

ISSN No. 2663-2314

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

Green Life Medical College Journal

Volume 8

Number 1

January 2023

Published in 2024

CONTENTS

Editorial

- Depression in Thyroid Disorders 1
Tanjina Hossain

Original Articles

- Renal Impairment in Septicaemic Newborn in a Tertiary Care Hospital 3
Malek A, Khan S
- Prevalence and Pattern of Ocular Diseases: A 4 years Retrospective Study in Ophthalmology
Outpatient Department of Green Life Medical College and Hospital, Dhaka, Bangladesh 7
Parvin S, Afrin M, Akhter K, Islam SA
- Breadth & Annular Circumference of Tricuspid Valve of Human Heart –
A Morphological Study 13
Reza R, Ara S

Review Article

- Vasomotor Rhinitis: An Overview 17
Khanam A, Islam NN, Lubana N

Case Report

- Branchial Cleft Cyst with Papillary Carcinoma of Ectopic Thyroid: A Case Report 23
Awal N, Rahman MS, Afroz F, Rahman MR, Alam MS

College News

27



Official Journal of
Green Life Medical College

Website: greenlife.edu.bd

ISSN No. 2663-2314

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

GREEN LIFE MEDICAL COLLEGE JOURNAL

Vol. 8, No. 1, January 2023

Published in 2024

Journal Committee

Chairman, Editorial Board	National Professor Shahla Khatun
Editor in Chief	Prof. A.B.M Bayezid Hossain
Executive Editor	Dr. Sheela Khan
Assistant Editors	Prof. Fahmida Kabir Dr. Md. Rifayet Rahman Dr. Tanjina Hossain Dr. Rashedul Hassan Dr. Rafzana Arifina
Members	Prof. Dr. Humaira Naushaba Prof. Md. Manjur Alam Prof. Dr. Aftab Uddin Ahmed Prof. Dr. Homayera Rahman Prof. Dr. Quazi Rakibul Islam Prof. Rabeya Begum Prof. Md. Rezaul Karim Dewan Prof. Dr. SK. Abdul Fattah Prof. Dr. Md. Zahidur Rahman Prof. Sanya Tahmina Jhora Prof. Dr. Mohammad Asifuzzaman Prof. Rowsan Ara Prof. Abdullah Al Tarique Prof. Dr. Nurun Nahar Chowdhury Prof. Lima Shompa Dr. Sanjida Akhter Dr. Salma Parvin Dr. Mafruha Afrin Dr. Suparna Bhowmik Dr. Md. Samia Shihab Uddin
Advisory Board	Prof. Shamsuddin Ahmed Dr. Md. Mainul Ahasan Prof. Pran Gopal Datta Prof. Abul Khair Prof. Abu Shafi Ahmed Amin

Address of Correspondence

Executive Editor, Green Life Medical College Journal
31 and 32, Bir Uttam K.M. Shafiullah Sarak, Dhanmondi, Dhaka-1205
Tel: 9612345-50 Ext. 1252

Email: greenlifejournal@gmail.com; contact@greenlife.edu.bd; Website: www.greenlife.edu.bd/gmc-journal

ABOUT THE JOURNAL

Full Name of the Journal	: Green Life Medical College Journal
Short Name	: GMCJ
Nature of Publication	: Bi-annual
Published From	: Green Life Medical College
Accreditation	: Recognized by Bangladesh Medical & Dental Council (BM&DC)
ISSN	: 2663-2314
Address	: 31 and 32, Bir Uttam K.M. Shafiullah Sarak, Dhanmondi, Dhaka-1205 Phone: 9612345-50 Ext. 1252

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

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

INSTRUCTION TO AUTHORS:

Papers:

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

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

Legal considerations:

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

Preparation of manuscript:

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

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

The standard layout of a manuscript:

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

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

Title page:

A separate page which includes the title of the paper. Titles should be as short and concise as possible (containing not more than 50 characters). Titles should provide a

reasonable indication of the contents of the paper. This is important as some search engines use the title for searches. Titles in the form of a question, such as ‘Is drinking frequent coffee a cause of pancreatic carcinoma?’ may be acceptable.

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

Abstract:

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

Keywords:

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

Introduction:

The recommended structures for this section are:

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

The introduction to a paper should not require more than about 300 words and have a maximum of 1.5 pages double-spaced. The introduction should give a concise account of the background of the problem and the object of the investigation. It should state what is known of the problem

to be studied at the time the study was started. Previous work should be quoted here but only if it has direct bearing on the present problem. The final paragraph should clearly state the primary and, if applicable, secondary aims of the study.

Methods:

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

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

Ethical clearance:

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

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

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

circumstances the editors may request blinded copies of these documents to address questions about ethics approval and study conduct.

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

Single case reports:

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

Animal studies:

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

Drugs:

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

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

Discussion:

Do not repeat the data in detail, already given in the results. Give implications of the findings, their strengths and limitations in comparison to other relevant studies. Avoid un-qualified statements and conclusions which are not supported by the data. Avoid claiming priority.

Conclusion:

Comments on the observation of the study and the conclusion derived from it. New hypothesis or implications of the study may be labeled as recommendations.

References:

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

Examples of correct forms of references:

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

Chapter in a book:

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

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

LETTER TO THE EDITOR:

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

Prof. Dr. ABM Bayezid Hossain

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.

Depression in Thyroid Disorders

Endocrine disorders are frequently accompanied by psychological disturbances. Conversely, psychiatric disorders, to significant extent demonstrate patterns of endocrine dysfunctions. Endocrinopathies manifest as clusters of psychiatric symptoms, because hormones affect a variety of organ systems function, including brain.¹ Psychiatric symptom may be the first manifestations of endocrine disease, but often are not recognized early. Psychiatric manifestations of endocrine dysfunction include mood disturbances, anxiety, cognitive dysfunction, dementia, delirium, and psychosis.²

Depression is one of the commonest mental disorders and regarded as the leading cause of disability worldwide. The lifetime prevalence of depression and anxiety is 11.8% to 36.8% and 5.0% to 41.2% respectively in the patients with previously known thyroid disorder.³

The association between thyroid function and psychiatric disorders particularly mood disorders has long been recognized. Parry in 1825 reported an increased incidence of “nervous affectations” in thyroid disorders. Gull in 1873 showed the relation between myxedema and psychosis. Later, Asher in 1949 described the term “myxedema madness” to describe the mental state of subjects with hypothyroidism.⁴

Psychiatric disorders often feature disruptions of the hypothalamus-pituitary-thyroid axis (HPTA): Patients with depression have been found to show abnormal responses to thyroid-stimulating hormone (TSH) and thyrotropin releasing hormone (TRH), as well as elevated TRH concentrations in cerebrospinal fluid and increased prevalence of antithyroid antibodies. Available data indicates that overt thyroid pathology is rare in subjects with depression, on the contrary, subclinical thyroid pathology appears to be a significant risk factor for psychiatric disorders.⁵

Psychiatric manifestations in thyroid disorders

Both hypothyroidism and hyperthyroidism may be accompanied by various neuro psychiatric manifestations ranging from mild depression and anxiety to overt psychosis. Dysphoria, anxiety, irritability, emotional lability, and impairment in concentration constitute the classical neuro psychiatric symptoms occurring in hyperthyroidism or thyrotoxicosis.⁶ On the other hand, hypothyroid patients frequently demonstrate features of depression, cognitive dysfunction, apathy, and psychomotor slowing. In severe forms of hypothyroidism, clinical symptoms may mimic that of melancholic depression and dementia.⁷

In patients with hypothyroidism treated with Thyroxine, psychological symptoms may persist even when they achieve an euthyroid state.⁸ Now it is thought that impaired psychological wellbeing in these subjects may be related to the occurrence of genetic polymorphisms in the D2 gene as well as the OATPC1 encoding gene.^{9,10}

Autoimmunity and depression

A prevalence of up to 20% of elevated titers of antithyroid antibodies has been documented in depressed patients in several reports compared to a 5–10% prevalence in general population.¹¹ But whether this link have any clinical significance remains unclear since it was most often accompanied by normal serum TSH concentrations.¹²

Fountoulakis et al. found higher thyroid binding inhibitory immunoglobulins (TBII) in depressed patients suggesting the presence of an autoimmune process involving the thyroid gland in depressed patients.¹³

Thyroid disorder in pregnancy and depression

Costantine et al found that at least one quarter of pregnant women with subclinical hypothyroidism screened positive for depression and that antenatal thyroxine treatment was not associated with improved depressive symptoms during pregnancy.¹⁴ The evidence for an association between sub clinical hypothyroidism and mood disorders for pregnant women is limited. However, antenatal total and free thyroxine levels in the lower range of normal in the third trimester of pregnancy are associated with a greater risk of developing postpartum depression.¹⁵ An attempt to decrease the incidence of postpartum depression in thyroid antibody positive women with daily administration of thyroxine for 18 weeks postpartum was unsuccessful.¹⁶

Use of Antidepressant and thyroid disorder

Study shows that the use of lithium in depressive disorder is associated with an increased risk of hypothyroidism. Most problems appeared to occur early during the treatment and among those who had higher blood lithium levels. Younger women (<60 years old) are most commonly affected. It is suggested that patients, especially young women, should be monitored for thyroid function problems when they are managed with lithium.¹⁷ Although this is not a recommendation stated in the current guidelines of the American Thyroid Association, consideration might be given to certain patients who are at a higher risk, such as those who have a strong family history or who have a goiter prior to start treatment.

Screening and Treatment for depression in thyroid disorders

According to the American Association of Clinical Endocrinologists, “The diagnosis of subclinical or clinical hypothyroidism must be considered in every patient with depression.¹⁸ Normalization of pretreatment thyroid function tests with remission of depression has been reported. Although whether this is related to clinical recovery or merely a result of a direct effect of antidepressants remains to be determined.¹⁹

Both tricyclic antidepressants and SSRIs appear to enhance the activity of D2 resulting in an increased conversion of T4 in to active T3 in the brain.^{20,21}

The mechanisms underlying the interaction between thyroid function and depression still remain to be clarified and a causal relationship between the two cannot be established yet. Screening patients presenting with depression for thyroid dysfunction seems reasonable though it is not routinely recommended. The continuing research in the biochemical, genetic, and neuroimaging fields seems most promising in providing a deeper understanding of the thyroid-depression interactions.

Journal of Green Life Med. Col. 2023; 8(1): 1-2

Tanjina Hossain

Associate professor

Department of Endocrinology and Metabolism
Green Life Medical College, Dhanmondi, Dhaka
e-mail: tanjina75@gmail.com

References:

- Lishman, W.A. (1998). Endocrine Diseases and Metabolic Disorders. In Organic psychiatry the psychological consequences of a cerebral disorder, 3rd ed, pp. 507-69. Oxford: Blackwell Science.
- Joffe RT, Brasch JS, MacQueen GM: Psychiatric aspects of endocrine disorders in women. Psychiatr Clin North Am. 2003;26:683.
- Shoib S, Mushtaq R, Dar MM et al. Psychiatric Manifestations in thyroid disorders IJCCI.2013. 5 (Issue 3),84:98, 1st October 2013.
- H. D’haenen, J. A. D. Boer, and P. Willner, Biological Psychiatry, vol. 1, Wiley, Chichester, UK, 2002.
- K. N. Fountoulakis, S. Kantartzis, M. Siamouli et al., “Peripheral thyroid dysfunction in depression,” e World Journal of Biological Psychiatry, vol. 7, no. 3, pp.131–137, 2009.
- J. W. Taylor, “Depression in thyrotoxicosis,” American Journal of Psychiatry, vol. 132, no. 5, pp. 552–553, 1975.
- P. C. Whybrow, A. J. Prange Jr., and C. R. Treadway, “Mental changes accompanying thyroid gland dysfunction. A reappraisal using objective psychological measurement,” Archives of General Psychiatry, vol. 20, no. 1, pp. 48–63, 1969.
- P. Saravanan, W. F. Chau, N. Roberts, K. Vedhara, R. Greenwood, and C. M. Dayan, “Psychological well-being in patients on ‘adequate’ doses of L-thyroxine: results of a large, controlled community-based questionnaire study,” Clinical Endocrinology, vol. 57, no. 5, pp. 577–585, 2002.
- V. Panicker, P. Saravanan, B. Vaidya et al., “Common variation in the DIO2 gene predicts baseline psychological well-being and response to combination thyroxine plus triiodothyronine therapy in hypothyroid patients,” Journal of Clinical Endocrinology and Metabolism, vol. 94, no. 5, pp. 1623–1629, 2009.
- W. M. Van Der Deure, B. C. Appelhof, R. P. Peeters et al., “Polymorphisms in the brain-specific thyroid hormone transporter OATP1C1 are associated with fatigue and depression in hypothyroid patients,” Clinical Endocrinology, vol. 69, no. 5, pp. 804–811, 2008.
- C. B. Nemeroff, J.S.Simon, J. J.Haggerty, and D.L.Evans, “Antithyroid antibodies in depressed patients,” American Journal of Psychiatry, vol. 142, no. 7, pp. 840–843, 1985.
- R. T. Joffe, “Antithyroid antibodies in major depression,” Acta Psychiatrica Scandinavica, vol. 76, no. 5, pp. 598–599, 1987.
- K. N. Fountoulakis, A. Iacovides, P. Grammaticos, G. S. Kaprinis, and P. Bech, “Thyroid function in clinical subtypes of major depression: an exploratory study,” BMC Psychiatry, vol. 4, article 6, 2004.
- Maged M. Costantine, MD, Karen Smith, Elizabeth A. Thom et al. Effect of Thyroxine Therapy on Depressive Symptoms Among Women With Subclinical Hypothyroidism. American College of Obstetricians and Gynecologists. Published by Wolters Kluwer Health, VOL. 135, NO. 4, APRIL 2020
- Haggerty JJ Jr, Evans DL, Golden RN, Pedersen CA, Simon JS, Nemeroff CB. The presence of antithyroid antibodies in patients with affective and nonaffective psychiatric disorders. Biol Psychiatry 1990;27:51–60.
- B. Harris, R. Oretti, J. Lazarus et al., “Randomised trial of thyroxine to prevent postnatal depression in thyroid-antibody positive women,” British Journal of Psychiatry, vol. 180, pp. 327–330, 2002.
- Shine B et al. Long-term effects of lithium on renal, thyroid, and parathyroid function: a retrospective analysis of laboratory data. Lancet 2015;386:461-68.
- H. J. Baskin, R. H. Cobin, and D. S. Duick, “American association of clinical endocrinologists medical guidelines for clinical practice for the evaluation and treatment of hyperthyroidism and hypothyroidism,” Endocrine Practice, vol. 8, no. 6, pp. 457–469, 2002.
- M. L. Rao, S. Ruhrmann, B. Revey et al., “Low plasma thyroid indices of depressed patients are attenuated by antidepressant drugs and influence treatment outcome,” Pharmacopsychiatry, vol. 29, no. 5, pp. 180–186, 1996.
- A. Campos-Barros, H. Meinhold, M. Stula et al., “The influence of desipramine on thyroid hormone metabolism in rat brain,” Journal of Pharmacology and Experimental Therapeutics, vol. 268, no. 3, pp. 1143–1152, 1994.
- A. Baumgartner, M. Dubeyoko, A. Campos-Barros, M. Eravci, and H. Meinhold, “Subchronic administration of fluoxetine to rats affects triiodothyronine production and deiodination in regions of the cortex and in the limbic forebrain,” Brain Research, vol. 635, no. 1-2, pp. 68–74, 1994.

Renal Impairment in Septicaemic Newborn in a Tertiary Care Hospital

MALEK A¹, KHAN S²

Abstract

Introduction: The morbidity and mortality of acute renal failure in newborn is high. Newborns with acute renal failure need lifelong monitoring of their renal function. Acute renal failure is a common complication of sepsis. The objective of this study was to find out the incidence of renal impairment in newborn suffering from septicaemia and the factors associated with septicaemia in a tertiary care hospital of Bangladesh.

Methods: This longitudinal 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 65 neonates suffering from septicaemia.

Results: Sixty five septicaemic newborn were studied to find out renal impairment. Mean age of the study babies was 3±1 days. Septicaemia was more observed in normal vaginal delivery (70.8%). The commonest history related to birth was premature rupture of membrane (56.9%) and the commonest manifestation of septicaemia was in the form of lethargy (73.8%). Oliguria was found in 34 (52.3%) study babies. The current study observed that 31 (47.7%) septicaemic babies were not suffered from renal impairment according to presence of oliguria, out of which 2 (3.1%) were suffered from renal impairment according to serum creatinine level. Renal failure was found in 1 (1.5%) baby and pre renal failure was found in 7 (10.8%) babies according to FENa level. Seven (10.8%) septicaemic babies died during the course of treatment and all who died had renal impairment.

Conclusion: Renal impairment occurs in significant number of septicaemic newborn. It can thus be recommended that rapid diagnosis and early and effective treatment of newborn with septicaemia and consequent renal impairment can reduce neonatal morbidity and mortality.

Keywords: Renal impairment, Septicaemia, Fractional excretion of sodium

Journal of Green Life Med. Col. 2023; 8(1): 3-6

Introduction:

Compromised kidney function in the perinatal period has been increasingly recognized during recent years, and acute renal failure is a frequent clinical situation in neonatal intensive care units. Acute Renal Failure (ARF) is associated with a primary condition such as sepsis, metabolic diseases, perinatal asphyxia and/or prematurity.¹ Renal failure occurred in 26% neonates with sepsis.²

Septicaemia is a serious manifestation of an infection localized outside the vascular system and/or of an immune defect resulting in reduced capacity of the patient to eliminate invading bacteria by phagocytosis or antibody response.³

Nearly 15% neonates with septicemia developed ARF which was predominantly oliguric in type. The mortality rate in the septicemic neonates with ARF was significantly high. Further the mortality in neonates with oliguric ARF was significantly higher than those with non-oliguric ARF.⁴

Risk factors for sepsis include birth to mothers with inadequately treated maternal group B Streptococcus colonization, intra-amniotic infection, maternal temperature greater than 100.4°F (>38°C), rupture of membranes greater than 18 hours, and preterm labor.

1. Abdul Malek, Associate Professor, Department of Paediatrics, Green Life Medical College

2. Sheela Khan, Associate Professor, Department of Community Medicine and Public Health, Green Life Medical College

Address of Correspondence: Abdul Malek, Associate Professor, Department of Paediatrics, Green Life Medical College, email: malekabdul003@yahoo.com

Received: 09.08.2023

Accepted: 06.07.2024

The organisms that most commonly cause early-onset sepsis include group B Streptococcus, Escherichia coli, and viridans streptococci. Infants often present within the first 24 hours after birth with clinical signs of sepsis, with respiratory distress as the most common presenting symptom.⁵

Newborns with acute renal failure need life-long monitoring of their renal function, blood pressure, and urinalysis. Newborns who have suffered substantial loss of nephrons as may occur in cortical necrosis are at risk for late development of renal failure after apparent recovery from the initial insult.⁵

Prerenal failure is completely reversible in the early course of the disease. If adequate treatment is delayed, however, structural damage to the kidneys by prolonged ischemia will ensue leading to a poor prognostic outcome. The study was done to find out the incidence of renal impairment in septicemic newborn of Bangladesh and the factors associated with septicaemia. The study finding may help to reduce septicaemia and consequent renal impairment by early diagnosis and adequate treatment of septicaemia and consequent renal impairment.

Methods:

This longitudinal descriptive 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. Sixty five clinically diagnosed cases of septicaemia who were available and gave consent were included in the study.

A preformed history sheet filled up at enrollment into study containing relevant information such as age of the child, sex of the child, maternal antenatal history etc. Then 2ml of venous blood sample was collected aseptically for the estimation of creatinine level and sodium. 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 and sodium using computerized auto-analyzer.

Following renal indices was calculated:

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

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

A 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 $\text{FENa} > 0.72$.⁶ Statistical analysis was carried out manually.

Follow up of the study newborn was carried out up to discharge to observe outcome.

Results:

This longitudinal descriptive study was done to find out the incidence of renal impairment among septicaemic newborn of Bangladesh and the factors associated with septicaemia.

Sixty five septicaemic newborn were studied to find out renal impairment. Mean age of the study babies was 3 days. Male female ratio of the newborn was 4:3. Septicaemia was more observed in normal vaginal delivery (70.8%).

Commonest history related to birth was premature rupture of membrane (56.9%) (Table I).

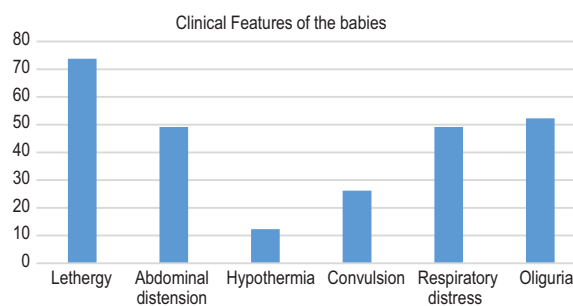
Table I

Distribution of septicaemic babies according to their history related to birth

History related to birth	Frequency	Percentage
Normal vaginal delivery	46	70.8
Caesarian section	19	29.2
Premature rupture of membrane	37	56.9
Prolong delivery	18	27.7
Obstructed labor	10	15.4

*Multiple response were present

The commonest clinical features of septicaemia was in the form of lethargy (73.8%) (Figure I). oliguria was found in 52.3% of the study baby. Proportion of the septicaemic babies with abdominal distension (49.2%) and respiratory distress (49.2%) were same.



*Multiple response were present

Figure I: *Distribution of babies according to their clinical features*

According to PBF study 44 (67.7%) babies had features of septicaemia. More than half 36 (55.4%) of the babies had CRP > 6 mg/L (Table II).

Table II
Distribution of Hematological indices of the study neonates

Hematological indices	Frequency	Percentage
Blood Hb gm/dl		
>12 gm/dl	15	23.1
≤12 gm/dl	50	76.9
Platelet count		
Normal (> 150'10 ⁹ /L)	40	61.5
Thrombocytopenia (< 150'10 ⁹ /L)	25	38.5
PBF		
Normal	21	32.3
Features of Septicemia	44	67.7
CRP		
<6 mg/L	29	44.6
>6 mg/L	36	55.4

Blood culture of 5 (9.1%) babies showed Klebsiella, 4 (7.3%) babies showed E. coli and 1 (1.8%) baby showed Staphylococcus aureus.

Table III
*Pattern of blood culture in neonates under study (*n= 55)*

Blood culture	Frequency	Percentage
No growth	43	78.2
Klebsiella	5	9.1
Staphylococcus aureus	1	1.8
E. coli	4	7.3
Salmonella Typhi	2	3.6
Total	55	100

*Blood cultures were not done in 10 patients due to some unavoidable circumstances.

Oliguria was found in 34 (52.3%) study babies. The current study observed that 31(47.7%) septicaemic babies were not suffered from renal impairment according to presence of oliguria, out of which 2 (3.1%) were suffered from renal impairment according to serum creatinine level (Table IV).

Table IV
Distribution of septicaemic babies according to presence of oliguria and levels of serum creatinine

Serum creatinine	Oliguria present	Oliguria absent	Total
	Up to 0.9 mg/dl	28 (43.1%)	
Above 0.9 mg/dl	6 (9.2%)	2 (3.1%)	8 (12.3%)
Total	34 (52.3%)	31 (47.7%)	65 (100%)

Renal failure was found in 1(1.5%) baby and pre renal failure was found in 7 (10.8%) babies according to FENa level (Table V).

Table V
Distribution of septicaemic babies according to renal impairment by level of Fractional Excretion of Sodium (FENa)

FENa level	Frequency	Percentage
Normal (up to 0.72%)	57	87.7
Pre renal failure (>0.72%)	7	10.8
Renal failure (>.25%)	1	1.5
Total	65	100

Fifty eight cases were discharged while 7 died during the course of treatment. All the babies who died had renal impairment (Table VI).

