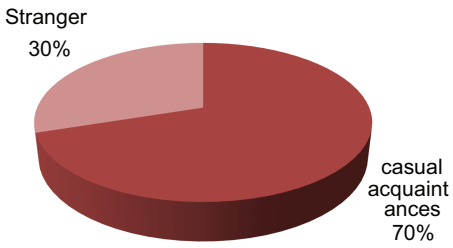


**Fig.4 :**

We noted a spectrum of sexual abuse types ranging from non-contact forms to contact forms of abuse, through to intercourse. In fact, 64% of victims were sodomized, 18% were subjected to fondling and 10% of cases had oral-genital intercourse. We also noted defloration in 8% and exposure to pornography in two cases. Furthermore, the sexual abuse was associated with physical violence in 21% of cases.

**Table-IV**
*Type of abuse*

| Type of abuse | No. of cases | Percentage % |
|---|---|---|
| Sexual intercourse | 25 | 8% |
| Sodomy | 199 | 64% |
| Fondling | 56 | 18% |
| Oral genital intercourse | 31 | 10% |
| Total | 311 | 100% |

CSA occurred as repeated episodes in 67% of cases: of these, victims were re-abused by the same perpetrator in 78% of cases.

Reporting delay ranges from a few hours to 24 months.

Clinical examination was normal in 61% of cases; it showed in the remaining cases nonspecific findings such as vulvovaginitis, erythema, anal fissures and perianal scars, as well as anal dilatation with stool soiling. We noted anal warts in one case. Signs of additional physical violence were noted in 11%.

We observed through our study an array of behavioral disorders with different degrees including fear, anxiety, irritability, regression in school performance, sleep disturbances, eating disorders, social problems as well as poor self-esteem. We also noted inappropriate sexualized in some cases. However, approximately 22% of our patients had no symptom. Incest victims had particularly severe problems such as depression and attempted suicide that was noted in three cases.

The main consequences included three cases of suicide attempts; five cases of pregnancies, three of whom were subjected to incest. Two cases of sexually transmitted infections (STIs) were noted, HIV infection in one case (revealed by the systematic screening for STIs) and HPV anal warts in the second case.

Management was based on a multidisciplinary approach with on numerous components ranging from medical and psychological treatment to reporting through social support.

**Discussion:**

Having a clear operational definition of child maltreatment - and CSA as a specific aspect of child maltreatment- is increasingly recognized as fundamental to effective preventative strategies.[1] The World Health Organization has defined child sexual abuse as being: "The involvement of a child in sexual activity that he or she does not fully comprehend, is unable to give informed consent to, or for which the child is not developmentally prepared and cannot give consent, or that violates the laws or social taboos of society. Child sexual abuse is evidenced by this activity between a child and an adult or another child who by age or development is in a relationship of responsibility, trust or power, the activity being intended to gratify or satisfy the needs of the other person. This may include but is not limited to: the inducement or coercion of a child to engage in any unlawful sexual activity; the exploitative use of a child in prostitution or other unlawful sexual practices; the exploitative use of children in pornographic performance and materials".[2]

Designing effective child protection measures requires a reliable understanding of the extent of the problem and its context. Globally, the number of studies on the prevalence of CSA has been growing. Based on a summary of existing studies, WHO estimates that between approximately 20 % of girls and 5 to 10 % of boys are victims of sexual abuse all over the world[4]. In Bangladesh, like many developing countries, there is a huge lack in data and the existing findings don't reflect the accurate magnitude of the problem, the main challenge is the socio-cultural context and the huge culture of silence that surround sexual issues. A further challenge is that current estimates vary widely as a function of the definitions used, the quality of data collection methods as well as the age of study participants and the age at which childhood is defined. As a global phenomenon, CSA was regarded as rare before the late 1970s. In the following decades, we noted through our study and many other series that the incidence increased dramatically (Figure 1).[5] Although much of this apparent increase probably reflected a growing awareness among the public and professionals, some studies suggest that the overall incidence of child abuse and neglect increased.[6] The increase in our study may also be due to the creation in 2008 of the children's listening and protection center of the child rights observatory, a structure that provides support and encourages victims to disclose their victimization; reported cases of CSA, however, declined since 2017. This decline could be due to the creation of new medical centers where new cases were referred instead of our department.

While it is impossible to create a profile of children who will be sexually abused, it is possible to describe characteristics that are more common among victims and are identified as risk factors for CSA.

There is some discrepancy in the available data about whether teenagers are at higher risk or whether the risk is more uniformly distributed. Some data[7] show a relatively uniform risk for children after age 3. Other studies found that over half of the children who were sexually victimized were between 15-17 years old.[8] In our study, nearly half of cases were between 6 and 11years while children aged 16 years and older counted 11% because most of them were referred to the gynecological department (for girls) and the adult emergency department (for boys). Moreover, some studies[9] believe that, as a risk factor, age operates differentially for girls and boys, with high risk starting earlier and lasting longer for girls.

Physical disabilities are associated with increased risk[10]. Three factors seem to contribute to this increased vulnerability: dependency, institutional care, and communication difficulties. In a study of 150 interviewed deaf youth at a residential school, 75 children reported being sexually abused, 19 reported being victims of incest, and 3 reported both physical and sexual abuse.[9] We identified, in our study, 3 cases of CSA with mental retardation.

All reliable studies conclude that girls experience more sexual abuse than do boys in 78% to 89% of cases.[11] Male children in our studies constitute a large proportion of victims before the age of 16 years.

Although low socioeconomic status is a powerful risk factor for physical abuse and neglect, it has much less impact on CSA. However, a disproportionate number of CSA cases reported to Child Protective Services come from lower socioeconomic classes.[12] In our study, victims coming from economically disadvantaged backgrounds accounted about three quarters of cases.

Parental inadequacy, unavailability, conflict, and a poor parent-child relationship show up most consistently in epidemiological studies[13-15]as risk factors for CSA. In many studies children with alcoholic, drug abusing, or emotionally unstable parents are also at risk, as are those with parents who are punitive or distant.[16, 17] However, many victims of sexual abuse display none of these markers.

Children who experience other forms of victimization are more likely to be the target of sexual victimization.[8, 18]

At the extreme end of the spectrum, sexual abuse includes sexual intercourse or its deviations. Yet all offences that involve sexually touching a child, as well as non-touching offenses and sexual exploitation, are just as harmful and devastating to a child's well-being. Touching sexual offenses include fondling; making a child touch an adult's sexual organs; and penetrating a child's vagina or anus no matter how slight with a penis or any object that doesn't have a valid medical purpose. Non-touching sexual offenses include: engaging in indecent exposure or exhibitionism; exposing children to pornographic material; deliberately exposing a child to the act of sexual intercourse; and masturbating in front of a child. Sexual exploitation can include engaging a child or soliciting a child for the purposes of prostitution; and using a child to film, photograph or model pornography. Physical violence is very rarely used; rather the perpetrator tries to manipulate the child's trust and hide the abuse.[8, 18] However, in our study CSA was associated with physical violence in 21% of cases.

The perpetrators of sexual abuse are overwhelmingly male. Male constituted 100% of the offenders in our study and more than 90% in many studies.[11, 19, 20] Although female perpetrators constitute a small percentage; abuse by female has been mushrooming recently.[21] According to studies, the third of convicted sex offenders were sexually abused as children.[22] Our study and several studies agree that approximately half of offenders are acquaintances.[7, 23] The studies differ more about the percentage who are family members, the range is going from 14% to 47% [18, 19, 24] with 16% in our work. Strangers make up the smallest group of perpetrators ranging from 7% to 25% [6, 11, 25, 26] with 24% in our study. The apparent percentage of extrafamilial perpetrators should not obscure the accurate proportion of intrafamilial abuse which tends to be underrepresented among reported cases given the socio-cultural restraints surrounding sexual issues especially in developing countries like Bangladesh.

CSA frequently occurs as repeated episodes that become more invasive with time. Perpetrators usually engage the child in a gradual process of sexualizing the relationship over time.[2] In our study CSA was repeated in 67% of cases: of these, victims were re-abused by the same perpetrator in 78% of cases.

Children rarely disclose sexual abuse immediately after the event.[27, 28] Disclosure tends to be a process rather than a single episode and is often initiated following a physical complaint or a change in behavior. Disclosure was delayed in the majority of cases in our study reaching 24 months in a 12 years old incest case.

The evaluation of children requires special skills and techniques in history taking, forensic interviewing and examination; the examiner may also need to address specific issues related to consent and reporting of child sexual abuse[29, 30]. In practice, clear physical findings of sexual abuse are seldom seen in children, as physical force is rarely involved. Many studies have found that normal and non-specific findings are common in sexually abused pre-pubertal girls[31,32]; clinical examination in our study was normal in 61% of cases. Moreover, in the vast majority of cases the medical examination will neither confirm nor refute an allegation of sexual assault. Clinical examination may reveal physical health consequences[27, 33], that include gastrointestinal disorders (e.g. irritable bowel syndrome, non-ulcer dyspepsia, chronic abdominal pain); gynecological disorders (e.g. chronic pelvic pain, dysmenorrhea, menstrual irregularities) and somatization (attributed to a preoccupation with bodily processes). Other serious consequences include pregnancy and sexually transmitted infections (STIs), pregnancy was noted in 5 cases in our study, three of whom were incest victims. A study of factors associated with teenage pregnancy[34], found that forced sexual initiation was the third most strongly related factor, after frequency of intercourse and use of modern contraceptives. An organization for teenage mothers in Costa Rica reported that 95% of its clients under the age of 15 had been victims of incest.[35] The prevalence of STIs in pediatric victims of sexual abuse depends on the type of abusive exposure, genital symptoms, prior consensual sexual activity in adolescents, and the regional prevalence of STIs in the adults.[36] Gellert et al[37] evaluated the risk for HIV seroconversion among children with a history of sexual abuse and found that 28 0.4% were HIV seropositive. Systematic screening for STIs in our study revealed HIV in one case. Sexual abuse is the most worrisome form of HPV transmission. One of our patients contracted HPV anal warts.

A variety of adult psychiatric conditions have been clinically associated with CSA. These include the disorders of major depression, borderline personality disorder, somatization disorder, substance abuse disorders, posttraumatic stress disorder (PTSD), dissociative identity disorder, and bulimia nervosa.[6] This apparent diversity can be explained in part by the heterogeneity of CSA experiences, the complexity of the confounds among abuse severity variables, and a host of moderating and mediating constitutional and environmental variables together with important individual differences in coping strategies that may come into play at different points in development in any given case.[38] Some studies suggest that penetration, the duration and frequency of the abuse, force, the relationship of the perpetrator to the child, and maternal support affected the degree of symptomatology.[39] For instance, survivors of incest may have particularly severe problems, especially if the offender was a father or stepfather. 53% of adult survivors of incest said the abuse caused "some" or "great" long-term psychological effects[22]; in our study, incest resulted in three cases of attempted suicide. Numerous studies have found that sexually abused children exhibited more sexualized behaviors than various comparison groups, including non-abused psychiatric patients.[39-41] These include such activities as kissing with one's tongue thrust into the other person's mouth, fondling one's own or another person's breasts or genitals, masturbation, and rhythmic pelvic thrusting. Furthermore, a history of CSA, but not a history of physical abuse or neglect, is associated with a significantly increased arrest rate for sex crimes and

prostitution irrespective of gender.[42] Despite the variety of behavioral disorders that was found in our study, initial psychological evaluation showed no symptoms in approximately 22% of our patients, this result was consistent with those of other studies.[39] The limited longitudinal data available, however, suggest that 10% to 20% of asymptomatic children will deteriorate over the next 12 to 18 months, this phenomenon is termed sleeper effects.[6] Thus, further studies will be needed to find out the long-term effects on our patients.

Includes STIs screening and treatment; decisions about STI testing in children should be made on a case-by-case basis. If testing is warranted, age-appropriate diagnostic tests should be used. Presumptive treatment of children for STIs is not generally recommended.[2] STIs screening in our study was systematic, it was repeated when the abuse occurred recently because STI cultures were likely to be negative.

An array of treatment protocols has been offered in the literature providing care for the victims, their families and also the perpetrators. Many studies showed that sexually abused children improved significantly over time.[6] A number of symptoms, especially aggression and sexualized behavior, remain largely resistant to these approaches, however.

Every community has its own set of laws governing how, and to whom, a report regarding suspicion of child sexual abuse should be made. Typically the reporting law leaves the final determination as to whether or not abuse occurred to the investigators, not the reporters.[43] Bangladesh like most communities also has a mandatory reporting structure for professionals working with children.

Provide support to the victim and to those caring him. This may be required even if the child itself is not assessed as needing therapy.

Is strongly recommended to ensure that the appropriate counseling referrals have been made and that there is adequate support for the child and family.

**Conclusion:**

Child sexual abuse has substantial consequences not only for the affected persons, but also for society as a whole, and these can no longer be ignored. This Urgent situation has now been recognized in Bangladesh which is responding with a diverse range of prevention and intervention programs. However,the serious shortcomings in data tend to impede the effectiveness of such measures. Thus, improved studies are required in order to provide data on the accurate magnitude of the CSA, on its distribution and factors that point to vulnerability.

**References:**


1. Alexander Butchart, Harvey AP, Tilman Fürniss, et al. Preventing child maltreatment: a guide to taking action and generating evidence. Geneva, World Health Organization. 2006. http://whqlibdoc.who.int/publications/2006/9241594365_eng.pdf.Accessed 23 April, 2014.
2. World Health organization. GUIDELINES FOR MEDICO-LEGAL CARE FOR VICTIMS OF SEXUAL VIOLENCE: Child sexual abuse (2003). pp: 75-93. http://whqlibdoc.who.int/publications/2004/924154628x.pdf. Accessed 10 March, 2014.
3. UNICEF. Opinions of children of Bangladesh on corporal punishment: children's opinion poll 2008, Dhaka: Ministry of Women and Children Affairs,Government of Bangladesh, Dhaka: UNICEF; 2009.
4. World Health Organization, Crimes against Children Research Center. Promoting Research to Prevent Child Maltreatment: Summary report of the XIXth ISPCAN International Congress on Child Abuse and Neglect. 2012. http://www.who.int/violence_injury_prevention/violence/child/ispscan_report_june201pdf. Accessed 24 April, 2014.
5. Finkelhor David. Child sexual abuse: New theory & research. NY: The Free Press. 1984; Vol 77(2): pp xii.
6. Frank WP. Ten-Year Research Update Review: Child Sexual Abuse. J AM ACAD/ Child adolesc psychiatry. 2003; 42(3): 269-278.
7. Bolen RM, Scannapieco M. Prevalence of child sexual abuse: A corrective metanalysis. Social Service Review. 1999; 73(3):281-313.
8. Finkelhor David, Heather Hammer, Sedlak AJ. Sexually assaulted children: National estimates and characteristics, in OJJDP: Juvenile Justice Bulletin. US Department of Justice. 2008. https://www.ncjrs.gov/pdffiles1/ojjdp/214383.pdf. Accessed 21 April, 2014.
9. Sullivan PM, Vernon McCay, Scanlan JM. Sexual Abuse of Deaf Youth. Am Ann Deaf. 1987; 132(4): 256-262.
10. Westcott Hl, Jones DP. Annotation: the abuse of disabled children. J Child Psychol Psychiatry. 1999; 40(4):497-506.
11. Synder HN. Sexual assault of young children as reported to law enforcement: Victim, incident and offender characteristics: a NIBRS Statistical Report. USA Department of Justice. 2000. http://www.bjs.gov/content/pub/pdf/saycrle.pdf. Accessed 20 April, 2014.
12. Finkelhor David. Epidemiological factors in the clinical identification of child sexual abuse. Child Abuse Negl. 1993; 17(1):67-70.
13. Moore KA, Nord CW, Peterson JL. Nonvoluntary sexual activity among adolescents. Family Planning Perspectives. 1989; 21(3):110-14.
14. Finkelhor, Sharon Araji. High-risk children. A sourcebook on child sexual abuse. 1986. Beverly Hills. Sage publications.
15. Paveza GJ. Risk factors in father-daughter child sexual abuse: A case control study. Journal of interpersonal violence. 1988; 3(3): 290-306.

16. Christopher Bagley, Richard Ramsay. Sexual abuse in childhood: Psychosocial outcomes and implications for social work practice. Journal of Social Work and Human Sexuality. 1986; 4(1):33-47.

17. Collings Steven. Childhood sexual abuse in a sample of South Africa university males: Prevalence and risk factors. South Afr J Psychol. 1991; 21(3): 153-158.

18. Kilpatrick DG, Acierno Ron, Saunders BE, et al. Risk factors for adolescent substance abuse and dependence: Data from a national sample. J Consult Clin Psychol. 2000; 68(1):19-30.

19. Jones LM, Finkelhor David, Kathy Kopiec. Why is sexual abuse declining? A survey of state child protection administrators. Child Abuse & Neglect. 2001; 25(9): 1139-1158.

20. Crewdson John. By Silence Betrayed: Sexual Abuse of Children in America.1st edition. 1988. NY. Little Brown & Co (T).

21. Allen CM. Women and men who sexually abuse children: A comparative study. Orwell, VT: Safer Society Press, 1984.

22. Groth NA. Men Who Rape: The Psychology of the Offender. 1979. NY. Plenum Press.

23. Wonderlich SA, Wilsnack RW, Wilsnack SC et al. Childhood sexual abuse and bulimic behavior in a nationally representative sample. Am J Public Health. 1996; 86(8): 1082-1086.

24. Briere John, Elliott DM. Prevalence and psychological sequelae of self-reported childhood physical and sexual abuse in a general population sample of men and women. Child Abuse Negl. 2003; 27(10): 1205-1222.

25. Finkelhor David, Jennifer Dziuba-Leatherman. Children as victims of violence: A national survey. Pediatrics. 1994; 94(4): 413-420.

26. Hanson RF, Kievit LW, Saunders BE et al. Correlates of adolescent reports of sexual assault: Findings from the National Survey of Adolescents. Child Maltreat. 2003; 8(4): 261-272.

27. Lori Frasier. Medical Evaluation of Child Sexual Abuse: A Practical Guide, 2nd ed. Arch PediatrAdolesc Med. 2002; 156(6): 627.

28. Summit RC. The Child Sexual Abuse Accommodation Syndrome. Child Abuse and neglect. 1983; 7(2):177- 193.

29. Finkel MA, Giardino AP. Medical evaluation of child sexual abuse: A practical guide, 2nd ed. 2002. Thousand Oaks, Calif. Sage Publications.

30. David Muram. The medical evaluation of sexually abused children. Journal of Pediatric and Adolescent Gynecology. 2003; 16(1): 5-14.

31. Emans SJ. Physical examination of the child and adolescent. In: Heger Astrid, Emans SJ, Muram David, eds. Evaluation of the sexual abused child, 2nd ed. NY. Oxford University Press, 2000: 57-78.

32. Berenson AB, Chacko MR, Wiemann CM et al. A case-control study of anatomic changes resulting from sexual abuse. Am J Obstet Gynecol. 2000; 182(4): 820-834.

33. Heger Astrid. Making the diagnosis of sexual abuse: ten years later. In: Heger Astrid, Emans SJ, Muram David, eds. Evaluation of the sexually abused child. NY. Oxford University Press. 2000: 1-10.

34. Jewkes R, Vundule C, Maforah F et al. Relationship dynamics and teenage pregnancy in South Africa. So Sci Med. 2001; 5 (5): 733-744.

35. Laura LO, Jessica RS. Gender violence interdisciplinary perspectives. NY. New York University Press. 1997. p:235.

36. Bechtel Kirsten. Sexual abuse and sexually transmitted infections in children and adolescents. Current Opinion in Pediatrics. 2010; 22 (1): 94-99.

37. Gellert GA, Durfee MJ, Berkowitz CD, et al. Situational and sociodemographic characteristics of children infected with human immunodeficiency virus from pediatric sexual abuse. Pediatrics. 1993; 91(1): 39-44.

38. Mark Chaffin, Jeffrey NW, Roscoe Dykman. School age children's coping with sexual abuse: abuse stresses and symptoms associated with four coping strategies. Child Abuse Negl.1997; 21(2): 227-240.

39. Kendall-Tackett KA, Williams LM, Finkelhor David. Impact of sexual abuse on children: A review and synthesis of recent empirical studies. Psychological Bulletin. 1993; 113(1): 164-180.

40. Paolucci Elizabeth, Genuis Mark, Violato Claudio. A meta-analysis of the published research on the effects of child sexual abuse. J Psychol. 2001; 135(1):17-36.

41. Friedrich WN, Fisher JL, Dittner CA et al. Child sexual behavior inventory: normative, psychiatric, and sexual abuse comparisons. Child Maltreat. 2001; 6(1): 37-49.

42. WidomCS, Ames MA. Criminal consequences of childhood sexual victimization. Child Abuse Negl. 1994; 18(4): 303-318.

43. Kalichman SC. Mandated reporting of suspected child abuse: ethics, law and policy. 1933. Washington DC. Amer Psychological Assn.

ORIGINAL ARTICLE

# In vitro study of antimicrobial activity of aqueous extracts of *Cinnamomum zeylanicum* bark against *Staphylococcus aureus* and *Escherichia coli*

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***Abstract***

***Introduction: Various spices which we use in food are obtained from nature which show antioxidant, antimicrobial, anti-inflammatory properties etc. Various studies have been conducted in order to determine the effects of oils and extracts obtained from natural sources.The antimicrobial activities are also studied in the point from the extracts obtained from natural sources.The aim of this study was to investigate the antibacterial activity of aqueous extracts of bark of Cinnamon (Cinnamomum zeylanicum) against two food spoilage bacteria, Gram negative Escherichia coli and Gram positive Staphylococcus aureus.***

***Methods: The in vitro antibacterial activity was performed by disc diffusion method. Different concentrations of aqueous extracts were prepared by using distilled water.***

***Results: The plant extracts were more active against Gram-positive bacteria than against Gram-negative bacteria. The maximum zones of inhibition at 100% concentrations were 24mm against Staphylococcus aureus and 25mm against Escherichia coli. A standard antibiotic Amikacin was also used to determine ZOI and it was compared with that result of aqueous extracts.***

***Conclusion: The results obtained in the present study suggest that the aqueous extracts of bark of Cinnamomum zeylanicum showed strong antibacterial activity against both test organisms.Thus cinnamon revealed a significant scope to develop a novel broad spectrum antibacterial herbal formulation.***

***Key words: Cinnamon, Cinnamomum zeylanicum, Antibacterial activity, Staphylococcus aureus, Escherichia. Coli, Disc diffusion, Aqueous extracts, Zone of inhibition.***

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**Introduction:**

Medicinal plants are the source of great economic value all over the world. Nature has bestowed on us a very rich botanical wealth and a large number of diverse types of plants grow in different parts of the country. Bangladesh is rich in all the 3 levels of biodiversity, namely species diversity, genetic diversity and habitat diversity and the people of this country are very much habituated with the use of various types of herbs for their treatment purpose in many occasions.[1] The natural products are found to be more effective with least side effects as compared to commercial antibiotics, so that they are used as an alternate remedy for treatment of various infections.[2]

Cinnamon is a spice obtained from the inner bark of several trees from the genus *Cinnamomum* that is used in both

sweet and savoury foods. Cinnamon grows best in almost pure sand; it prefers a sheltered place, heavy moisture, and warm, unvarying temperature. The tree usually grows up to 30 feet high, has thick outer bark, and strong branches. The top sides of the leaves are shiny & emerald green in colour. The flowers are small in panicles; the fruit is an oval berry which is bluish when ripe. The cinnamon root bark smells like cinnamon and tastes like camphor, which it yields on distillation.[3]

Cinnamon has been used in food preparations and in traditional medicine by the Egyptians and the Chinese since ancient times.[4] In addition, this spice has been found to have strong antibacterial, antipyretic[5] and anti-inflammatory properties, which play an important role in tissue repair. The bark of cinnamon has been used as a spice and to make tea and also as an herbal remedy for the treatment of common colds, cardio-vascular diseases and chronic gastrointestinal and gynecological disorders in oriental herbal medicine.[6] Cinnamon has likewise been used for treating sore throats, cough, indigestion, abdominal cramps, intestinal spasms, nausea, flatulence and diarrohea. Moreover, it has been found that cinnamon slows down food spoilage and displays antifungal properties. It also clears up urinary tract infection, diabetes etc.[7] The present study was undertaken in order to evaluate the in-vitro antibacterial activity of cinnamon bark extract.