Table VI
Distribution of the septicaemic babies according to their outcome at discharge

Outcome at discharge	Frequency	Percentage
Alive	58	89.2
Dead	7	10.8
Total	65	100

Discussion:

This longitudinal descriptive study was carried out with an objective to find out the impairment of renal function in newborns suffering from septicaemia by doing fractional excretion of sodium (FEN α), in addition to, serum creatinine and oliguria. A total of 65 newborn were included in the study.

The present study found male female ratio in the newborn suffering from septicaemia was almost 4:3. Male were more than female may be due to male baby receive more care than female in Bangladeshi culture, so that they seek more medical care.

Septicaemia was more observed in normal vaginal delivery (70.8%) in this study. More handling is done in normal vaginal delivery so the chance of septicaemia is more in this procedure.

The commonest history related to birth was premature rupture of membrane (56.9%) (Table I). Premature rupture of membrane takes more time to complete delivery process so more chance of septicaemia to occur.

The manifestation of septicaemia were in the form of lethargy (73.8%), abdominal distension (49.2%), and

respiratory distress (49.2%) (Figure I). The results obtained in the current study regarding the clinical features are comparable with Khan study.⁷

In the present study micro-organism found in the blood culture revealed Klebsiella in 5 (9.1%), Staphylococcus aureus in 1 (1.8%), E. Coli in 4 (7.3%) and Salmonella Typhi in 2 (3.6%) septicaemic babies respectively. Khan reported that among microorganisms isolated from blood culture in septic neonates E. coli were the most common pathogen. The author mentioned among isolated organism's 3 (10.0%) samples showed E.coli, 2 (6.6%) samples showed Klebsiella pneumoniae, 1 (3.3%) sample showed Pseudomonas aeruginosa and acinetobacter each.⁷

The current study observed 31 (47.7%) septicaemic babies were suffered from renal impairment according to presence of oliguria, out of which 2 (3.1%) were suffered from renal impairment according to serum creatinine level (Table IV). Tack ED showed 28 neonates presented with Oliguria, out of which 8 were suffered from renal impairment according to serum creatinine level.⁸ Another study found renal failure occurred in 26% neonates with sepsis. Although ARF in neonates has been reported to be predominantly oliguric, it was observed that ARF secondary to neonatal sepsis was predominantly non oliguric. The mortality being three times higher in neonates with ARF demands a greater awareness of this entity among practitioners and better management of this condition.²

In the present study the newborn was divided into three categories i.e. normal, pre-renal failure and renal failure according to fractional excretion of sodium of the newborn. Renal function was normal in 57 (87.7%) septicaemic babies while 7 (10.8%) develop pre renal failure and 1 (1.5%) developed renal failure. Matthew observed the mean FENa was significantly ($p < 0.05$) higher in renal failure compared to prerenal Oliguria, where the mean FENa was $4.25 \pm 2.18\%$ and $0.95 \pm 0.55\%$ in renal failure and prerenal Oliguria respectively.⁹ Almost similar findings obtained by Airede, Bello and Weerasinghe.¹⁰

In the present study, 7 (10.8%) 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 septicaemia.

Conclusion:

Renal impairment occurs in significant number of septicaemic newborn. Renal failure can be anticipated in septicaemia if present with increased fractional excretion of sodium in addition to oliguria or increased serum creatinine level. Prognosis of neonates with Acute renal failure requiring peritoneal dialysis is very poor. It can thus be recommended that rapid diagnosis and early and effective treatment of newborn with septicaemia and consequent renal impairment can reduce neonatal morbidity and mortality.

References:

1. Pinar Isik Agras I, Aylin Tarcan, Esra Baskin, Nurcan Cengiz, Berkan Gürakan, Umit Saatci. Acute renal failure in the neonatal period. *Ren Fail.* 2004 May;26(3):305-9. doi: 10.1081/jdi-200026749.
2. N B Mathur I, Himanshu S Agarwal, Arti Maria. Acute renal failure in neonatal sepsis. *Indian J Pediatr.* 2006 Jun;73(6):499-502. doi: 10.1007/BF02759894.
3. S R Norrby, A M Geddes. Management of septicaemia. *Scand J Infect Dis Suppl.* 1982;31:112-7.
4. G Jayashree I, A Saili, M S Sarna, A K Dutta. Renal dysfunction in septicemic newborns. *Indian Pediatr* 1991 Jan;28(1):25-9.
5. Sharon Phillips Andreoli I. . Acute renal failure in the newborn. 2004 Apr;28(2):112-23. doi: 10.1053/j.semperi.2003.11.003
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. Khan TH. Effect of rhG-CSF for the treatment of neonates in presumed sepsis with neutropenia. Bangabandhu Sheikh Mujib Medical University, Thesis, 2010.
8. Tack ED, Perlman JM and Robson AM. Renal Injury in Sick Newborn Infants: A Prospective Evaluation Using Urinary β_2 -Microglobulin Concentrations. *Pediatrics* 1988;81: 432-440.
9. Matthew OP, Jones AS, James E, Bland H, Groshong T. Neonatal renal failure: Usefulness of diagnostic indices. *Pediatrics.* 1980; 65: 57-60.
10. Airede AL. Birth asphyxia and hypoxic-ischaemia encephalopathy: incidence and severity. *Ann Trop Paediatr* 1988; 11:331-335.

Prevalence and Pattern of Ocular Diseases: A 4 years Retrospective Study in Ophthalmology Outpatient Department of Green Life Medical College and Hospital, Dhaka, Bangladesh

PARVIN S¹, AFRIN M², AKHTER K³, ISLAM SA⁴

Abstract

Introduction: Ophthalmology Out Patient Department (OPD) of Green life medical college has been serving a significant number of patients everyday coming from different parts of Dhaka city. Our study aims to determine the prevalence & pattern of ocular diseases in this study area so that optimum management of ocular diseases can be possible with necessary improvement of efficient eye care services. The objective of this study was to identify the prevalence and patterns of ocular diseases among patients visited to eye OPD, Green life medical college.

Methods: This observational descriptive study was conducted from April, 2018 to May, 2022 among 5417 patients in outpatient department of Green life Medical College and Hospital, Dhaka, Bangladesh. Data were collected on the basis of the clinical records. Both descriptive and inferential statistics were performed by using MS Excel and SPSS-23 software.

Results: Among 5417 patients, maximum 1951 (36.01%) ocular diseases patients were from middle age group 39 to 60 years followed by 1947 (35.94%) were from adolescent age group 19 to 38, 1078 (19.90%) were from child (0-18) age group and only 441 (8.14%) from Old age group (61-95). Highest 3040 (56.12%) were female and 2377 (43.88%) were male. This study find out the prevalence and pattern of ocular diseases among the out patients of Ophthalmology Department of Green life medical college and hospital and the results shows highest 3079 (56.83%) were refractive error followed by Conjunctival diseases 946 (17.46%), Cataract & Pseudophakia 620 (11.45%), Lid diseases 262 (4.84%), Lacrimal disease 109 (2.01%), Corneal diseases 108 (1.99%), Uveitis 89 (1.64%), Retinal diseases 72 (1.32%), Ocular Trauma 51 (0.95%), Glaucoma 31 (0.57%), Ocular motility disorder 8 (0.15%) and others 42 (0.79%).

Conclusion: In this study the prevalent ocular diseases were refractive error, Conjunctival diseases, Cataract & Pseudophakia. Among the refractive errors, Presbyopia & Astigmatism found in most cases, followed by Myopia & Hypermetropia. It can be used as baseline study for those who need to conduct similar studies in other eye care hospitals in Bangladesh and for making regional health policy.

Keywords: Ocular diseases, Prevalence of ocular diseases, Pattern of ocular diseases

Journal of Green Life Med. Col. 2023; 8(1): 7-12

Introduction:

Ocular disease patterns vary between developing and developed nations, as well as frequently within

1. Salma Parvin, Associate Professor and Head, Department of Ophthalmology, Green life Medical College, Dhaka Bangladesh.
2. Mafruha Afrin, Associate Professor, Department of Ophthalmology, Green life Medical College, Dhaka Bangladesh.
3. Kashana Akhter, Registrar, Department of Ophthalmology, Green life Medical College, Dhaka Bangladesh.
4. Sheikh Ashikul Islam, Medical Officer, Department of Ophthalmology, Green life Medical College, Dhaka Bangladesh.

Address of Correspondence: Salma Parvin, Associate Professor and Head, Department of Ophthalmology, Green life Medical College, Dhaka Bangladesh. email: salma99parvin@gmail.com

Received: 30.03.2024

Accepted: 06.07.2024

communities. The visually impaired 285 million people worldwide are projected to have a disability, out of which 246 million people with impaired vision and 39 million blind people.¹⁻⁵ Various articles have emphasized that 80% of the worldwide burden of visual impairment can be possible to reduce by treatment.⁶ The primary causes of blindness worldwide include cataracts, glaucoma, uncorrected refractive errors, and age-related macular degeneration of the retina. Other significant factors include trachoma, diabetic retinopathy, and corneal opacities. There are avoidable and treatable conditions like cataract, Refractive error still contribute 62.6%.⁷ According to CDC, the most common type of

eye issues in the US are refractive error. Myopia, hyperopia, astigmatism, and presbyopia are all refractive errors that can be corrected with eyeglasses, contact lenses, or in some cases surgery. According to the National Eye Institute, 150 million Americans vision might be improved with the proper refractive correction. Other common eye disorder and diseases included Macular degeneration, often called age-related macular degeneration (AMD), Cataract, Diabetic retinopathy (DR), Glaucoma, Amblyopia and Strabismus.⁸ Dry eye, allergic conjunctivitis, glaucoma, watery eyes, night blindness, vision loss or blindness, blepharitis, myopia, hazy vision, and cataract are among the eye conditions that are frequently seen in Bangladesh.⁹ Recent studies on the prevalence of refractive errors found that 20.6% of adults (30 years of age or older) had hyperopia (greater than +0.5 D) and 22% had myopia (less than -0.5 D).¹⁰ One of the primary health problem is visual impairment, which affects an estimated 253 million people globally, of whom 36 million are blind. However, over 80% of these disorders, vision impairment can be avoided or treated.¹¹

According to the World Health Organization (WHO), Bangladesh is part of the South East Asia region and that has about one-fourth of the world's population. The government hospital, private hospitals, and clinics local and international non-governmental organizations (NGO), and charitable organizations are providing eye care services in Bangladesh.¹² Poor ocular health knowledge has been revealed to be a major factor in the ongoing disparity between disease prevalence and service uptake.¹³

This study aimed to identify prevalence and patterns of ocular diseases among patients who visited eye OPD at Green life medical college. This study findings may help for making regional health policy.

Methods:

This was an observational descriptive study. The study place was outpatient department of Ophthalmology of Green Life Medical College and Hospital, Dhaka, Bangladesh. The study duration was 4 years, conducted from April 2018 to May 2022. Total 5417 patient's data were collected from the clinical record. Both descriptive and inferential statistics were performed by using MS Excel and SPSS-23 software.

Results:

In this study among 5417 patients, maximum 1951 (36.01%)

ocular diseases patients were from middle age group 39 to 60 years followed by 1947 (35.94%) were from adolescent age group 19 to 38, 1078 (19.90%) were from child (0-18) age group and only 441 (8.14%) from old age group (61-95). The age distribution of patients with ocular diseases are shown below Figure 1.

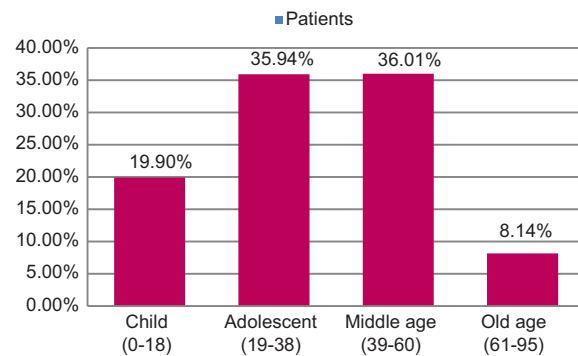


Figure 1: Age distribution of patients with ocular diseases (N=5417)

Figure 2 shows that among the 5417 patients with ocular diseases highest 3040 (56.12%) were female and 2377 (43.88%) were male.