**Methods:**

**Plant Material:** The spice cinnamon barks (*Cinnamomum zeylanicum*) were purchased from local market of Charpara, Mymensingh, Bangladesh in February 2018. The spices were botanically identified.

**Preparation of Extract**: Fresh cinnamon barks were cleaned with deionized water and dried first in sunlight for two days and then in hot air oven at 40°C for 1 day. Finally the dried materials were pulverized into fine powdered substance by a grinder. After weighing with the electric balance 100 grams powder of cinnamon were taken in measuring conical flask and 200ml of distilled water was added. The flask was closed by foil paper and put on dark place for five days. The aqueous extract was then filtered by passing through Whatman No.1 filter paper and then concentrated under vacuum at 40°C by using a rotary evaporator. Thus the extract produced had 100 ml volume and 1g/ml or 100% concentration and it was used as Stock solution. This extract was stored in refrigerator at 4°C in small and sterile plastic bottles. Extract of the spice were further diluted to make different concentrations such as 80%,60%,40%, 20% and 10% by mixing with appropriate volumes of distilled water.

**Tested Bacterial strains:** Two bacterial strains *Staphylococcus aureus* (ATCC 25923) and *Escherichia coli* (ATCC 25922) were used in study. Pure cultures of these bacteria were obtained from the Department of Microbiology, Mymensingh Medical College, Mymensingh.

**Maintenance of bacterial culture and inoculum preparation:** Pure cultures were refreshed and maintained on nutrient agar slants and plates on regular basis. The cultures were streaked on sterile nutrient agar plates and kept in incubator for 24 hours at 37°C and stored at 4 °C. Bacterial cultures were refreshed after every 1 to 2 weeks to avoid contamination. Inoculum was prepared by growing the pure bacterial culture in nutrient broth over night at 37°C.

**Antibacterial activity testing using disc diffusion method:** Filter paper disc of 6mm diameter using Whatman no. 1 filter paper was prepared and sterilized. The test microorganisms were transferred from nutrient broth to sterile Mueller Hinton agar plates with the help of sterile cotton swabs. Using an ethanol dipped and flamed forceps the discs were aseptically placed over the Mueller Hinton agar plates seeded with the test microorganisms. Then with the help of micropipette 10 µl of 100%, 80%, 60%, 40%, 20% and 10% concentrations of aqueous extracts were transferred to different disc aseptically. Plates were incubated at 37°C for 24 hours. 10 µl of 95 % ethanol was added in sterile filter paper disc as negative control as because in this trace amount it has negligible antibacterial activity to suppress the growth of microorganisms in culture media. After 24 hours the results were recorded. The antibacterial activity results were expressed in terms of the diameter of zone of inhibition (ZOI) and <9mm zone was considered as inactive; 9-12mm as partially active; while13-18mm as active and >18mm as very active[2].

**Testing antimicrobial activity of a standard antibiotic:** The test microorganisms i.e. *Staphylococcus aureus* and *Escherichia coli* were also tested for their activity against Amikacin (inj.500mg) by disc diffusion method.

**Results:**

In this research study bark of cinnamon (*Cinnamomum zeylanicum*) were found effective against the test bacterial strains. The maximum ZOI at 100% concentration was shown against *Escherichia coli* (25mm). But *Staphylococcus aureus* started showing definite activity from 20% conc. whereas *Escherichia coli* from 80% conc.The diameter of ZOI obtained against aqueous extract at 100% concentration by disk diffusion method was also compared to those obtained against a standard antibiotic Amikacin as shown in table-III. Aqueous extract produced a wider zone of inhibition as compared to Amikacin for

both *Staphylococcus aureus* (24mm) and *E.coli* (25mm). Amikacin was effective against both *Staphylococcus aureus* and *E.coli* forming zones of 17mm and 16mm, respectively.

**Table I**

*Antibacterial activity of different concentrations of ACE measured in Zone of Inhibition by disk diffusion method.*

| Concentrations of ACE solutions in % | Zone of Inhibition (ZOI) in mm | |
|---|---|---|
| | *Staphylococcus aureus* | *Escherichia coli* |
| 10 | 08 | 06 |
| **20** | **15** | 06 |
| 40 | 16 | 06 |
| 60 | 20 | 08 |
| **80** | 22 | **18** |
| 100 | 24 | 25 |
| Control | 06 | 06 |

**Table-II**

*Antibacterial activity of Amikacin measured in Zone of Inhibition by disk diffusion method.*

| | Zone of Inhibition (ZOI) in mm | |
|---|---|---|
| | *Staphylococcus aureus* | *Escherichia coli* |
| Amikacin (500mg) | 17 | 16 |

**Table-III**

*Comparison of Antibacterial activity of ACE & Amikacin measured in Zone of Inhibition by disk diffusion method.*

| | Zone of Inhibition (ZOI) in mm | |
|---|---|---|
| | *Staphylococcus aureus* | *Escherichia coli* |
| ACE (100%) | 24 | 25 |
| Amikacin (500mg) | 17 | 16 |

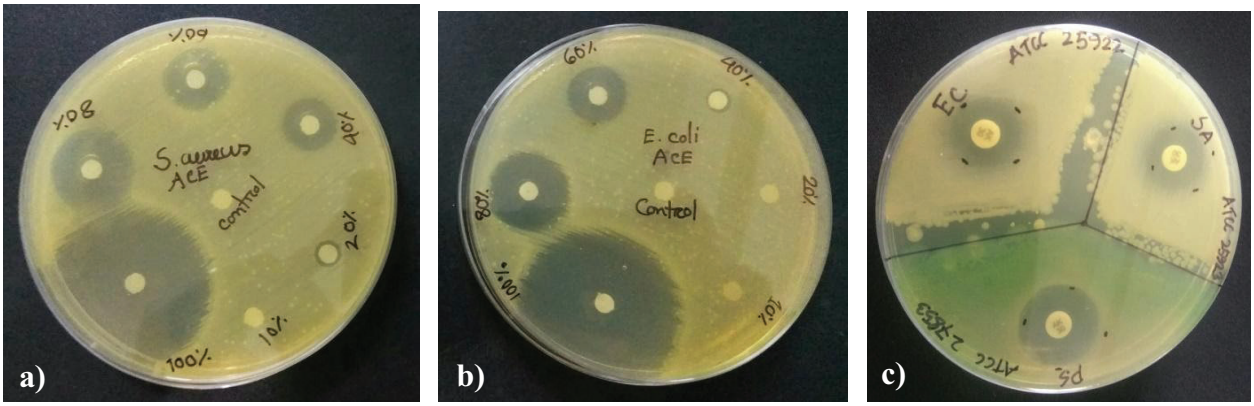


**Fig.-1:** *Testing antibacterial activity of aqueous cinnamon extract against a) Staphylococcus aureus, b) Escherichia coli and c) testing antibacterial activity of Amikacin against above organisms.*

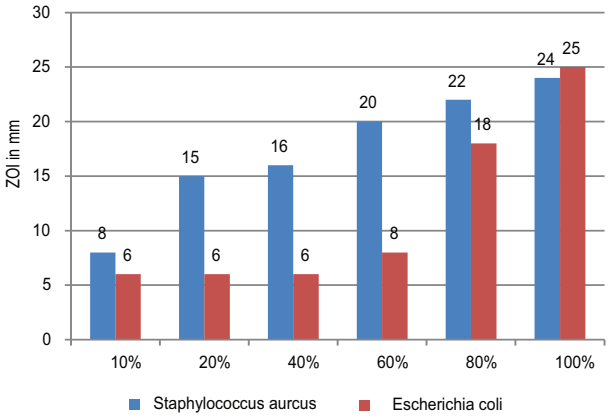


**Fig.-2:** *Multiple bar diagram showing ZOI at different concentration of ACE againstStaphylococcus aureus Escherichia coli*

**Discussion:**

Aqueous extract of cinnamon bark were found active against both *Staphylococcus aureus* and *E. coli*. The extract showed relatively better zones of inhibition against *Staphylococcus aureus* than *E.coli* and when compared with Amikacin it produced larger zone than Amikacin.

Aqueous extract produced zone of inhibition of 08mm,15mm,16mm,20mm,22mm and 24mm against *Staphylococcus aureus* and 06mm,06mm, 06mm, 08mm, 18mm and 25mm against *E. coli* at 10%,20%,40%,60%,80% and 100% conc. respectively (table-I). Amikacin produced zone of inhibition of 17mm against *Staphylococcus aureus* and 16mm against *E. coli*. The findings agree with the work of Zainab A. Al-dhaher (2008).[11] In that study the antibacterial activity of aqueous extract of cinnamon bark

and clove were investigated against *Staphylococcus aureus* by Agar diffusion technique. The ZOI of aqueous extract of cinnamon were 17mm, 15.5mm, 13mm, 8mm and 0mm at 70%, 60%, 40%, 20% and 10% concentration. In this study they were 22mm, 20mm, 16mm, 15mm and 8mm at 80%, 60%, 40%, 20% and 10% conc. respectively. There is quite similarity with this study except at 20% conc. He also stated that Fan et. al (2001), Yuste et.al (2006) also found similar antibacterial activity in their studies.

Sana Mukhtar and Ifra Ghori in 2012 investigated the antibacterial activity of aqueous and ethanolic cinnamon bark extract against *E.coli* ATCC 25922 by disc diffusion method.[2] This study showed that both aqueous and ethanolic extracts were active against gram negative bacteria *E.coli* but ethanolic extracts comparatively showed better results which were similar with this study. ZOI were (9.3 ± 0.38) mm at 60%, (10 ± 0.40) mm at 80% and (10.3 ± 0.41) mm at 100% conc. of aqueous extract which is almost similar with this study result.

This study results were also in consistent with study conducted by Madhumita and C.Ramalingam (2011).[12] They proved that aqueous bark extract of *Cinnamomum verum* has antibacterial activity against *Staphylococcus aureus,Bacillus cereus, Enterococcus fecalis* and *Escherichia coli, Proteus mirabilis*. For *Staphylococcus aureus* the diameter of ZOI were between 0 to 16mm and for *E.coli* between 0mm to 17.5mm. According to that study the sensitivity of aqueous extract against bacteria was *E.coli* > *S.aureus*. But according to this study *S.aureus* was more active and it begins to be active from 20% conc. whereas *E.coli* shows activity at high conc. from 80% and 100%. Odhav et al. (2002), suggested that the mechanism of antibacterial action of spices involve the hydrophobic and hydrogen bonding of phenolic compounds to membrane proteins, membrane disruption and destruction of electron transport systems and cell wall disruption. The antimicrobial activity of aqueous extracts could be due to anionic components such as thiocyanate, nitrate, chlorides and sulphates in addition to many other compounds naturally present in plants (Darout , 2000).The antibacterial activity of cinnamon might be due to the presence of cinnamaldehyde compound which inhibits the amino acid decarboxylation activity in the cell which leads to energy deprivation and microbial cell death (Wendakoon and Sakaguchi, 1995).[2]

**Conclusion:**

The aqueous extracts of *Cinnamomum zeylanicum* were found to be effective against two important food spoilage bacteria like *E.coli* and *Staphylococcus aureus*. This is an absolutely in vitro study. Whether the extract of cinnamon does act as an antimicrobial agent in using particularly during infection- it is yet to be studied by MIC estimation process.Furthermore the chemical compound of extract which is thought to have antimicrobial activity has yet to be isolated and identified.So further extensive studies are needed in order to find out the rational use of cinnamon as an antimicrobial agent among the patients suffering from infection particularly with *E.coli* and *Staphylococcus aureus.*

From the above studies we can come to the conclusion that the extract of cinnamon could be used in food preservation to prevent spoilage of food caused by *E.coli* and *S. aureus* as because cinnamon is found safer when consumed along food.

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**References**:

1. Joshi B, Lekhak S, Sharma A. Antibacterial Property of Different Medicinal Plants: *Ocimum sanctum, Cinnamomum zeylanicum, Xanthoxylum armatum* and *Origanum majorana.* Kathmandu University Journal Of Science, Engineering And Technology 2009 ; 5(1):143- 150.
2. Mukhtar S, Ghori I. Antibacterial activity of aqueous and ethanolic extracts of garlic, cinnamon and turmeric against *Escherichia coli* ATCC 25922 and *Bacillus subtilis* DSM 3256. International Journal of Applied Biology and Pharmaceutical Technology 2012 ; 3(2):131-136.
3. Tomar RS, Shrivastava V. In- Vitro efficacy evaluation of ethanolic extract of Cinnamomum zeylanicum (Cinnamon) as potential antimicrobial agent against clinical isolates. International Research Journal of Pharmaceutical Sciences and Research 2015; 6(5): 2201-2204.
4. Elshafie MM, Nawar IA, Algamal MA, Mohammad AS. Evaluation of the biological effects for adding cinnamon volatile oil and TBHQ as antioxidant on fat's lipid profiles. Asian Journal of Plant Science 2012; 11: 100-108.
5. Lu J, Zhung K, Nam S, Anderson RA, Jove R, Wen W. Novel angiogenesis inhibitory activity in cinnamon extract blocks VEGFR2 kinase and downstream signaling. Carcinogenesis 2010 ; 31: 481-488.
6. Hong JW, Yang GE, Kim YB, Eom SH, Lew JH, Kang H. Anti-inflammatory activity of cinnamon water extraction in vivo and in vitro LPS –induced models. BMC Complement Alternative Medicine 2012; 12: 237.

7. Hamidpour R, Hamidpour M, Hamidpour S, Shahlari M. Cinnamon from the selection of traditional applications to its novel effects on the inhibition of angiogenesis in cancer cells and prevention of Alzheimer's diseases and a series of functions such as antioxidant, anticholesterol, antidiabetes, antibacterial, antifungal, nematicidal, acaricidal and repellent activities.Journal of Traditional and Complementary Medicine 2015; 5:66-70.

8. Elahi RK. The effect of cinnamon on a dog's heart performance by focusing on the korotkoff sound. Journal of Animal Veterinary Advances 2012; 11: 3604-3608.

9. Usha M, Ragini S, Naqvi S.M.A. Antibacterial Activity of Acetone and Ethanol Extracts of Cinnamon (*Cinnamomum zeylanicum*) and Ajowan (*Trachyspermum ammi)* on four Food Spoilage Bacteria. International Research Journal of Biological Sciences 2012;1(4):7-11.

10. Odhav B, Juglal S, Govinden R. Spices oils for the control of co-occurring mycotoxins producing fungi. Journal of Food Production 2002; 65: 683-687.

11. Al-dhaher ZA. The antibacterial activity of aqueous extract of cinnamon and clove against *Staphylococcus aureus*. Journal of Al-Nahrain University 2008;11(2) :131-135.

12. Rahshit M, Ramalingam C. In-vitro antibacterial and antioxidant activity *of Cinnamomum verum* (Cinnamon) aqueous bark extract in reference to its total phenol content as natural preservative of food. International Journal of Biology and Biotechnology 2011;.8(4): 529-537.

REVIEW ARTICLE

# Irritable Bowel Syndrome: A Clinical Review

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*Abstract*

***Irritable bowel syndrome (IBS) is a chronic gastrointestinal disorder characterized by abdominal pain and altered bowel habits in the absence of any organic cause. The prevalence varies from country to country ranging from 10 to 15 percent and younger women are more sufferer. Many factors have been proposed among which disorder in gastric motility, visceral hypersensitivity, intestinal inflammation and infection, alteration in gut flora, food sensitivity, genetic predisposition and psychosocial dysfunction are remarkable. Rome IV criteria is commonly used for diagnosis of IBS. Subtypes of IBS are recognized based on the patient's reported predominant bowel habit. Investigations are done to exclude other differentials. Treatment includes lifestyle modification, dietary restrictions of certain foods, adjunctive pharmacological therapy with antispasmodics, antidepressants, antibiotics, probiotics and behavior modifications.***

***Keywords: Irritable bowel syndrome, Bristol Stool Form Scale, Rome IV criteria, diarrhoea, constipation, abdominal pain.***

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**Introduction:**

Irritable bowel syndrome (IBS) is a chronic gastrointestinal disorder characterized by abdominal pain and altered bowel habits in the absence of any organic cause.[1] The main cause of IBS is not entirely understood as various factors play a key role in its pathophysiology. There is no specific diagnostic test and it is diagnosed through some criteria and exclusion of other diseases. It negatively affects quality of life and work productivity. So accurate diagnosis of IBS is important to minimize unnecessary invasive investigations and to reduce social and economic effects of the disease.

This clinical review covers the epidemiology, pathophysiology, diagnosis and management of IBS.

**Methods:**

Evidence to support this review was obtained from searches from MEDLINE, PUBMED, Google scholar, Science direct, Cochrane databases for the terms *pathophysiology, etiology, pathogenesis, diagnosis, irritable bowel syndrome* and *IBS* from January 2000 to December 2019. The range was expanded from January 1978 to December 2019 for *IBS, diet, treatment, therapy*. A total 168 articles were found among which 36 articles were selected for inclusion.

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**Epidemiology**

**Prevalence -**

The prevalence of IBS varies among countries ranging from 10 to 15 percent. The prevalence is 25 percent higher in those aged less than 50 years as compared with those who were older. Women are more sufferer and likely to have constipation predominant IBS as compared with men.[2]

**Associated conditions -**

IBS is associated with other conditions including chronic fatigue syndrome, fibromyalgia, non-cardiac chest pain, gastroesophageal reflux disease, functional dyspepsia, and psychiatric disorders including anxiety, somatization and major depression.[3]

**Pathophysiology**

The pathophysiology of IBS is unclear though multiple factors has been proposed -

**Gastrointestinal motility-**

Some sort of motor abnormalities are observed in some IBS patients including increased frequency and irregularity of luminal contractions, prolonged transit time in

constipation-predominant IBS, and an exaggerated motor response to cholecystokinin and meal ingestion in diarrhea-predominant IBS.[4]

**Visceral hypersensitivity-**

Visceral hypersensitivity is observed frequently in IBS patients which may result from stimulation of various gut wall receptors triggered by bowel distention or bloating. These receptors transmit signals via afferent neural pathways to the dorsal horn of the spinal cord and ultimately to the brain. It is not clear whether this heightened sensitivity is mediated by the local GI nervous system, by central modulation from the brain, or by combination of the two.[5]

**Intestinal inflammation-**

Immunohistologic investigation has revealed mucosal immune system activation characterized by alterations in particular immune cells and markers in some patients with IBS. Release of chemical mediators (nitric oxide, histamine and proteases) by increased colonic infiltration with lymphocytes and mast cells stimulating the enteric nervous system lead to abnormal motor and visceral responses within the intestine.[5]

**Post infectious-**

Some patients give a history of an acute diarrheal illness (bacterial, protozoan, helminth infections, and viral infections) preceding the onset of irritable bowel symptoms.[6] Risk factors for post infectious IBS included young age, prolonged fever, longer duration, anxiety, and depression. Several theories have been proposed for bowel symptoms following acute infection, e.g., bile acid malabsorption, increase in serotonin-containing enteroendocrine cells and use of antibiotics.[7,8]

**Alteration in fecal microflora-**

It has been found that the fecal microbiota in individuals with IBS differ from healthy controls and vary with the predominant symptom. This theory is supported by improvement of symptoms with probiotics in some diarrhea-predominant IBS patients.[9]

**Bacterial Overgrowth-**

There are some conflicting data reporting an association between IBS and small intestinal bacterial overgrowth (SIBO). Some studies demonstrated improvement in symptoms after eradication of the overgrowth evidenced by reduction in abnormal breath hydrogen levels.[10] In addition, constipation predominant IBS patients exhibited increased methane production, a gas by product of intestinal bacteria.[11]

**Food Sensitivity -**

Multiple factors have been considered to contribute to food sensitivity in patients with IBS:

*Food allergy* - IBS patients show increased number of positive food skin-prick tests compared with controls.[12]

*Carbohydrate malabsorption* - Fermentable oligo-, di-, and monosaccharides and polyols (FODMAPs) in patients with IBS produce symptoms and increase intestinal permeability and possibly inflammation after they are fermented in the distal small and large bowel.[13]

Fructose and lactose intolerance may cause gastrointestinal (GI) symptoms such as flatus, pain, bloating, belching and altered bowel habits. Dietary restriction of fructose and lactose have shown some benefit in alleviation of these symptoms.[14]

*Gluten sensitivity* - Several studies suggest some overlap between celiac disease and IBS.[15] So steps should be taken to confirm the absence of celiac disease prior to making a diagnosis of IBS.

**Genetics -**

Some genetic predisposition may play a role in some patients with IBS.[16]

**Psychosocial Dysfunction -**

Psychosocial factors may influence the expression of IBS.[17] Studies showed increased stress, anxiety, depression, phobias, somatization and history of abuse in patients with IBS compared to control.

**Clinical manifestations**

Chronic abdominal pain -

The pain is usually described as a cramping sensation in abdomen with variable intensity and periodic exacerbations with wide variety in location and character. Defecation may improve or worsen the pain.[18] Some patients report abdominal bloating and increased gas production in the form of flatulence or belching. Psychological stress and some meals may exacerbate the pain.

Altered bowel habits -

It includes diarrhea, constipation, alternating diarrhea and constipation, or normal bowel habits alternating with either diarrhea and/or constipation.

*Diarrhea* - Diarrhea is usually characterized by frequent loose stools of small to moderate volume occurring during waking hours, most often in the morning or after meals. It may be preceded by cramping abdominal pain, urgency with a sense of incomplete evacuation or tenesmus. About 50% of all patients complain of discharge of mucus with stools.[19]

*Constipation* — Stools are often hard and pellet-shaped. Patients may experience tenesmus even in empty rectum.