Pattern and prevalence of different types and subtypes of ocular diseases are shown in table I to table X.

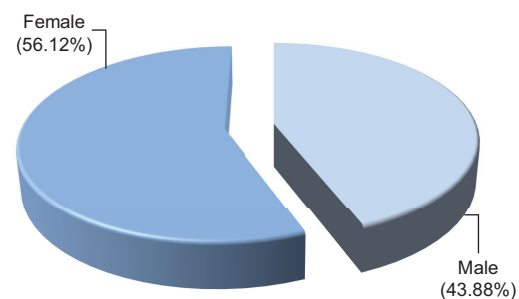


Figure 2: Sex of the Patients with Ocular disease (N=5417)

Figure 3 shows that among the 5417 patients with ocular diseases highest 3079 (56.83%) were refractive error followed by Conjunctival diseases 946 (17.46%), Cataract & Pseudophakia 620 (11.45%), Lid diseases 262 (4.84%), Lacrimal disease 109 (2.01%), Corneal diseases 108 (1.99%), Uveitis 89 (1.64%), Retinal diseases 72 (1.32%), Ocular Trauma 51 (0.95%), Glaucoma 31 (0.57%), Ocular motility disorder 8 (0.15%) and others 42 (0.79%).

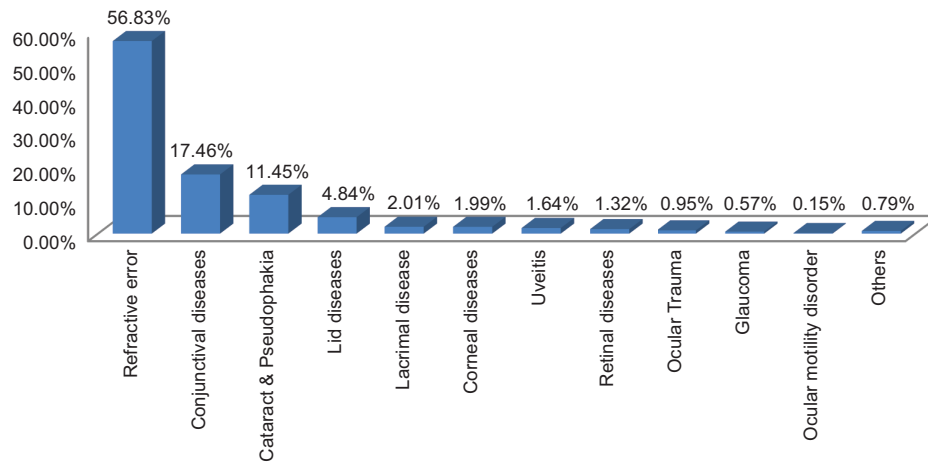


Figure 3: Four years Prevalence of Leading Ocular Diseases in OPD of Ophthalmology Department of Greenlife Medical College

Patient attended with Presbyopia(34.2%), Astigmatism(31.4%), Myopia(26.6%), Hypermetropia(7.8%)

Table I
Distribution of different refractive errors (n=3079)

Sub Type	Male(n)	Male %	Female(n)	Female %	Total %	Total patients
Hypermetropia	174	5.7	66	2.1	7.8	240
Astigmatism	406	13.2	562	18.3	31.4	968
Myopia	488	15.9	330	10.7	26.6	818
Presbyopia	390	12.7	663	21.5	34.2	1053
Total	1458	47.4	1621	52.6	100.0	3079

Acute anterior uveitis (74.16%), Posterior uveitis (24.72%), Intermediate uveitis (1.12%).

Table II
Different types of Uveitis (n=89)

Sub Type	Male(n)	Male %	Female(n)	Female %	Total %	Total patients
Acute Anterior Uveitis	34	38.2	32	35.96	74.16	66
Intermediate Uveitis	0	0	1	1.12	1.12	1
Posterior Uveitis	10	11.24	11	13.48	24.72	22
Total	44	49.44	45	50.56	100	89

Chalazion (34.0%), Blepharitis(25.2%), MGD (21.4%), Sty (12.6%), Internal Hordeolum (6.9%)

Table III
Distribution of different Eye Lid diseases (n=262)

Sub Type	Male(n)	Male %	Female(n)	Female %	Total %	Total patients
Blepharitis	28	10.7	38	14.5	25.2	66
Chalazion	45	17.2	44	16.8	34.0	89
Internal Hordeolum	7	2.7	11	4.2	6.9	18
Meibomian Gland Dysfunction	29	11.1	27	10.3	21.4	56
Stye	8	3.1	25	9.5	12.6	33
Total	117	44.7	145	55.3	100.0	262

Conjunctivitis (95.70%), Sub Conj. Haemorrhage (3.00%), Pterygium (1.4%)

Table IV
Conjunctival disease (n=946)

Sub Type	Male(n)	Male %	Female(n)	Female %	Total %	Total patients
Conjunctivitis	354	37.4	551	58.2	95.7	905
Pterygium	7	0.7	6	0.6	1.4	13
SubconjunctivalHaemorrhage	11	1.2	17	1.8	3	28
Total	372	39.3	574	60.7	100.0	946

Cataract (68.70%), Pseudophakia (31.30%).

Table V
Cataract & Pseudophakia (n=620)

Sub Type	Male(n)	Male %	Female(n)	Female %	Total %	Total patients
Cataract	150	24.2	276	44.5	68.7	425
Pseudophakia	100	16.1	94	15.2	31.3	194
Total	250	40.3	370	59.7	100.0	620

Congenital Naso Lacrimal duct obstruction (70.60%), Chronic dacryocystitis (29.40%)

Table VI
Lacrimal disease (n=109)

Sub Type	Male(n)	Male Percent	Female (n)	Female Percent	Total%	Total patients
Chronic Dacryocystitis	6	5.5	26	23.9	29.4	32
Congenital Naso Lacrimal Duct Obstruction	29	26.6	48	44.0	70.6	77
Total	35	32.1	74	67.9	100.0	109

Viral keratitis (50.00%), Corneal abrasion (27.80%), Foreign body in cornea (16.70%), Corneal Ulcer (5.6%).

Table VII
Pattern of Corneal disease (n=108)

Sub Type	Male(n)	Male Percent	Female(n)	Female Percent	Total%	Total patient
Corneal Abrasion	9	8.3	21	19.4	27.8	30
Corneal Ulcer	1	0.9	5	4.6	5.6	6
Foreign Body	10	9.3	8	7.4	16.7	18
Viral Keratitis	23	21.3	31	28.7	50.0	54
Total	43	39.8	65	60.2	100.0	108

Diabetic Retinopathy is more common (86.10%) than Macular Dystrophy (8.30%), Hypertensive Retinopathy (4.2%) & Macular Oedema (1%) respectively

Table VIII
Distribution of different Retinal disease (n=72)

Sub Type	Male(n)	Male Percent	Female(n)	Female Percent	Total %	Total patients
Diabetic Retinopathy	35	48.6	27	37.5	86.1	62
Hypertensive Retinopathy	1	1.4	2	2.8	4.2	3
Macular Dystrophy	4	5.6	2	2.8	8.3	6
Macular Odema	1	1.4	0	0.0	1.4	1
Total	41	56.9	31	43.1	100.0	72

Ocular trauma mostly blunt trauma (96.10%) then chemical injury (3.90%)

Table IX
Pattern of Ocular Trauma (n=51)

Sub Type	Male(n)	Male Percent	Female(n)	Female Percent	Total %	Total patient
Blunt Trauma	18	35.3	31	60.8	96.1	49
Chemical Injury	2	3.9	0	0.0	3.9	2
Total	20	39.2	31	60.8	100.0	51

Discussion:

In this study, among the 5417 patients with ocular diseases highest 3040 (56.12%) were female and 2377 (43.88%) were male. This deviates from the overall finding in the majority of studies, which states that in developing nations, fewer females than males are seen at medical clinics.¹⁴ But a comparable study in Nigeria also revealed a predominance of women.

In our situation, this may be related to the independent women's lifestyle trend that is currently prevailing in our society. Also, the hospital's location makes it accessible to women who do not need to rely on their family members to accompany them to the hospital. The community is also well-aware of the importance of regular eye checkup and prevention of various blinding ocular disorders, especially in urban areas. This was also observed in a study when 10.07% of the participants visited the hospital solely for a checkup on their eyes, with no other evident symptoms or pathological or clinical features.^{15,16}

In this study of 5417 patients, the highest 1951 (36.01%) ocular diseases patients were from middle age group 39 to 60 years followed by 1947 (35.94%) were from adolescent age group 19 to 38, 1078 (19.90%) were from child (0-18) age group and only 441 (8.14%) from old age group (61-95). This results suggests that, adults experienced ocular issues at higher rates than kids. Similar trends were documented by Ajaiyeoba and Scott.¹⁷ The location of the urban city is likely to be the cause of this. According to our study region, the majority of the population was urban, and since our study area is also near some colleges and universities, we discovered the largest concentration of students here. Nowadays, people in urban areas live diverse lifestyles and have more mindful adult lives. Children may not be able to appropriately communicate their issues, which could explain why they may not present to the hospital until the symptoms are severe enough to qualify as ocular morbidity.

In our study, the most common ocular disease among the 5417 patients was refractive error, which accounted for

(3079, 56.83%) of all cases. Refractive error was then followed by conjunctival diseases (946, 17.46%), cataract and pseudophakia (620, 11.45%), lid diseases (262, 4.84%), lacrimal disease (109, 2.01%), corneal diseases (108, 1.9%), uveitis (89, 1.64%), retinal diseases (72, 1.32%), ocular trauma 51 (0.95%), glaucoma (31, 0.57%), ocular motility disorder (8, 0.15%) and others (42, 0.79%).

A likely explanation for increased incidence of refractive error in our study is abundance of schools and colleges in close vicinity of our hospital. High rate of cataract, conjunctival and lid disorders in this study may be related to the humid and dusty climate of Dhaka City. This is consistent with other research conducted at hospitals and with questionnaires conducted among students about their eye health.^{15,18,19} Globally, uncorrected refractive errors are a significant contributor to eye health issues.²⁰ It affects life quality and has socioeconomic and educational repercussions. Studies from Sudan^{21, 22,23} have also demonstrated that refractive errors were previously undetected as a major source of eye disorders in school-aged children. Ocular trauma has recently received attention as a significant contributor to visual morbidity.²⁴ The prevalence of trauma was 0.95% in this study, which is close to Olukorde and Oluymka's study, which found that trauma rates were less than 3%. However, other studies found that ocular trauma was the third^{25,26} most common cause of ocular morbidity.

The prevalence of retinal illness observed in this study (1.32%) is close to that observed in clinical investigations conducted in other developing countries; these studies revealed prevalence rates ranging from 3.9% in South Eastern Nigeria²⁷ to 12.5% in Ethiopia.²⁸ Diabetic retinopathy was the most commonly observed retinal disorders in our OPD. Geographical and environmental factors, as well as sedentary style of living among urban population, may be the likely causes of these.

Conclusion:

In this study, the prevalent ocular diseases were refractive error, Conjunctival diseases, Cataract & Pseudophakia.

Among the refractive errors, Presbyopia & Astigmatism found in most cases, followed by Myopia & Hypermetropia. The second most disease found is conjunctivitis then cataract & pseudophakia. Diseases of eye lid, uveitis, glaucoma, retinal diseases, ocular trauma are found in small amount. This pattern of ocular disease, therefore can make the strategy to ensure better service and quality treatment to the patients. It also can be used as baseline study for those who need to conduct similar studies in other eye care hospitals in Bangladesh and for making regional health policy.