**Diagnosis**

**Diagnostic criteria -** The most widely used diagnostic criteria are the Rome IV criteria.

- **Rome IV criteria for IBS** – According to the Rome IV criteria, IBS is defined as recurrent abdominal pain, on average, at least one day per week in the last three months, associated with two or more of the following criteria:[18]
  - Related to defecation
  - Associated with a change in stool frequency
  - Associated with a change in stool form (appearance)
- **IBS subtypes** – Subtypes of IBS are recognized based on the patient's reported predominant bowel habit on days with abnormal bowel movements. The Bristol stool form scale (BSFS) should be used to record stool consistency.

**IBS subtypes are defined for clinical practice as follows:**

- **IBS with predominant constipation(IBS-C)** - Patient reports that abnormal bowel movements are usually constipation (type 1 and 2 in the BSFS)
- **IBS with predominant diarrhea (IBS-D)** - Patient reports that abnormal bowel movements are usually diarrhea (type 6 and 7 in the BSFS)
- **IBS with mixed bowel habits (IBS-M)** - Patient reports that abnormal bowel movements are usually both constipation and diarrhea (more than one-fourth of all the abnormal bowel movements were constipation and more than one-fourth were diarrhea)
- **IBS unclassified (IBS-U)** - Patients who meet diagnostic criteria for IBS but cannot be accurately categorized into one of the other three subtypes
- **Other criteria** - The Manning criteria include relief of pain with bowel movements, looser and more frequent stools with onset of pain, passage of mucus, and a sense of incomplete emptying.[19] No symptom-based criteria have ideal accuracy for diagnosing IBS.

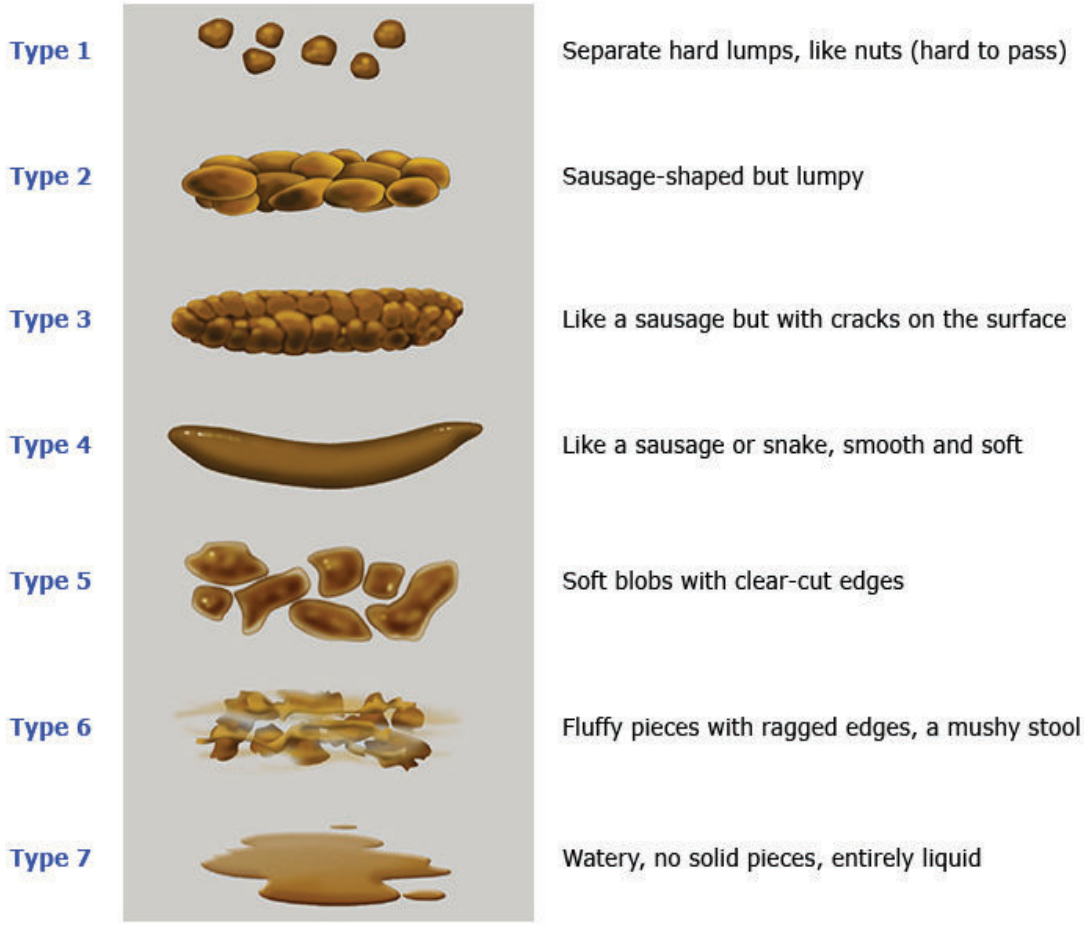


**Fig.-1:** *The Bristol stool form scale (BSFS)*

**Initial evaluation**

*History and physical examination* — Thorough history including past medical, medication and family history is required to exclude other disorders that may produce similar symptoms. The physical examination is usually normal in patients with IBS.

*Laboratory investigation* — There is no definitive diagnostic laboratory investigation for IBS. The purpose of investigating is primarily to exclude an alternative diagnosis. A complete blood count is performed in all patients. In patients with diarrhea, fecal calprotectin or lactoferrin, stool testing for giardia and serologic testing for celiac disease should be done.[20] If fecal calprotectin and fecal lactoferrin cannot be performed, C-reactive protein levels should be checked. Other tests are guided by the clinical presentation; such as age-appropriate colorectal cancer screening in all patients, abdominal radiograph to assess for stool accumulation in IBS patients with constipation, etc. Anorectal manometry and balloon expulsion testing should be done to rule out dyssynergic defecation in patients with severe constipation that is refractory to management with dietary changes and osmotic laxative therapy.

Additional evaluation based on the presence of alarm features —

- **Alarm features** – Alarm features include:[21]
  - Age of onset e 50 years
  - Melena or hematochezia
  - Nocturnal diarrhea
  - Progressive abdominal pain
  - Unexplained weight loss
  - Laboratory abnormalities, e.g., iron deficiency anemia, elevated C-reactive protein or positive fecal calprotectin or lactoferrin
  - Family history of IBD or colorectal cancer
- **Patients without alarm features** – Additional investigations are not required beyond the initial evaluation in patients meeting diagnostic criteria for IBS having no alarm features.
- **Patients with alarm features** – In patients with alarm features, additional evaluation are required to exclude other causes of similar symptoms. The evaluation is based on the clinical presentation and usually includes endoscopic evaluation in all patients and imaging in selected cases. In patients with diarrhea, colonoscopy is done to evaluate for the presence of IBD and perform biopsies to exclude microscopic colitis.[22] Abdominal ultrasonography or computed tomography scan is done if there is a clinical suspicion for a structural lesion.

**Disease course**

Most patients with IBS have chronic symptoms that vary in severity over time. In a systematic review, 2 to 5 percent of patients were diagnosed with an alternate gastrointestinal disease over a course of long term follow up. Some patients may experience a change in IBS subtype over time.

**Treatment**

Initial therapy

**Lifestyle modification:** In patients with mild and intermittent symptoms that do not impair quality of life.

***Education and reassurance:*** Patients should be counseled about the chronicity of IBS and no additional risk of malignancy. The clinician should establish realistic therapeutic limitations with expectations and involve the patient in treatment decisions.

***Dietary modification:*** A careful dietary history may reveal patterns of symptoms related to specific foods. Some patients with IBS may benefit from exclusion of gas-producing foods (eg, beans, onions, celery, carrots, raisins, bananas, apricots, prunes, Brussels sprouts, wheat germ, pretzels, and bagels, alcohol, and caffeine);[23] a diet low in FODMAPs; and in selected cases, lactose and gluten avoidance.

Both a low FODMAP diet and a strict traditional IBS diet (regular meal pattern; avoidance of large meals; reduced intake of fat, insoluble fibers, caffeine, and gas-producing foods such as beans, cabbage, and onions) improve IBS symptoms.[24]

***Lactose avoidance —*** Patients with known lactose intolerance should be placed on a lactose-restricted diet. An empiric trial of a lactose-free diet in patients who complain of persistent abdominal bloating despite exclusion of gas-producing foods may be tried.

***Gluten avoidance —*** Gluten has been demonstrated to alter bowel barrier functions in patients with IBS-D, but evidence to support gluten avoidance in patients with IBS has been conflicting.[25]

***Fiber —*** The role of fiber in patients with IBS is controversial, but given the absence of serious side effects and potential benefit, psyllium/ispaghula should be considered in patients with IBS whose predominant symptom is constipation.[26]

***Physical activity* —** Physical activity may improve symptoms in some IBS patients and should be advised.[27]

**Adjunctive pharmacologic therapy**

Patients having impaired quality of life due to moderate to severe symptoms of IBS are treated with pharmacologic agents. Treatment should be based on the predominant symptom and subtype.

**Constipation —** Polyethylene glycol (PEG) is suggested in patients with IBS-C who have failed a trial of soluble fiber (eg, psyllium/ispaghula). If the patient still remains constipated, lubiprostone, linaclotide, or plecanatide can be tried. In women under the age of 65 who fail these agents, a trial with tegaserod is an alternative. Tegaserod reduces abdominal pain in IBS as well as improves constipation. Dyspepsia overlaps with IBS, and tegaserod may provide symptom benefit for dyspepsia.[28]

**Diarrhea —** In diarrhea-prone patients with IBS, the stools are characteristically loose and frequent but of normal total daily volume. In patients with diarrhea-predominant symptoms, antidiarrheals (eg, loperamide)[21,29]are used as initial treatment and bile acid sequestrants (eg, cholestyramine, colestipol, colesevelam)[30] as second-line therapy.

Eluxadoline, an agent that combines a mu-opioid receptor agonist and a delta-opioid receptor antagonist, has also been approved for treatment of IBS-D.[31]

Alosetron, a 5-hydroxytryptamine-3 receptor (5HT-3) antagonist, is approved for the treatment of severe diarrhea-predominant IBS in female patients whose symptoms have lasted for six months and who have failed to respond to all other conventional treatment.

**Abdominal pain and bloating —**

***Antispasmodics* —** In patients with abdominal pain due to IBS, antispasmodics (eg, mebeverine and pinaverine, dicyclomine and hyoscyamine) are used on an as-needed basis. In patients with IBS-C, antispasmodics are initiated only if the abdominal pain persists despite treatment of constipation. The selective inhibition of gastrointestinal smooth muscle by antispasmodics and peppermint oil reduce stimulated colonic motor activity and may be beneficial in patients with postprandial abdominal pain, gas, bloating, and fecal urgency.[32,33]

***Antidepressants* —** Antidepressants have analgesic properties along with their mood improving effects.[21,32] Tricyclic antidepressants (TCAs), via their anticholinergic properties, also slow intestinal transit time, which may provide benefit in diarrhea-predominant IBS,[32] hence should be used cautiously in patients with constipation. Antidepressants should be started at a low dose for the treatment of abdominal pain in IBS. At least three to four weeks of therapy should be attempted before increasing the dose because of their delayed onset of action. Amitriptyline, nortriptyline, and imipramine can be started at a dose of 10 to 25 mg at bedtime. If the patient is intolerant of one TCA, another can be tried.

As there are lack of consistent high-quality evidence demonstrating an improvement in symptoms, SSRIs/SNRIs are not used for the treatment of IBS unless depression acts as a cofactor.