References:

1. Paudel P, Ramson P, Naduvilath T, Wilson D, Phuong HT, et al. (2014) Prevalence of vision impairment and refractive error in school children in Ba Ria-Vung Tau province, Vietnam. *ClinExpOphthalmol* 42(3): 217-226.
2. Edussuriya K, Sennanayake S, Senaratne T, Marshall D, Sullivan T, et al. (2009) The prevalence and causes of visual impairment in central Sri Lanka the Kandy Eye study. *Ophthalmology* 116(1): 52-56.
3. Katibeh M, Pakravan M, Yaseri M, Pakbin M, Soleimanizad R (2015) Prevalence and Causes of Visual Impairment and Blindness in Central Iran; The Yazd Eye Study. *J Ophthalmic Vis Res* 10(3): 279-285.
4. Mariot SP (2010) Global data on visual impairments 2010. World Health Organization.
5. Resnikoff S, Pascolini D, Etyaale D, Kocur I, Pararajasegaram R, et al. (2004) Global data on visual impairment in the year 2002. *Bull World Health Organ* 82(11): 844-851.
6. (2017) National Programme for Control of Blindness.
7. Lawrence JM (2014) Pattern of ocular findings among patients aged 40 years and above attending eye clinic at Juba teaching hospital in Southern Sudan. University of Nairobi.
8. Common eye disorders and diseases. (2020, June 4). Cdc.gov. <https://www.cdc.gov/visionhealth/basics/ced/index.html>
9. Rubel MR, Ashrafudoulla M, Mizan MF, Fuad F, Islam MS, Parvin S. Pharmacovigilance study on the different drugs used for the management of eye disorders in Bangladesh. *The Pharma Innovation*. 2017;6:173-180
10. Rupert R. A. Bourne, Brendan P. Dineen, Deen M. NoorulHuq, Syed M. Ali, Gordon J. Johnson; Correction of Refractive Error in the Adult Population of Bangladesh: Meeting the Unmet Need. *Invest. Ophthalmol. Vis. Sci.* 2004;45:410-417.
11. Al-Lahim WA, Al-Ghofaili RS, Mirghani H, ALBalawi H. Evaluation of Awareness and Attitudes towards Common Eye Diseases among the General Population of Northwestern Saudi Arabia. *The Egyptian Journal of Hospital Medicine*. 2018;70:1201-1208.
12. Bourne RR, Dineen BP, Ali SM, Huq DM, Johnson GJ. Outcomes of cataract surgery in Bangladesh: results from a population based nationwide survey. *Br J Ophthalmol*. 2003;87:813-819.
13. Shrestha MK, Guo CW, Maharjan N, Gurung R, Ruit S. Health literacy of common ocular diseases in Nepal. *BMC Ophthalmol*. 2014;14:2.
14. Kawuma M. Eye diseases and blindness in Adjumani refugee settlement camps, Uganda. *East Afr Med J* 2000;77:580-2.
15. Oluokorde OA, Oluymka JS. Pattern of eye diseases in air force hospital in Nigeria. *Pak J Ophthalmol* 2012;28:144-8
16. Lakho KA, Mohamed Ali AB. Pattern of eye diseases at tertiary eye hospital in Sudan (Makah Eye Hospital, Khartoum). *AlbasarInt J Ophthalmol* 2015;3:15-8.
17. Ajaiyeoba AI, Scott SC. Risk factors associated with eye diseases in Ibadan, Nigeria. *Afr J Biomed Res* 2002;5:1-3.
18. Murad MA, Alam MS, Miah AKMA, Akter MS, Kabir MH. Pattern of eye diseases in a tertiary hospital in a suburban area: A retrospective study. *Orion Med J* 2007;28:492-4.
19. Sarita T, Sachin D. A pattern of ocular morbidity in patients attending an ophthalmic clinic in a rural part of western Nepal. *J Nobel Med Coll* 2012;2:27-30.
20. Dandona L, Dandona R, Naduvilath TJ, McCarty CA, Srinivas M, Mandal P, et al. Burden of moderate visual impairment in an urban population in southern India. *Ophthalmology* 1999;106:497-504
21. Balarabe AH, Adamu S, Musa R. Presbyopia among health workers in a tertiary hospital in north western Nigeria. *Sub-Saharan Afr J Med* 2015;2:10-3
22. Rushood AA, Azmat S, Shariq M, Khamis A, Lakho KA, Jadoon MZ, et al. Ocular disorders among schoolchildren in Khartoum State, Sudan. *East Mediterr Health J* 2013;19:282-8
23. Catherine UU. Pattern of ocular morbidity in Nigeria. *Asian Pac J Trop Dis* 2013;3:164-166.
24. Asaminew T, Gelaw Y, Alemseged F. A 2-year review of ocular trauma in Jimma University specialized hospital. *Ethiop J Health* 2009;19:67-73.
25. Bodunde OT, Onabolu OO. Childhood eye diseases in Sagamu. *Niger J Ophthalmol*. 2004;12:6-9.
26. Ezegwui IR, Onwasigwe EN. Pattern of eye disease in children at Abakaliki, Nigeria. *Int J Ophthalmol* 2005;5:1128
27. EzeBI, Uche JN, ShiweobiJO. The burden and spectrum of vitreo-retinal diseases among ophthalmic outpatients in a resource deficient tertiary eye care setting in South-Eastern Nigeria. *Middle East Afr J Ophthalmol* 2010;17:46-55.
28. Teshome T, Melaku S, Bayu S. Pattern of retinal diseases at a teaching eye department, Addis Ababa, Ethiopia. *Ethiop Med J* 20

Breadth & Annular Circumference of Tricuspid Valve of Human Heart – A Morphological Study

REZAR¹, ARAS²

Abstract

Introduction: One of the oldest pictures of the tricuspid valve was found in *De Humanis Corpori Fabrica* written by Vesalius in the 16th century that, the tricuspid valve had three leaflets. The anatomy of the tricuspid valve is highly sophisticated, but understanding it may be helpful in the practice of cardiac surgery. Repair of tricuspid valve is usually performed by the cardiac surgeons instead of valve replacement surgery. Therefore knowledge of a detailed morphology of this valve is more and more necessary. The objective of this study was to provide information about the age related morphological changes of breadth & annular circumference of the tricuspid valve leaflets, which will serve as reference data for further studies and clinical uses in patients with various cardiac valvular diseases.

Methods: This cross sectional descriptive study was conducted in the Department of Anatomy, Dhaka Medical College, Dhaka from July 2010 to June 2011. The present study was conducted on 70 human hearts collected from unclaimed dead bodies undergoing routine autopsy examination in the morgue of the Department of Forensic Medicine and Department of Anatomy, Dhaka Medical College, Dhaka. All samples were collected within 24-36 hours after death. The samples were divided into three different age groups. Group A (18-40 years), group B (41-64 years) and group C (≥ 65 years). All the samples were studied morphologically.

Results: In the present study, the mean \pm SD breadth of the anterior leaflet of tricuspid valve was 32.27 ± 4.52 , 34.06 ± 3.79 and 35.26 ± 2.77 mm in group A, group B and group C respectively. The mean \pm SD breadth of the posterior leaflet was 27.40 ± 4.10 mm in group A, 29.77 ± 3.53 mm in group B and 30.76 ± 2.77 mm in group C. The mean \pm SD breadth of the septal leaflet was 28.23 ± 2.28 , 29.01 ± 2.33 and 30.44 ± 2.60 mm in group A, B and C respectively. The mean \pm SD annular circumference of the tricuspid valve was 102.97 ± 5.83 , 107.43 ± 4.71 and 109.06 ± 7.04 mm in group A, group B and group C respectively.

Conclusion: Breadth of the posterior leaflet of tricuspid valve increased significantly between age group A and B. The mean difference of annular circumference in between group A and B ($P < 0.01$) and group A and C ($P < 0.05$) was statistically significant, but between group B and C was not significant.

Keywords: Tricuspid valve, Breadth of valve leaflets, Human heart

Journal of Green Life Med. Col. 2023; 8(1): 13-16

Introduction:

The tricuspid valve is situated on the right atrioventricular orifice and joins two right chambers of the heart which differ greatly in shape.¹ Traditionally the tricuspid valve has been described as being formed by three leaflets and three or more papillary muscles.² The tricuspid valve is more differentiated

during evolutionary development than the mitral valve.³ The tricuspid valve is more complex than the mitral valve – not only because it is a trileaflet valve but, more importantly, because of its high variability.⁴ Tricuspid valve dysfunction can occur from morphological variations in the valve or from functional aberrations of the myocardium.⁵ Accurate knowledge of the morphology and morphometry of the tricuspid valve is mandatory for differentiation between functional and organic tricuspid regurgitation.⁶ The objective of this study was to describe precisely different anatomical structures of human tricuspid valve, with variations in relation to age. The study findings may help in conservative surgical techniques and for the development and manufacture of prostheses similar to the natural valve.

1. Rumana Reza, Professor, Department of Anatomy, Green Life Medical College

2. Shamim Ara, Professor, Department of Anatomy, Holyfamily Medical College

Address of Correspondence: Rumana Reza, Professor, Department of Anatomy, Green Life Medical College, email: rumanaana@gmail.com

Received: 29.08.2023

Accepted: 06.07.2024

Methods:

The samples of human heart were collected from unclaimed dead bodies undergoing routine autopsy examination in the morgue of Department of Forensic Medicine and Department of Anatomy, Dhaka Medical College, within 24-36 hours of death after legal formalities. During collection of the samples, appropriate age, sex and cause of death were noted from the morgue's record book. In the dissection room, the hearts were cleaned by removing all the associated tissues. The specimens were washed thoroughly with tap water and gently squeezed to remove blood clots from the cavity of the heart and from the lumen of the blood vessels as much as possible.

Preservation: Then the samples were kept in a jar containing 10% formalin saline solution and a tag was given with the identification number and label showing age, sex, cause of death and date of collection etc. For convenience of description in relation to age the collected samples were divided into 3 groups according to Skwarek and Hreczecha.¹

Table-I
Age distribution in different groups

Group	Age limit	No. of samples
Group A	18-40 years	36
Group B	41-64 years	30
Group C	≥65 years	04

Measurement of the breadth & annular circumference of tricuspid valve leaflets: The samples were taken in a dissection tray. The adipose tissue and coronary vessels were cleaned from the coronary sulcus with small pointed scissors and forceps. An incision was made along the upper limit of whole extent of the coronary sulcus with the help of a sharp scissors and a Bard Parker blade. Then the two atrioventricular orifices along with their valves (tricuspid and mitral) were exposed from above.

To open the right atrioventricular orifice (tricuspid orifice), the heart was dissected from the right end of first cut at the coronary sulcus & extended along the inferior border of heart near the anteroposterior commissure of the tricuspid valve (following Kocak et al. 2004¹⁰). Then tricuspid annular ring was excised and the papillary muscles were separated from the myocardium at their bases. Thus the tricuspid valve complex was completely removed from the rest of the heart.

The isolated tricuspid valve was taken on a 'waxed floor' tray. The antero-septal commissure was divided at its mid

region.⁷ Breadth of the leaflets was measured along the annular margin between the commissures by digital slide calipers.² Annular circumferences were estimated by summation of breadth of the leaflets and width of commissures at their bases (following Silver et al. 1971²).

Results: The mean ± SD breadth of the anterior leaflet of tricuspid valve was 32.27 ± 4.52 mm in group A, 34.06 ± 3.79 mm in group B and 35.26 ± 2.77 mm in group C. The mean ± SD breadth of the posterior leaflet was 27.40 ± 4.10, 29.77 ± 3.53 and 30.76 ± 2.77 mm in group A, B and C respectively. The mean ± SD breadth of the septal leaflet was 28.23 ± 2.28, 29.01 ± 2.33 and 30.44 ± 2.60 mm in group A, B and C respectively. The mean difference of breadth of anterior leaflet and septal leaflet among three age groups was not statistically significant. But mean difference of breadth of posterior leaflet in between group A and B was statistically significant ($P < 0.05$), but mean difference in between group A & C and group B & C was not significant.

The mean ± SD annular circumference of the tricuspid valve was 102.97 ± 5.83, 107.43 ± 4.71 and 109.06 ± 7.04 mm in group A, group B and group C respectively. The mean difference of annular circumference in between group A and B ($P < 0.01$) and group A and C ($P < 0.05$) was statistically significant, but between group B and C was not significant.



Figure 1: Showing breadth of the anterior leaflet indicated by a black dotted line and measured by a digital slide calipers.