***Antibiotics* —** While antibiotics should not be routinely recommended in all patients with IBS, in patients with moderate to severe IBS without constipation, particularly those with bloating, who have failed to respond to other therapies (eg, a diet low in FODMAPs, antispasmodics, and TCAs), a two-week trial of rifaximin is suggested.[34]

***Probiotics* —** Probiotics are not routinely recommended in patients with IBS. Although they have been associated with an improvement in symptoms, the magnitude of benefit and the most effective species and strain are uncertain.[35]

**Refractory symptoms**

A small subset of patients with irritable bowel syndrome (IBS) has refractory symptoms. Patients with continued symptoms despite adjunctive pharmacologic therapy should be carefully reassessed, paying specific attention to the type of ongoing symptoms, the degree to which symptoms have changed, compliance with medications, and the presence of alarm features that should prompt further evaluation.

**Behavior modification —** Patients with unrelenting symptoms that are associated with psychiatric impairment may benefit from behavioral modification in conjunction with antidepressants.

**Anxiolytics —** The use of anxiolytic agents in patients with IBS should be limited to short-term (less than two weeks) reduction of acute situational anxiety that may be contributing to symptoms. Side effects of anxiolytics include the risk of habituation, rebound withdrawal, and drug interactions. Furthermore, benzodiazepines may lower pain thresholds by stimulating gamma aminobutyric acid (GABA) receptors, thereby decreasing brain serotonin.

**Other therapies —** Other therapies have been evaluated in patients with IBS (eg, herbs, acupuncture, enzyme supplementation, fecal microbiota transplantation, antihistamines (Ebastine) and mast cell stabilizers (Ketotifen) but their role in the treatment of IBS remains uncertain.[36]

**Conclusion:**

IBS remains a significant cause of distress, morbidity and to some extent, disability among people of all ages around the world. As there is no specific investigation to diagnose IBS, it is hoped that novel biomarkers will be invented to aid in accurate diagnosis. The physicians should understand the pathophysiology well and take into mind about the role of dietary, lifestyle and behavioral modifications with or without pharmacological interventions for effective treatment of IBS. The management should be individualized for each patient for an effective result.

**References:**


1. Drossman DA, Camilleri M, Mayer EA, Whitehead WE. AGA technical review on irritable bowel syndrome. Gastroenterology 2002; 123:2108.
2. Lovell RM, Ford AC. Effect of gender on prevalence of irritable bowel syndrome in the community: systematic review and meta-analysis. Am J Gastroenterol 2012; 107:991.
3. Chey WD, Kurlander J, Eswaran S. Irritable bowel syndrome: a clinical review. JAMA 2015; 313:949.
4. Agrawal A, Houghton LA, Reilly B, et al. Bloating and distension in irritable bowel syndrome: the role of gastrointestinal transit. Am J Gastroenterol 2009; 104:1998.
5. Valdez-Morales EE, Overington J, Guerrero-Alba R, et al. Sensitization of peripheral sensory nerves by mediators from colonic biopsies of diarrhea-predominant irritable bowel syndrome patients: a role for PAR2. Am J Gastroenterol 2013; 108:1634.
6. Zanini B, Ricci C, Bandera F, et al. Incidence of post-infectious irritable bowel syndrome and functional intestinal disorders following a water-borne viral gastroenteritis outbreak. Am J Gastroenterol 2012; 107:891.
7. Dunlop SP, Jenkins D, Neal KR, Spiller RC. Relative importance of enterochromaffin cell hyperplasia, anxiety, and depression in postinfectious IBS. Gastroenterology 2003; 125:1651.
8. Törnblom H, Holmvall P, Svenungsson B, Lindberg G. Gastrointestinal symptoms after infectious diarrhea: a five-year follow-up in a Swedish cohort of adults. Clin Gastroenterol Hepatol 2007; 5:461.
9. Jeffery IB, O'Toole PW, Öhman L, et al. An irritable bowel syndrome subtype defined by species-specific alterations in faecal microbiota. Gut 2012; 61:997.
10. Pimentel M, Chow EJ, Lin HC. Eradication of small intestinal bacterial overgrowth reduces symptoms of irritable bowel syndrome. Am J Gastroenterol 2000; 95:3503.
11. Chatterjee S, Park S, Low K, et al. The degree of breath methane production in IBS correlates with the severity of constipation. Am J Gastroenterol 2007; 102:837.
12. Jun DW, Lee OY, Yoon HJ, et al. Food intolerance and skin prick test in treated and untreated irritable bowel syndrome. World J Gastroenterol 2006; 12:2382.
13. Gibson PR, Shepherd SJ. Personal view: food for thought—western lifestyle and susceptibility to Crohn's disease. The FODMAP hypothesis. Aliment Pharmacol Ther 2005; 21:1399.
14. Austin GL, Dalton CB, Hu Y, et al. A very low-carbohydrate diet improves symptoms and quality of life in diarrhea-predominant irritable bowel syndrome. Clin Gastroenterol Hepatol 2009; 7:706.
15. Verdu EF, Armstrong D, Murray JA. Between celiac disease and irritable bowel syndrome: the "no man's land" of gluten sensitivity. Am J Gastroenterol 2009; 104:1587.
16. Saito YA, Petersen GM, Locke GR 3rd, Talley NJ. The genetics of irritable bowel syndrome. Clin Gastroenterol Hepatol 2005; 3:1057.
17. Chang L. The role of stress on physiologic responses and clinical symptoms in irritable bowel syndrome. Gastroenterology 2011; 140:761.
18. Simren M, Palsson OS, Whitehead WE. Update on Rome IV Criteria for Colorectal Disorders: Implications for Clinical Practice. Curr Gastroenterol Rep 2017; 19:15.
19. Manning AP, Thompson WG, Heaton KW, Morris AF. Towards positive diagnosis of the irritable bowel. Br Med J 1978; 2:653.
20. Smalley W, Falck-Ytter C, Carrasco-Labra A, et al. AGA Clinical Practice Guidelines on the Laboratory Evaluation of Functional Diarrhea and Diarrhea-Predominant Irritable Bowel Syndrome in Adults (IBS-D). Gastroenterology 2019; 157:851.
21. American College of Gastroenterology Task Force on Irritable Bowel Syndrome, Brandt LJ, Chey WD, et al. An evidence-based position statement on the management of irritable bowel syndrome. Am J Gastroenterol 2009; 104 Suppl 1:S1.
22. Kamp EJ, Kane JS, Ford AC. Irritable Bowel Syndrome and Microscopic Colitis: A Systematic Review and Meta-analysis. Clin Gastroenterol Hepatol 2016; 14:659.
23. Hasler WL, Owyang C. Irritable bowel syndrome. In: Textbook of Gastroenterology, 4th ed, Yamada T (Ed), JB Lippincott, Philadelphia 2003. p.1817.
24. Böhn L, Störsrud S, Liljebo T, et al. Diet low in FODMAPs reduces symptoms of irritable bowel syndrome as well as traditional dietary advice: a randomized controlled trial. Gastroenterology 2015; 149:1399.
25. Biesiekierski JR, Newnham ED, Irving PM, et al. Gluten causes gastrointestinal symptoms in subjects without celiac disease: a double-blind randomized placebo-controlled trial. Am J Gastroenterol 2011; 106:508.
26. Rao SS, Yu S, Fedewa A. Systematic review: dietary fibre and FODMAP-restricted diet in the management of constipation and irritable bowel syndrome. Aliment Pharmacol Ther 2015; 41:1256.
27. Johannesson E, Simrén M, Strid H, et al. Physical activity improves symptoms in irritable bowel syndrome: a randomized controlled trial. Am J Gastroenterol 2011; 106:915.

28. Vakil N, Laine L, Talley NJ, et al. Tegaserod treatment for dysmotility-like functional dyspepsia: results of two randomized, controlled trials. Am J Gastroenterol 2008; 103:1906.

29. Efskind PS, Bernklev T, Vatn MH. A double-blind placebo-controlled trial with loperamide in irritable bowel syndrome. Scand J Gastroenterol 1996; 31:463.

30. Wedlake L, A'Hern R, Russell D, et al. Systematic review: the prevalence of idiopathic bile acid malabsorption as diagnosed by SeHCAT scanning in patients with diarrhoea-predominant irritable bowel syndrome. Aliment Pharmacol Ther 2009; 30:707.

31. Traynor K. Eluxadoline approved for irritable bowel syndrome with diarrhea. Am J Health Syst Pharm 2015; 72:1078.

32. Ruepert L, Quartero AO, de Wit NJ, et al. Bulking agents, antispasmodics and antidepressants for the treatment of irritable bowel syndrome. Cochrane Database Syst Rev 2011; :CD003460.

33. Merat S, Khalili S, Mostajabi P, et al. The effect of enteric-coated, delayed-release peppermint oil on irritable bowel syndrome. Dig Dis Sci 2010; 55:1385.

34. Pimentel M, Lembo A, Chey WD, et al. Rifaximin therapy for patients with irritable bowel syndrome without constipation. N Engl J Med 2011; 364:22.

35. Moayyedi P, Ford AC, Talley NJ, et al. The efficacy of probiotics in the treatment of irritable bowel syndrome: a systematic review. Gut 2010; 59:325.

36. Klooker TK, Braak B, Koopman KE, et al. The mast cell stabilizer ketotifen decreases visceral hypersensitivity and improves intestinal symptoms in patients with irritable bowel syndrome. Gut 2010; 59:1213.

## CASE SERIES

# Cervical Thymic Duct Cyst: A Rare Cystic Lateral Neck Mass in Children

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*Abstract*

***Introduction: Thymic duct cyst is a very rare differential diagnosis of pediatric lateral cystic neck swellings and often misdiagnosed as either branchial cleft cyst or cystic hygromas.***

***Case series: Three cases of thymic duct cysts in under 20 years of age are being presented here. Pre-operatively these cases were not diagnosed as thymic cyst but histopathological reports confirmed the diagnosis.***

***Conclusion: Due to its rarity, it almost always escapes a correct preoperative diagnosis. Moreover, it is related to some important structures in neck. So, surgeons should aware of this condition in lateral cystic neck mass in children particularly in the first two decades of life. Greater awareness among the pathologists may decrease the misdiagnosis.***

***Key words: Thymus, Thymic duct cyst, Thymic cyst, Lateral cystic mass in children, Branchial apparatus, Branchial cyst, Cystic hygroma.***

***Journal of Green Life Med. Col. 2020; 5(1): 43- 48***

**Introduction:**

Thymic cysts are uncommon, accounting for only 2% of congenital neck masses,[1] usually presenting in the 1st decade of life.[2]

Thymic cysts are almost all unilateral, mostly on the left side of the neck. They are cystic in 90% cases. Typically, a thymic cyst presents as an asymptomatic mass, but it may be painful if infected, or rapidly increase in size.[3]

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**Thymus - its Anatomical and Embryological Considerations and anomalies**

**History**

"Thymus" derives from the Greek word *thymos* which means soul or spirit. Galen first thought that it has a role for purification of the nervous system in 2nd century AD. Vesalius suggested that it acted as a damper to protect the major vessels in the mediastinum located behind the sternum in 15th century. In 1777, William Hewson was first to identify correctly the thymus as a lymphmodifying gland and in 1832 Sir Astley Cooper described detailed anatomy of the thymus. In 1846, Hassall and Vanarsdale used compound microscope to describe the differences between the thymus and other lymphoid organs, specifically the histological characteristic known as Hassall's corpuscles.[4]

**Anatomy**

The thymus is an encapsulated soft, bilobed organ.The two lobes are joined in the midline by connective tissue that merges with the capsule of each lobe. The greater part of the thymus lies in the superior and anterior mediastina; the inferior aspect of the thymus reaches the level of the fourth costal cartilages. Its superior poles join at, and extend above, the level of the suprasternal notch; the left usually extends higher and is seen first behind the strap muscles. It sometimes reaches the inferior poles of

the thyroid gland or even higher, and is connected to the thyroid gland by the thyrothymic ligament.[5]

**Development**

The thymus is primarily derived from the third branchial pouch, with minor contributions from the fourth branchial pouch. Beginning in the sixth week of gestation, the thymus descends into the anterior mediastinum along paired thymopharyngeal ducts, deep to the thyroid gland and sternocleidomastoid muscle[1]. Both the third and fourth branc-hial pouch originate at the pyriform sinus. Development may arise from the incomplete closure of the thymopharyn-geal duct of the third pouch.[6]

Anatomic pathways for deep tract of the third and fourth branchial cleft anomalies -

In the third branchial cleft anomalies,the deep tract passes posterior to the internal and external carotid arteries, between the 9thand 12th cranial nerves, and ends in the apex of the pyriform sinus.