Table II

Breadth of the anterior, posterior and septal leaflets of the tricuspid valve in different age groups

Age group	Breadth (mm)		
	Anterior Mean±SD	Posterior Mean±SD	Septal Mean±SD
A (n=36)	32.27±4.52 (23.24†41.46)	27.40±4.10 (14.19†36.14)	28.23±2.28 (24.58†35.34)
B (n=30)	34.06±3.79 (26.31†41.46)	29.77±3.53 (21.14†36.54)	29.01±2.33 (25.55†36.64)
C (n=4)	35.26±2.77 (31.88 38.47)	30.76±2.77 (27.51 34.28)	30.44±2.60 (28.65 34.21)
	<i>P value</i>	<i>P value</i>	<i>P value</i>
A vs B	>0.05 ^{ns}	<0.05 ^s	>0.10 ^{ns}
A vs C	>0.10 ^{ns}	>0.05 ^{ns}	>0.05 ^{ns}
B vs C	>0.50 ^{ns}	>0.50 ^{ns}	>0.10 ^{ns}

Figures in parentheses indicate range. Comparison between age groups done by One way ANOVA (PostHoc), ns = not significant, S = significant

- Group A : Age 18-40 years.
- Group B : Age 41-64 years.
- Group C : Age 65 years and above

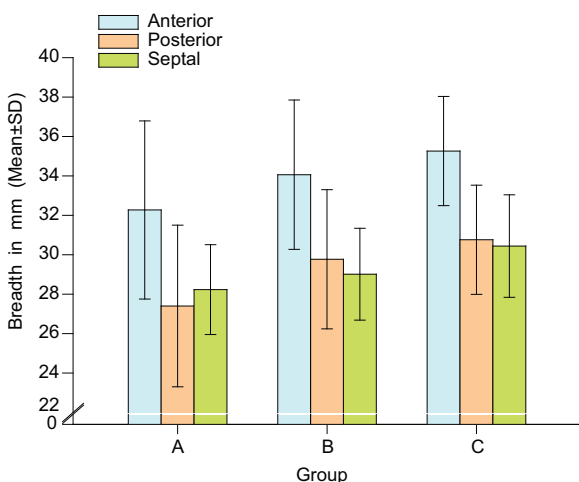


Figure 2: Breadth of the anterior, posterior and septal leaflets of the tricuspid valve in different age groups

- Group A : Age 18-40 years.
- Group B : Age 41-64 years.
- Group C : Age 65 years and above

Table III

Annular circumference of the tricuspid valve in different age groups

Age group	Circumference (mm) (Mean±SD)
A (n=36)	102.97±5.83 (93.37†114.15)
B (n=30)	107.43±4.71 (98.13†115.32)
C (n=4)	109.06±7.04 (98.79†114.51)
	<i>P value</i>
A vs B	<0.01 ^s
A vs C	<0.05 ^s
B vs C	>0.50 ^{ns}

Figures in parentheses indicate range. Comparison between age groups done by One way ANOVA (PostHoc), ns = not significant, S = significant

- Group A : Age 18-40 years
- Group B : Age 41-64 years
- Group C : Age 65 years and above

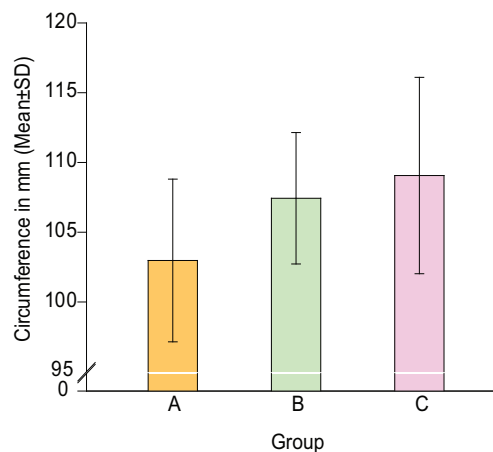


Fig. 3 Annular circumference of the tricuspid valve in different age groups

- Group A : Age 18-40 years.
- Group B : Age 41-64 years.
- Group C : Age 65 years and above

Discussion:

The mean breadth of the anterior leaflet of tricuspid valve observed by Skwarek et al.¹ was 33.56 ± 6.32 mm (group A), 34.68 ± 6.41 mm (group B) and 34.20 ± 6.57 mm (group C) and their results were similar to the present study which revealed that the mean breadth was 32.27 ± 4.52 mm (group A), 34.06 ± 3.79 mm (group B) and 35.26 ± 2.77 mm (group C)

C). Silver⁸ and Waller & Schlant⁹ described that the average breadth was 3.7 cm which was similar to the present study. Mean breadth observed by Silver et al.² and Kocak et al.¹⁰ was 3.9 ± 0.8 cm and these values were higher than the present study. The mean breadth observed by Begum¹¹ was 3.24 cm and her findings have similarity with the present study.

Mean breadth of the posterior leaflet of tricuspid valve observed by Kocak et al.¹⁰ and Silver et al.² was 3.1 ± 0.7 cm and 3.0 ± 0.9 cm respectively and their results were similar to the present study in which the mean breadth is 29.77 ± 3.53 mm. Average breadth described by Silver⁸ and Waller & Schlant⁹ was 7.5 cm which was higher than the present study. Mean breadth observed by Skwarek et al.¹ ranged from 28.56 ± 7.10 mm to 38.4 ± 11.28 mm. Their minimum values have similarity with the present study but maximum values are much higher. Mean breadth measured by Begum¹¹ was 3.32 cm and the result was similar to the present study.

The mean breadth of septal leaflet observed by Begum¹¹ was 2.87 cm and the result was similar to the present study. Silver et al.², Kocak et al.¹⁰, Silver⁸ and Waller & Schlant⁹ observed that the mean values 3.7 cm, 3.9 cm, and 3.6 cm respectively and all these findings were higher than the present study. In age group A, B and C the mean breadth of septal leaflet measured by Skwarek et al.¹ ranged from 28.5 ± 4.64 to 29.98 ± 5.29 mm and the result was similar to the present study where the mean breadth was 28.23 ± 2.28 to 30.44 ± 2.60 mm.

The annular circumference of tricuspid valve described by Waller & Schlant (1998) and Silver (1983) was 10–12.5 cm and 12cm respectively and their results were similar to the present study where the mean \pm SD annular circumference of the tricuspid valve was 102.97 ± 5.83 mm. Mean annular circumference of the tricuspid valve observed by Skwarek et al. (2008) in different age groups ranged from 104.06 to 120.90 mm which conforms to the present study in which the mean \pm SD annular circumference ranged from 102.97 ± 5.83 to 109.06 ± 7.04 mm. Kocak et al. (2004) observed that the tricuspid valve annular circumference of the individuals whose age ranged from 17-68 years and the mean was 12.4 ± 1.1 cm and the result was similar to the present study. The mean annular circumference of fresh and fixed heart observed by Tei et al. (1982) in an autopsy study was 13.5 and 12.2 cm respectively and the result was similar to the present study. Silver et al. (1971) observed that the mean annular

circumference was 114 ± 11 mm which has similarity with the values of the present study.

Conclusion:

Breadth of the posterior leaflet of tricuspid valve increased significantly between age group A (18-40 years) and B (41-64 years). The mean difference of annular circumference in between group A and B ($P < 0.01$) and group A and C (≥ 65 years) ($P < 0.05$) was statistically significant.

References:

1. Skwarek M, Hreczecha J, Dudziak M et al. Morphometric features of the right atrio-ventricular orifice in adult human hearts. *Folia Morphol*, 2008. vol. 67, No.1: p53-7.
2. Silver M.D, Lam J.H.C, Ranganathan N and Wigle ED. Morphology of the human tricuspid valve. *Circulation* 1971; 43: p333-48.
3. Skwarek M, Dudziak M, Hreczecha J and Grzybiak M. The connection between the papillary muscles and leaflets of the tricuspid valve. *Folia Morphol* 2006; 65(4): 322-28.
4. Joudinaud TM, Flecher EM and Duran CMG. Functional terminology for the tricuspid valve. *The Journal of Heart Valve Disease* 2006; 15: p382-88.
5. Rahman MT, Das M, Ullah M et al. A case of severe tricuspid stenosis of rheumatic origin. *Cardiovasc. J.* 2011; 3(2): p235-38.
6. Grondin P, Lepage G, Castonguay Y, Meere C. The tricuspid valve: a surgical challenge. *J Thorac Cardiovasc Surg.* 1967; 53 (1): 7 – 20.
7. Lam JHC, Ranganathan N, Wigle ED and Silver MD. Morphology of the human mitral valve: I. Chordae tendineae: A new classification. *Circulation* 1970; 41: p449-58.
8. Silver MD. Chapter 1 Gross examination and structure of the heart (vol- 1). In: Meredith M. Silver. *Cardiovascular pathology*. 1st ed. New York: Churchill Livingstone; 1983. p1-104.
9. Waller BF and Schlant RC. Chapter 2 Anatomy of the heart. In: Alexander RW, Schlant RC and Fuster V. *Hurst's The Heart, arteries and veins*. 9th ed. United States of America: McGraw Hill; 1998. p19-48.
10. Kocak A, Govsa F, Aktas EO, Boydak B and Yavuz IC. Structure of the human tricuspid valve leaflets and its chordate tendineae in unexpected death. A forensic autopsy study of 400 cases. *Saudi Med J.* 2004; 25(8): p1051-59.
11. Begum J. An anatomical study of atrioventricular valves, muscular part of interventricular septum and papillary muscle of the heart among the adult Bangladeshi people. Dhaka: Institute of Post-Graduate Medicine and Research. Dhaka, Bangladesh. 1996; p15-156.
12. Tei C, Pilgrim JP, Shah PM, Ormiston JA and Wong M. The tricuspid valve annulus: study of size and motion in normal subjects and in patients with tricuspid regurgitation. *Circulation* 1982; 66: 665 – 71.

Vasomotor Rhinitis: An Overview

KHANAMA¹, ISLAM NN², LUBANAN³

Abstract

Vasomotor rhinitis (VMR) is one of the most prevalent forms of non-allergic rhinitis. Non-allergic rhinitis (NAR) is recognized by the presence of nasal symptoms together with negative allergic tests, indicating no evidence of allergic disease. This article aims to review VMR, illustrate the potential causes and, provide a thorough analysis of the pathophysiological background, diagnostic approach, and main treatment options. NAR comprehends multiple distinct conditions that may even co-exist with allergic rhinitis (AR). They are indifferent in their presentation and treatment. There are many conditions that have similar presentations to both NAR and AR, including asthma, nasal polyps, autoimmune diseases, metabolic states, hormonal diseases, genetic conditions, emotional condition and immunodeficiency, but there should be a differentiation between NAR and AR. There are a number of causes which causes NAR to have symptoms that are similar to AR like nasal congestion, pruritus, rhinorrhea, nose itching, eye itching, eye congestion, chest congestion, cough but the allergic tests are all negative. In this comprehensive overview, we perceive a better understanding of the role of neuropeptide & nitric oxide in the development of VMR and apprehend a clinical diagnosis aided by investigations. Evidence-based treatment approaches for VMR comprise of environmental intervention, pharmacotherapy, and in refractory cases, surgical recourse, hence a tailored approach for each patient.