In the fourth branchial cleft anomalies, the deep tract begins with a sinus at the apex of the pyriform sinus and travels inferiorly in the tracheoesophageal groove, posterior to the thyroid gland, and into the thorax. Next, it loops below1fte aorta on the left or below the subclavian artery on the right and then ascends posterior to the common carotid artery to loop over the hypoglossal nerve and ends at the anterior border of the sternocleidomastoid muscle.[7]

We are reporting three cases in our case series, two female and one male.

**Case Series**

**Case no. 1**

A 14 yrs. old girl was our second case and was admitted in National Institute of ENT, Tejgaon, Dhaka on 13th January, 2016with the complain of a swelling in left side of neck since early childhood which was gradually increasing. There was an oval shaped swelling in the left side of neck occupying in lower half of anterior triangle. The overlying skin was normal and no visible pulsation was seen. It did not move with swallowing.The consistency was softand the margins were not well delineated. It was non tender. Transillumination test was negative. Ultrasonogram revealed a multiseptate cystic mass on the left side of neck. Cytological examination suggested branchial cyst.

A dark blue or blackish coloured cyst was found within the carotid sheath starting from the level of hyoid bone during surgery. It extended lower down and was turned into a cord like structure before entering into the superior mediastinum. After opening the carotid sheath it was found between the internal jugular vein and carotid vessels, the left Vagus nerve was found closely related to the cyst.After removal of the mass it was sent for histopathological examination.It was found a single cystic mass with tubular elongation on gross examination which was multilocular on cut.They found thymic tissue with cholesterol granuloma in cyst and ectopic thymic tissue in cord in microscopic examination. Their diagnosis was thymic cyst.

**Case no.2**

A 7 yrs. old girl was admitted in National Institute of ENT, Tejgaon, Dhakaon 28th February, 2016 with the complain of a swelling in the upper portion of the left side of neck since birth.It was painless and gradually increasing in size. The swelling was occupying in the upper half of anterior triangle of neck on left side. Nothing abnormality was detected in overlying skin. It was non tender, soft in consistency and the margins were not well delineated. Transillumination test was negative. Cystic mass was found on ultrasonography. Cytological examination suggested cystic hygroma.

Our approach was with collar incision in neck at the lower end of mass. We found a dark blue colour cystic mass within the carotid fork extended into the carotid sheath. Within the carotid sheath a cord like structure descended from its lower end into the superior mediastinum. Vagus nerve was found in close relation to the mass. After excision it was sent for histopathological examination. It was found a single cystic mass with tubular elongation on gross examination. On cut the cyst was found multilocular with straw colour fluid. Microscopic examination revealed thymic tissue with cholesterol granuloma in cyst and ectopic thymic tissue in tubular elongated part. Histopathological diagnosis was thymic cyst with ectopic thymic tissue.

**Case no.3**

Our third case was a 7 years old boy (Figure 1). He was admitted in National Institute of ENT, Tejgaon, Dhakaon 7th July, 2019 with complain of a swelling in right side of neck for 01 year which was gradually increasing insize. There was no complain of pain, respiratory distress nor any change of voice. There was an oval shaped swelling in the mid and some lower part of anterior triangle of neck on right side, overlying skin was normal, did not move with swallowing, no visible pulsation was seen. On palpation it was cystic in consistency, non-tender, margins

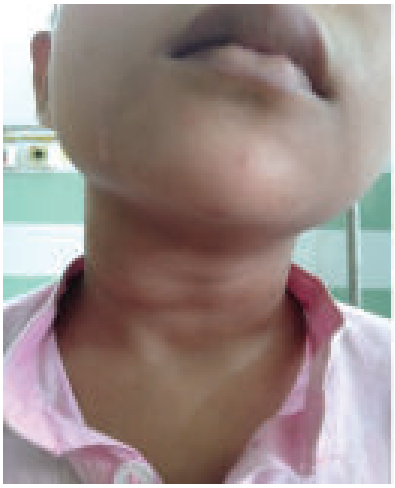

**Fig.-1:** *07 years old boy.*

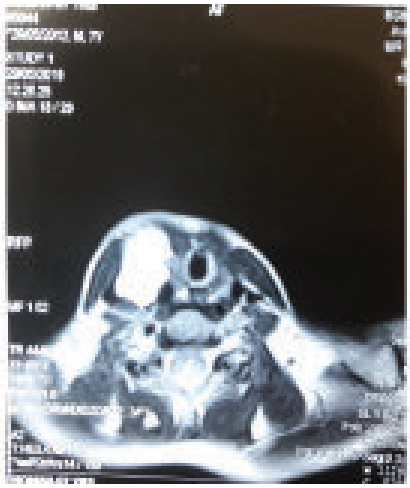

**Fig.-2:** *MRI Axial view.*

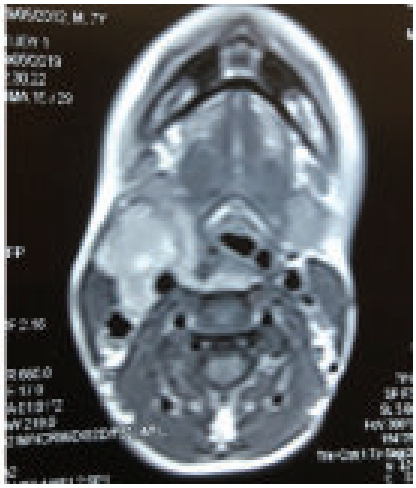

**Fig.-3:** *MRI Axial view.*

were not well delineated. It was not decreased in size on compression.On coughing, it was neither increased in size nor felt any pulsation. Transillumination test was negative. Ear nose throat examination reveals no abnormality. Ultrasonogram of neck mass revealed a multiseptate cystic mass on the right side of neck. Cytological examination revealed cystic mass suggested Branchial cyst. MRI of neckand base of skull (Figure 2, Figure 3 & Figure 4)showed T1WI low and T1WI high signal intensity welldefined cystic structure measuring about 7x5 cm. isnoted arising from right supraglottic region through thyrohyoid membrane into the Parapharyngeal and Paratracheal spaces. Laryngeal lumen is compressed. After IV Contrast (Gd- DTPA) administration, there was no enhancement of lesion was noted. In comment they noted it might be a Laryngocoele but Branchial Cleft cyst and Cystic Lymphangioma may be the other differential diagnosis.Fibreoptic laryngoscopic examination revealed no abnormality in larynx or hypopharynx.

During operation, a yellowish white coloured cystic mass was found within the carotid sheath pushing internal jugular vein superficial and carotid vessels anteromedial in deep. The right Vagus nerve was found superficially and in close contact to the wall of mass (Figure 5). The cystic mass was mobilized and a cord like structure was found descending from its lower end (Figure 6). The mass was then removed intact with some part of cord. The surgical specimen was sent for histopathological examination. Direct laryngoscopic examination revealed no abnormality in larynx or hypopharynx particularly in right pyriform fossa.

Post-operative period was uneventful. The histo-pathological examination found one cyst measuring 7X5X2.5 cm. on gross examination. On cut they found a multilocular cyst within which straw coloured fluid.In their microscopic examination they found thymic tissue with cyst lined by squamous epithelium and supported by fibrous tissue. Their diagnosis was thymic cyst.

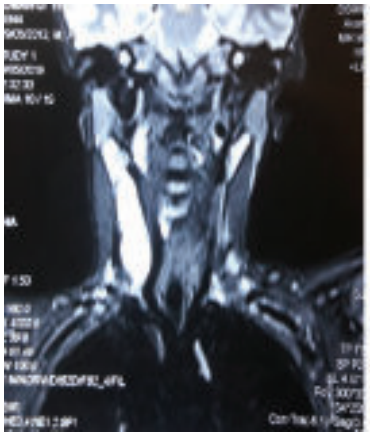

**Fig.-4:** *MRI Coronal view.*

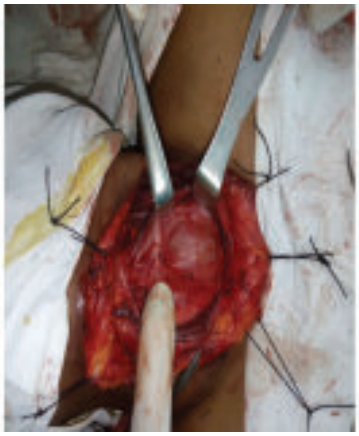

**Fig.-5:** *Mass per operatively*

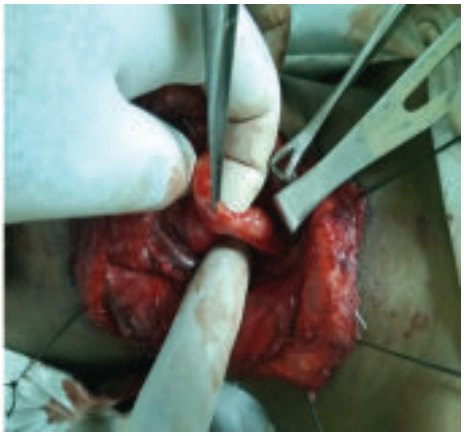

**Fig.-6:** *Duct descending from lower end.*

**Table-I**

*Showing three cases of Thymic duct cysts:*

| Sl. No. | Age/ Sex | Admission Year | Side | Consistency | Transillumi -nation Test | Ultrasonogram | Cytological Report | Clinical Diagnosis |
|---|---|---|---|---|---|---|---|---|
| 1. | 14/F | January/2016 | Left | Soft | Negative | A multiseptate cystic mass on the left side of neck. | Cystic mass suggested branchial cyst. | Branchial cyst. |
| 2. | 7/F | February/2016 | Left | Soft | Negative | Cystic masson the left side of neck. | Cystic mass suggested cystic hygroma. | Cystic hygroma. |
| 3. | 7/M | July/2019 | Right | Soft | Negative | Multiseptate cystic mass on the right side of neck. | Cystic mass suggested branchial cyst. | Branchial cyst . |

**Results:**

We found three cases of Thymic duct cyst, a rare variety of congenital neck mass in last four years. All the three cases were presented as lateral cystic mass, clinically diagnosed as congenital cystic neck mass. These were presented in the first two decades of life. In our case series, we found two female & one male, two of them were presenting in first decade and the other one was in the second decade of life. Swelling were unilateral and painless.Two of them were presented on left side & one on right side of neck. They all were soft in consistency and transillumination tests were negative. In our observation, we found two cysts of dark blue and one with yellowish white in colour.In all cases, the cysts were single and multiloculated. The cyst were found within the carotid sheath in all those three cases. A cord like structure descended into the superior mediastinum from the lower end of the cyst in two cases. In histopathological report of surgical specimens of all those three cases showed ectopic thymic tissue with thymic cyst.