Keywords: Nasal congestion, Non-allergic rhinitis (NAR), Rhinorrhea, Vasomotor rhinitis (VMR)

Journal of Green Life Med. Col. 2023; 8(1): 17- 22

Introduction:

The nose and nasal cavities have a number of important functions. Airflow into the nasal passages is essential for both the senses of smell and respiration. The nasal passages also act as a filter for dust, allergen, bacteria, virus and protection of the lungs. Moreover, the relatively large surface area of the mucosa-covered turbinates acts to warm and humidify air prior to entry into the lungs. When airflow is considerably inhibited, all of these functions can be adversely affected. In rhinitis, a combination of nasal mucosal inflammation, oedema and increased mucous production can lead to such airflow obstruction. Rhinitis is a common inflammatory condition

of the nasal mucosa that frequently leads to several unpleasant symptoms, such as nasal itch, rhinorrhea, nasal congestion, sneezing, postnasal drip and irritation or discomfort in or around the nose and sometimes produce sinusitis.¹ Rhinitis occurs as 2 major types: allergic rhinitis and non-allergic rhinitis. The allergic type is caused by an environmental trigger (such as pollen, grains, molds, animal dander, trees, grass) and is classified as persistent or intermittent based on the duration of symptoms. Allergic rhinitis is diagnosed by verifying the causative allergen/antigen by skin and blood tests. The diagnosis should be confirmed with the patient's history as well.^{2, 3} NAR is a chronic rhinitis with clinical manifestations of local endonasal inflammation but without systemic allergic inflammation [negative allergy skin prick test (SPT), negative total IgE, and radioallergosorbent test (RAST)].^{4, 5}

NAR consists of multiple clinical entities. The most common type of NAR is considered to be vasomotor rhinitis, also known as nonallergic rhinopathy and idiopathic rhinitis. This type of rhinitis consists of approximately 71% of nonallergic rhinitis, with a worldwide

1. Afroza Khanam, Associate Professor, Department of Otorhinolaryngology, Green Life Medical College, Dhaka
2. Nashiat Nazrul Islam, Registrar, Department of Dermatology and Venereology, Green Life Medical College, Dhaka
3. Nasreen Lubana, Indoor Medical Officer, Department of Otorhinolaryngology, Green Life Medical College, Dhaka

Address of Correspondence: Afroza Khanam, Associate Professor, Department of Otorhinolaryngology, Green Life Medical College, Dhaka, email: afrozakhanam85@gmail.com

Received: 11.05.2024

Accepted: 06.07.2024

prevalence of 320-million people and without a clear interaction with the rates of comorbid asthma.⁶

In one series, VMR accounted for approximately 60% of the cases as the most commonly diagnosed form of NAR.⁷

Therefore the goal of this study was to summarize the causes of VMR, identify any associated pathogenic pathways, evaluate the investigations and outline the therapeutic options.

Methods:

This review article is based on the evidence obtained from sources such as PubMed, Google Scholar using the combination of keywords NAR, VMR. Searching articles focusing on the details of VMR was supported by the literatures, case reports and guidelines related to this topic. Exclusion criteria for articles were non-English language and studies in non-human populations. Types of Vasomotor Rhinitis

- Nonallergic rhinitis with eosinophilia syndrome (NARES): sign-symptoms are like allergic rhinitis.
- Hormonal-induced rhinitis: occurs in puberty, menstruation, pregnancy.
- Drug-induced rhinitis, including rhinitis medicamentosa: Antihypertensive drugs, such as beta-blockers, alpha-blockers, angiotensin-converting enzyme inhibitors and vasodilators can cause nasal congestion and stuffiness. Anticholinesterase drug such as neostigmine can result in nasal stuffiness. Aspirin and NSAID cause asthma. Oestrogen in contraceptive pills can lead to nasal obstruction. Overuse of topical nasal decongestants e.g. xylometazoline, oxymetazoline, leads to rhinitis medicamentosa.
- Gustatory rhinitis: characterized by mucoid or watery nasal discharge when hot spicy food is ingested.
- Emotional rhinitis: such as during grief, anxiety, humiliation etc.
- Senile rhinitis
- Occupational rhinitis: such as pyrethrum insecticide, acid anhydrides in adhesive industry, toluene in body spray, latex, grains, paint industries.
- Temperature change as in hot and cold.

Pathophysiology

There are significant knowledge gaps regarding the pathogenesis of vasomotor rhinitis. Generally it is thought to arise from an imbalance of autonomic input into the nasal mucosa. A primary stimulus results in nasal

congestion and/or rhinorrhea incited by tachykinins from the central nervous system, which also inhibit sympathetic mediators and thus further amplify the parasympathetic response^{8,9}, see Figure 1.

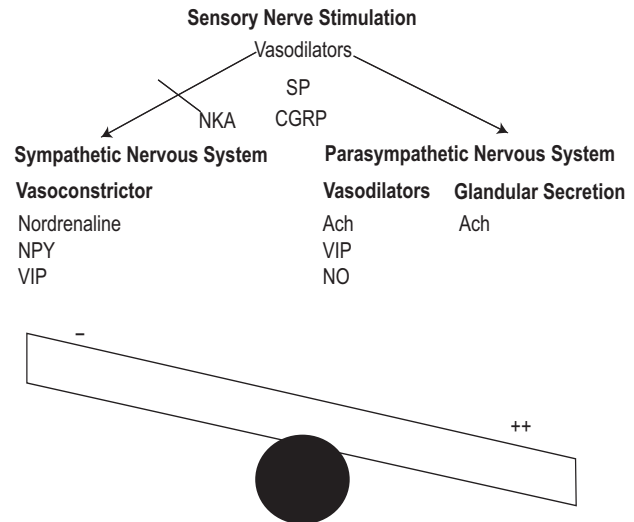


Figure 1: Pathogenesis

Sensory C-fiber stimulation leads to release of substance P (SP) and calcitonin-gene related peptides (CGRP). This in return leads to increased plasma excretion and glandular secretion (via acetylcholine and muscarinic receptors) manifesting as pain and stuffiness or congestion.¹⁰ Ozone augments nasal mucosal levels of SP and neurokinin A (NKA).¹¹ NKA also causes vascular smooth muscle contraction. Therefore, sensory stimulation generates release of SP, CGRP, NKA with subsequent nasal congestion and pain.

Elevated levels of neuropeptide tyrosine (NPY), vasoactive intestinal peptide (VIP), and SP in patients with irritant rhinitis maybe caused by cigarette smoke exposure.¹² NPY is principally distributed around blood vessels and intranasal administration of NPY has been shown to cause nasal vasoconstriction and a decrease in nitric oxide levels.¹³ Noradrenaline also acts to vasoconstrict nasal mucosal blood vessels. VIP is an inhibitory neurotransmitter that can cause vasodilatation and hypersecretion and thus congestion and rhinorrhea. Furthermore, Acetylcholine (Ach) release leads to glandular secretion and vasodilation.¹⁴

The significance of the free radical, nitric oxide (NO), in the context of rhinitis has been examined. NO is produced by nitric oxide synthase (NOS), which requires

nicotinamide adenine dinucleotide phosphate (NADPH) as a cofactor. NO is present only transiently therefore its presence and activity are deduced by the measure of NADPH diaphorase levels in tissue, which correlate with NOS activity. In patients with VMR, epithelial damage correlates with increased NADPH diaphorase activity and thus escalated NO activity. Collectively, these data advocate that NO, which has known cytotoxic effects, can cause epithelium damage. This damage could result in diminished mucociliary clearance, loss of tight junctions and basement membrane damage. This would in turn allow for increased reactivity of afferent trigeminal fibers, secretory and vascular reflexes resulting in the constellation of symptoms seen in VMR.^{15, 16}

Epidemiology

Three studies were identified that attempted to systematically subtype NAR by performing testing that included, at a minimum, nasal examination, skin testing for sensitivity to specific aeroallergens, total IgE, nasal cytology, and sinus x-rays.¹⁷⁻¹⁹ Each of these studies consists of examination of nasal cytology in an attempt to screen out NARES or eosinophilic rhinosinusitis.

The data from these 3 studies, when merged, gives a total of 200 NAR subjects. VMR was identified as the most common subtype, making up 71% of NAR diagnoses, while nonallergic rhinitis with eosinophilia syndrome (NARES) make up majority of the remaining diagnoses. Sex and age demographic data suggest a 2:1 female-to-male ratio and a higher mean age (40 years old) for VMR subjects as compared with that of allergic rhinitis subjects.^{17, 18, 20}

The studies by Mullarkey,¹⁷ Enberg,¹⁸ and Settupane¹⁹ unanimously support VMR as the most common NAR subtype, making up approximately 71% of NAR diagnoses, with NARES making up the greater part of the remaining NAR conditions. Applying the 71% frequency of VMR occurrence to the 20 million Americans who suffer from NAR, it would be estimated that VMR affects 14 million people in the United States. Applying the same frequency to the 450 million worldwide populations suffering from NAR yields an estimate of a worldwide prevalence of VMR of 320 million.

Clinical Features

Usually there is no history of allergies and infection. Typically adult onset, with sporadic or persistent nasal symptoms.

Features include:²¹

- Episodes of sneezing persisting all over the year
- Nasal congestion
- Watery nasal discharge or rhinorrhea
- Watery eye and itching in eye
- Throat clearing and itch
- Coughing and chest congestion
- Postnasal drip
- Headache
- Facial pressure

Patients with VMR fall into two groups; “runners” who have “wet” rhinorrhea, and “blockers” who have “dry” congestion.^{21, 22} These reactions can be triggered by strong smells like perfume and incense, particulate materials like vehicle exhaust and tobacco smoke, cold air, changes in temperature, humidity, barometric pressure, strong emotions, ingestion of alcohol and/or spicy foods, changes in hormone levels and hypothyroidism.²³

Physical exam reveals:²³

- boggy oedematous mucosa with clear mucoid secretions
- mucosal injection and lymphoid hyperplasia involving the tonsils, adenoids, and lingual tonsils may be present

Evaluation

The diagnosis is usually made following exclusion of AR.

The workup for vasomotor rhinitis includes skin testing and/or serum-specific IgE antibodies. As a diagnosis of exclusion, vasomotor rhinitis will typically have a negative skin test and serum antibodies for relevant allergens. Nasal cytology can yield information about cell types constituting the mucosa and help to identify the presence of inflammatory markers. Scrapings from the inferior turbinate, nasal lavage or nose blowing can provide epithelial cells for analysis. The presence of 5 to 25 eosinophils in high-powered fields is compatible with the diagnosis of nonallergic rhinitis with eosinophilia syndrome (NARES).²⁴

A validated questionnaire revealed that patients with onset of symptoms after the age of 35 years, a negative family history of allergies or atopy, and symptoms accompanying exposure to perfumes and fragrances had a 96% likelihood of having vasomotor rhinitis.²⁵

Nasal provocation testing involves exposing a patient to a respective allergen, assessing the clinical response, and

collecting objective data with rhinomanometry and acoustic rhinometry.²⁶

A computed tomography scan of the paranasal sinuses is a diagnostic option for patients with suspected sinus disease, and magnetic resonance imaging can assist with suspected mass lesions of the head and neck. However, in cases of vasomotor rhinitis, imaging will rarely reveal pathology and is not exceptionally helpful.

A detailed history will explain an infectious process from an allergic one or nonallergic one. The three cardinal symptoms of infectious rhinitis contain purulent nasal discharge, nasal obstruction, and facial pressure, which are often present for ten days or longer. A fever may or may not be present.²⁷ A physical exam will usually disclose purulence in the region of the middle meatus with hyperemia, oedema, or crusting along the middle turbinate.

Management

Educate the patient on avoiding environmental triggers.²¹ After coming from outside, the patient should wash the face and nasal cavity. Limiting exposure to inciting factors such as perfumes, tobacco smoke, and cleaning supplies can significantly reduce symptoms.²⁸

Intranasal corticosteroids and/or intranasal antihistamines are the mainstay of therapy.