**Discussion:**

Developmental anomalies are fairly common in the pediatric population. Failure of involution embryologic structu-res and duplication of structures can lead to fistulas, sinuses, and cysts. These include first, second, third, and fourth branchial cleft anomalies, as well as preauricular cysts and sinuses and thyroglossal duct cysts.Thyroglossal duct cysts are the most common congenital neck anomaly in children. For the branchial cleft anomalies, the second branchial cleft anomalies is the most common.[7]

Separated thymic tissue is often found scattered around the gland, and ectopic thymic rests are sometimes discovered in unusual mediastinal locations. Small accessory nodules may occur in the neck, representing separated portions, detached during embryological descent, and sometimes reaching more superiorly than the thyroid cartilage. Ectopic intrathyroidal thymi have been reported in children.[5] Thymic rests may be deposited any where along the path from the angle of the mandible to the midline of the neck, between the common carotid artery and the vagus nerve.[1]

Two varieties of thymic cysts are described, congenital and acquired. Persistence of thymopharyngeal tracts and the degeneration of Hassall's corpuscles within ectopic thymic remnants are the two most important etiologies of thymic cysts.[8]

Hsieh et al. conducted a study in 20 years on 331 patients under the age of 18 years presenting with cystic neck masses. They found thyroglossal cysts were the commonest in 181 (54.68%) patients, followed by cystic hygromas (83 patients, 25.08%), branchial clef cysts (54 patients, 16.31%), and bronchogenic cysts (3 patients, 0.91%), and in remained 9 cases (2.72%) were unclassifed. Only one case was diagnosed as thymic cyst (0.30%). They concluded that the cervical thymic cysts were rare.[9]

Ectopic thymic masses are congenital lesions of either solid or cystic in nature and usually present between 2 and 13 years of age as asymptomatic nodules or neck swellings on routine examination. Most cervical thymic lesions are unilateral and commonly on the left side of

neck and in male patients.[10,11]Thymic cysts are more common in children, in contrast to ectopic cervical thymus, which is more common in adults.[12]

Thoracoscopy is an important tool both for diagnostic and therapeutic purposes in mediastinal thymic diseases or thymic cyst. Akihiko Kitami et al in their study on 34 patients (15 males and 19 females, aged between 20 to 78 years with a mean of 49.0 years) with mediastinal diseases underwent diagnostic or therapeutic thoracoscopy, where 9 cases were found thymic diseases including 5 cases with thymic cyst, all were located in anterior mediastinum.[13]

Contrast enhanced computed tomogram (CT) scans can differentiate thymic cysts from other pediatric neck swellings, such as branchial cleft cysts and lymphangiomas; the second branchial cysts are located superficial and lateral to internal jugular vein and common carotid artery, and lymphangiomas are found in the posterior triangle of the neck while thymic cysts are situated in close association with the carotid sheath, between internal jugular vein and carotid vessels.[14,15] Also, thymic cysts tend to be longer, extending toward the anterosuperior mediastinum[15]. Mediastinal extension is seen in 50% of cervical thymic cysts.[16]

Thymic cysts are unilocular or multilocular containing brownish fluid. The cyst wall lining ranges from flattened squamous or cuboidal cells to multilayered stratified squamous epithelium to even primitive respiratory epithelium. Lobulated lymphoid tissue in the cyst wall contains Hassall's corpuscles.[17]Increasing number of cervical thymic cysts reported in the last few years probably reflects greater awareness of this condition among pathologists. It is also possible that in the past, many cases of thymic cyst had been missed and diagnosed as brachial cleft cyst because of inadequate sampling of the specimen. The frequent atrophic condition of the thymic remnants may require sampling from various portions of specimen before a diagnosis of thymic cyst could be rendered.[18]

The preferred management is surgical excision, and diagnosis is confirmed by histologic identification of Hassall corpuscles[3]. Presence of mediastinal thymic tissue should be confirmed prior to surgery to avoid inadvertent removal of the only thymic tissue in a young child, which has the potential to result in serious immune dysfunction.[1]

Thymectomy during childhood has been documented to produce impairment of immune status in later life.[19] Hence, it is imperative that the existence of mediastinal thymus is confirmed before proceeding with the excision of the cervical thymic tissue. Cervical thymic cysts are not known to recur or undergo malignant transformation.[14]

**Conclusion:**

Thymic cysts are rare causes of lateral cystic cervical masses. Due to its rarity, it almost always escapes a correct preoperative diagnosis. CT, MRI, and FNA are all helpful investigations in the diagnosis of cervical thymic cysts, but a definitive diagnosis requiresidentification of thymic tissue containing Hassall's corpuscles. Moreover, it is related to some important structures in neck. So, surgeons should aware of this condition in lateral cystic neck mass in children particularly in the first two decades of life. Greater awareness among the pathologists may decrease the misdiagnosis.

**References:**


1. Sataloff's Comprehensive Text Book of Otolaryngology Head and Neck Surgery, Paeditric Otolaryngology - Vol 6. Page no. 661.
2. P. J. Bruy' ere, P. Moreau, and B. Ghaye, "Congenital thymic cyst,". JBR-BTR, vol. 90, no. 3, pp. 178–179, 2007.
3. Cummings Otolaryngology and Head Neck Surgery 6th Ed. Page 3060
4. Congenital Anomalies of Thymic Origin in the Neck. Álvaro Antonio Herrera Hernández, Paola Aranda Valderrama, and Julio Alexander Díaz Pérez. Acta Otorrinolaringológica Española · June 2008.
5. Grays Anatomy-The Anatomical Basis of Clinical Practice - 41E (2015), Pp 983-986.
6. Scott-Brown's Otorhinolaryngology Head and Neck Surgery. Volume 2. Paeditrics, The Ear, Skull Base. 8th Edition 2018, page 470.
7. Baileys Head Neck Surgery Otolaryngology. 5th Ed. Vol. 1. Page no. 1589, 1609.
8. D.Saggese,G.C.CompadrettiandC.Cartaroni,"Cervical ectopic thymus: a case report and review of the literature," International Journal of Pediatric Otorhinolaryngology, vol.66, no. 1, pp. 77–80, 2002.
9. Hsieh YY, Hsueh S, Hsueh C, Lin JN, Luo CC, Lai JY, et al. Pathological analysis of congenital cervical cysts in children: 20 Years of Experience at Chang Gung Memorial Hospital. Chang Gung Med J 2003;26:107-13.
10. Guba AM, Adam AE, Jaques DA, Chambers RG. Cervical presentation of thymic cysts. Am J Surg.1978;136:430–436.
11. Miller MB, DeVito MA. Cervical thymic cyst.Otolaryngol Head Neck Surg. 1995;112:585–588.
12. MadhiraSrivalli, Hamid Abdul Qaiyum, Prayaga N. Srinivas Moorthy, and KollojuSrikanth.A Case Report of Cervical Thymic Cyst and Review of Literature. Indian J Otolaryngol Head Neck Surg. 2011 January; 63(1): 93–95.
13. Akihiko Kitami, Takashi Suzuki, RyosukeUsuda, Mikio Masuda and Shuichi Suzuki. Diagnostic and Therapeutic Thoracoscopy for Mediastinal Disease. Ann ThoracCardiovascSurg Vol. 10, No. 1 (2004).

14. Kaufman MR, Smith S, Rothschild MA, Som P. Thymopharyngeal duct cyst. Arch Otolaryngol Head Neck Surg 2001;127:1357-60.

15. Daga BV, Chaudhary VA, Dhamangaokar VB. Case Report: CT diagnosis of thymic remnant cyst / thymopharyngeal duct cyst. Indian J Radiol Imaging 2009;19:293-5.

16. Cervical Thymic Cyst: A Rare Differential Diagnosis in Lateral Neck Swelling. Vijendra Shenoy, M. Panduranga Kamath, Mahesh Chandra Hegde, Raghavendra Rao Aroor,1 and Vijetha V. Maller. Hindawi Publishing Corporation Case Reports in Otolaryngology, Volume 2013, Article ID 350502, 4 pages.

17. K. K. Koeller, L. Alamo, C. F. Adair, and J. G. Smirniotopoulos, "From the archives of the AFIP. Congenital cystic masses of the neck: radiologic-pathologic correlation," Radiographics, vol.19, no. 1, pp. 121–146, 1999.

18. K.K.Prasad, R.K.Gupta, M.Jain, D.K.Kar, and G.Agarwal, "Cervical thymic cyst: report of a case and review of the literature,"Indian Journal of Pathology and Microbiology,vol.44, no. 4, pp. 483–485,2001.

19. Brearley S, Gentle TA, Baynham MI, Roberts KD, Abrams LD, Thompson RA. Immunodeficiency following neonatal thymectomy in man. ClinExpImmunol 1987;70:322-7.

# COLLEGE NEWS

Presentation held during CME/CPD sessions organized by Medical Education Unit (MEU) of Green Life Medical College from July to December 2019:

| Date | Topics | Department |
|---|---|---|
| 03.07.19 | Pre-diabetes | Department of Biochemistry |
| 10.07.19 | Laser in Dermatology | Department of Dermatology |
| 14.07.19 | Professional negligence in Medical Practice | Department of Forensic Medicine |
| 17.07.19 | Guest Lecture | Department of Urology |
| 24.07.19 | Endodontic emergency | Department of Dentistry |
| 31.07.19 | Story of struggling pain | Department of Gynae and Obs |
| 07.08.19 | Immunotherapy | Department of Microbiology |
| 18.08.19 | Epidemiological and Microbiological aspects of Dengue Syndrome | Medical educational unit |
| 21.08.19 | World hepatitis day | Department of Medicine |
| 04.09.19 | How to send biopsy specimen in pathology lab | Department of Pathology |
| 07.09.19 | Medical Ethics | Department of Forensic Medicine |
| 11.09.19 | Ocular manifestation of tuberculosis | Department of Ophthalmology |
| 15.09.19 | World Alzheimer's day | Department of Medicine |
| 18.09.19 | HPV& Cervical Carcinoma | Department of Microbiology |
| 25.09.19 | Peri-operative Fluid Management | Department of Anesthesia |
| 02.10.19 | Snake bite awareness | Department of Medicine |
| 05.10.19 | International Day of older persons | Department of Medicine |
| 06.10.19 | Biosafety Level | Department of Microbiology |
| 07.10.19 | National guidelines & tools of Quality Assurance Scheme( SAG) for Medical College in Bangladesh | Department of Community Medicine |
| 10.10.19 | Health promotion and Suicide prevention world-Mental Health Day-Awareness program | Department of Psychiatry |
| 16.10.19 | Evaluation of Insulin sites technology | Department of Endocrinology& Medicine |
| 13.10.19 | Osteoporosis and orthopedics | Department of Orthopedics Surgery |
| 27.10.19 | Breast Cancer Treatment | Department of Surgery |
| 06.11.19 | Colour Blindness | Department of Physiology |
| 13.11.19 | Endodontic Flare up | Department of Conservative Dentistry |
| 20.11.19 | Intra-abdominal lamp- a rare variety | Department of Surgery |
| 04. 12.19 | World AIDS Day 2019 | Department of Medicine |
| 11.12.19 | Scapula is a dimorphic bone & its clinical correlation | Department of Anatomy |

## Corrigendum

A word in the title of an original article named *"Assessing Materal Care of Mothers having Children Less Than Two Years of Age in the Rural Area of Dhamrai Upazilla, Dhaka"* was misspelled due to typo which was published in the volume 4, number 2 (July 2019) of Green Life Medical College Journal.

The correct title is mentioned below.

"Assessing Maternal Care of Mothers having Children Less Than Two Years of Age in the Rural Area of Dhamrai Upazilla, Dhaka"

The editorial committee expresses their regret for this unintended mistake and the inconvenience caused to the readers and its authors.

**Executive Editor**
GMCJ

# GREEN LIFE MEDICAL COLLEGE JOURNAL

## Reviewers Panel



***Name of the reviewers are listed according to alphabetic order, not according to order of precedence.***