Nasal corticosteroids are considered for congestion and obstructive symptoms. They work on the nasal mucosa resulting in reduced neutrophil and eosinophil chemotaxis, declined mast cell and basophil mediator release, and ultimately diminished oedema and inflammation.²⁹ Topical steroids are overall well-tolerated, and side effects are infrequent. The most commonly reported side effects are nasal dryness, crusting, nasal bleeding and septal perforation.³⁰ Integrated analysis of three double-blinded randomized controlled trials displayed that fluticasone 200 or 400 mcg was particularly more effective than a placebo.³¹ Fluticasone propionate and beclomethasone are currently the only topical steroid preparations approved by the FDA for vasomotor rhinitis.^{28, 31} Budesonide has also shown to be efficacious and is at present the only topical steroid agent with a pregnancy category B rating.^{32, 33}

Azelastine is helpful in patients with rhinorrhea and nasal congestion. Azelastine is an H1-receptor antagonist. It also suppresses the synthesis of leukotriens, kinins, cytokines, and adhesion molecule expression, while providing anti-inflammatory effects irrelevant to histamine.^{34, 35} This characteristic makes it effective in VMR. Azelastine provides a notable reduction in vasomotor symptomatology, including nasal obstruction, rhinorrhea,

and nasal oedema, as shown in two randomized control trials. These trials demonstrated significant improvement in all symptoms within the first week of treatment.^{36, 37}

Topical capsaicin has also demonstrated efficacy as an adjunct therapy for those with rhinorrhea and nasal congestion.³⁸ The mechanism is thought to center around the modulation of the C fibers associated with nociceptive neurons. Capsaicin targets transient receptor potential vanilloid type 1 (TRPV1), an ion channel that presents on epithelial cells, submucosal glands, and nerves in the human nasal mucosa and assists in regulating nasal secretions and congestion. Repeated intranasal applications of capsaicin can desensitize TRPV1 making it less sensitive to physical and chemical nociceptive stimuli. However, the limiting factor with capsaicin use is a patient's ability to tolerate its irritant quality.³⁹

A topical anticholinergic like ipratropium bromide is the first choice for rhinorrhea. Two separate randomized controlled trials exhibited the effectiveness of ipratropium bromide in controlling rhinorrhea.^{40, 41} Topical anticholinergics act locally and block parasympathetic input to the nasal mucosa glands only.⁴⁰ Epistaxis and nasal dryness may occur.⁴¹ Ipratropium bromide also carries a pregnancy category B rating and can be used in children as young as six.

Botulinum toxin (BTX) inhibits acetylcholine release from the presynaptic nerve terminal. Injecting BTX into the inferior and middle turbinates subsides rhinorrhea and nasal mucous gland secretion in patients with vasomotor rhinitis. The improvement is usually short-lived, lasting roughly four weeks.⁴²

When medical management alone does not adequately control vasomotor rhinitis symptoms, surgical interventions may be employed.

The therapeutic transection of the vidian nerve is a well-known surgical option for vasomotor rhinitis. The technique aims to disturb the autonomic nerve supply of the nasal cavity, thus decreasing nasal secretions.²⁸ The vidian nerve, a branch of facial nerve, carries preganglionic parasympathetic fibers to the pterygopalatine ganglion. These nerve fibers function in the regulation of blood flow to the nasal mucosa. The vidian nerve forms from the confluence of the greater superficial petrosal and deep petrosal nerves. The deep petrosal nerve contains sympathetic fibers. The greater superficial petrosal nerve contains preganglionic parasympathetic secretomotor fibers for the lacrimal, palatine, and nasal mucus glands, as well as the nerves responsible for vasodilating the vessels of the nasal mucosa. A systematic review outlined

that all published case series associated with vidian neurectomy improved rhinorrhea and nasal congestion.⁴³ The most notable complication associated with neurectomy includes postoperative dry eyes from decreased lacrimation with a reported aggregate rate of 48%, dysesthesia, and mucosal engorgement when supine.⁴⁴

Investigations

- Absolute eosinophil count
- Nasal smear cytology
- Skin allergy tests
- Acoustic rhinometry for measuring nasal patency
- Smell testing
- CT scan in cases of sinus disease⁴⁵

Prognosis

It is not completely curable but may be controlled.

Complications:

Hypertrophied inferior turbinate, asthma, nasal polyp.

Conclusion:

Under the control of the hypothalamus is the autonomic nervous system, which supplies the nasal mucosa. In cases of NAR, the autonomic nervous system is not stable. Nasal mucosa becomes hyper-reactive and responds disproportionately to various nonspecific heterogenous stimuli, such as change in temperature, humidity, blasts of air, small amounts of dust or smoke. In some cases, pathophysiologic mechanisms of VMR are not adequately understood. Further investigation may result in well-targeted therapies, which, in turn, will offer superior symptom relief, control associated comorbidities, and decrease economic burden related to this highly prevalent condition.

VMR detrimentally affects the quality of life in a significant portion of the population. The co-morbidities analogous with VMR can have a further negative influence on patients' wellbeing. Treatment of VMR requires that underlying triggers be discerned and if at all possibly modified. A stepwise approach using pharmacologic therapies can then be implemented, typically with a satisfactory outcome for patients and physicians alike.

Conflicts of interest:

No potential conflict of interest relevant to this article has been reported.

References:

1. Tran N, Vickery J, Blaiss M. Management of rhinitis: Allergic and non-allergic. *Allergy Asthma Immunology Research*. 2011 Jul;3(3):148
2. Small P, Kim H. Allergic rhinitis. *Allergy, Asthma & Clinical Immunology*. 2011;7(1):1-2.
3. Settipane R, Kaliner M. Nonallergic Rhinitis. *American Journal of Rhinology & Allergy*. 2013 May 1; 27(3):48-51.
4. Papadopoulos, N.G.; Bernstein, J.A.; Demoly, P.; Dykewicz, M.; Fokkens, W.; Hellings, P.W.; et al. Phenotypes and endotypes of rhinitis and their impact on management: A PRACTALL report. *Allergy*. 2015 May; 70(5): 474-494.
5. Papadopoulos, N.G.; Guibus, G.V. Rhinitis Subtypes, Endotypes and Definitions. *Immunology and Allergy Clinics of North America*. 2016 May; 36(2): 215-233.
6. Russel, A. Settipane Epidemiology of Vasomotor Rhinitis. *World Allergy Organization Journal*. 2009 June; 2(6): 115-118.
7. Settipane RA, Lieberman P: Update on non-allergic rhinitis. *Annals of Allergy, Asthma & Immunology*. 2001 May; 86(5): 494-507.
8. Jaradeh SS, Smith TL, Torrico L, Prieto TE, Loehrl TA, Darling RJ, et al: Autonomic nervous system evaluation of patients with vasomotor rhinitis. *Laryngoscope*. 2000 Nov; 110(11): 1828-1831.
9. Loehrl TA, Smith TL, Darling RJ, Torrico L, Prieto TE, Shaker R, et al: Autonomic dysfunction, vasomotor rhinitis, and extraesophageal manifestations of gastroesophageal reflux. *Otolaryngology Head and Neck Surgery*. 2002 May; 126(4): 382-387.
10. Tai CF, Baraniuk JN: Upper airway neurogenic mechanisms. *Current Opinion in Allergy and Clinical Immunology*. 2002 Feb 1; 2(1):11-19.
11. Schierhorn K, Hanf G, Fischer A, Umland B, Olze H, Kunkel G: Ozone-induced release of neuropeptides from human nasal mucosal cells. *International Archives of Allergy and Immunology*. 2002 Oct; 129(2): 145-151.
12. Groneberg DA, Heppt W, Cryer A, Wussow A, Peiser C, Zweng M, et al: Toxic rhinitis-induced changes of human nasal mucosa innervation. *Toxicologic Pathology*. 2003 May; 31(3): 326-331.
13. Tosun F, Sezen I, Gerek M, Ozkaptan Y, Yapar M, Caliskaner Z, et al: Electrophoretic evaluation of nasal discharge in patients with allergic rhinitis and vasomotor rhinitis. *American Journal of Rhinology*. 2002 May; 16(3): 141-144.
14. Salib RJ, Harries PG, Nair SB, Howarth PH. Mechanisms and mediators of nasal symptoms in non-allergic rhinitis. *Clinical and Experimental Allergy*. 2008 Mar; 38(3): 393-404.
15. Giannesi F, Fattori B, Ursino F, Giambelluca MA, Soldani P, Scavuzzo MC, et al: Ultrastructural and ultracytochemical study of human nasal respiratory epithelium in vasomotor rhinitis. *Acta Oto-Laryngologica*. 2003 Oct; 123(8):943-949
16. Ruffoli R, Fattori B, Giambelluca MA, Soldani P, Giannesi F: Ultracytochemical localization of the NADPH-d activity in the human nasal respiratory mucosa in vasomotor rhinitis. *The Laryngoscope*. 2000 Aug; 110(8):1361-1365.

17. Mullarkey MF, Hill JS, Webb DR: Allergic and nonallergic rhinitis: their characterization with attention to the meaning of nasal eosinophilia. *Journal of Allergy and Clinical Immunology*. 1980; 65(2):122-126.
18. Enberg RN. Perennial nonallergic rhinitis: a retrospective review. *Annals of Allergy*. 1989 Dec; 63(6):513-516.
19. Settipane GA, Klein DE. Non allergic rhinitis: demography of eosinophils in nasal smear, blood total eosinophil counts and IgE levels. *Allergy and Asthma Proceedings*. 1985; 6(4):363-366.
20. Molgaard E, Thomsen SF, Lund T, Pedersen L, Nolte H, Backer V: Differences between allergic and nonallergic rhinitis in a large sample of adolescents and adults. *European Journal of Allergy and Clinical Immunology*. 2007 Sep; 62(9):1033-1037.
21. Pattanaik D, Liebermann P. Vasomotor rhinitis. *Current Allergy and Asthma Reports*. 2010 Mar; 10(2):84-91.
22. Kaliner MA. Classification of Nonallergic Rhinitis Syndromes With a Focus on Vasomotor Rhinitis, Proposed to be Known hence forth as Nonallergic Rhinopathy. *World Allergy Organization Journal*. 2009 Jun 15; 2(6):98-101.
23. Meggs WJ, Cleveland CH. Rhinology examination of patients with the multiple chemical sensitivity syndrome. *Archives of Environmental Health*. 1993 Jan-Feb; 48(1): 14-8.
24. Greiner AN, Meltzer EO. Pharmacologic rationale for treating allergic and nonallergic rhinitis. *Journal of Allergy and Clinical Immunology*. 2006 Nov; 118(5):985-98.
25. Brandt D, Bernstein JA. Questionnaire evaluation and risk factor identification for nonallergic vasomotor rhinitis. *Annals of Allergy, Asthma & Immunology*. 2006 Apr; 96(4):526-32.
26. Gosepath J, Amedee RG, Mann WJ. Nasal provocation testing as an international standard for evaluation of allergic and nonallergic rhinitis. *The Laryngoscope*. 2005 Mar; 115(3):512-6.
27. Rosenfeld RM. CLINICAL PRACTICE. Acute Sinusitis in Adults. *The New England Journal of Medicine*. 2016 Sep 08; 375(10):962-70.
28. Yan CH, Hwang PH. Surgical Management of Nonallergic Rhinitis. *Otolaryngologic Clinics of North America*. 2018 Oct; 51(5):945-955.
29. Pipkorn U, Proud D, Lichtenstein LM, Kagey-Sobotka A, Norman PS, Naclerio RM. Inhibition of mediator release in allergic rhinitis by pretreatment with topical glucocorticosteroids. *The New England Journal of Medicine*. 1987 Jun 11; 316(24):1506-10.
30. Cervin A, Andersson M. Intranasal steroids and septum perforation - an overlooked complication? A description of the course of events and a discussion of the causes. *Rhinology*. 1998 Sep; 36(3):128-32.
31. Webb DR, Meltzer EO, Finn AF, Rickard KA, Pepsin PJ, Westlund R, et al. Intranasal fluticasone propionate is effective for perennial nonallergic rhinitis with or without eosinophilia. *Annals of Allergy, Asthma & Immunology*. 2002 Apr; 88(4):385-90.
32. Creticos P, Fireman P, Settipane G, Bernstein D, Casale T, Schwartz H. Intranasal budesonide aqueous pump spray (Rhinocort Aqua) for the treatment of seasonal allergic rhinitis. *Rhinocort Aqua Study Group. Allergy and Asthma Proceedings*. 1998 Sep-Oct; 19(5): 285-94.
33. Wight RG, Jones AS, Beckingham E, Andersson B, Ek L. A double blind comparison of intranasal budesonide 400 micrograms and 800 micrograms in perennial rhinitis. *Clinical Otolaryngology & Allied Sciences*. 1992 Aug; 17(4):354-8.
34. Lieberman PL, Settipane RA. Azelastine nasal spray: a review of pharmacology and clinical efficacy in allergic and nonallergic rhinitis. *Allergy and Asthma Proceedings*. 2003 Mar-Apr; 24(2):95-105.
35. Lieberman P. Intranasal antihistamines for allergic rhinitis: mechanism of action. *Allergy and Asthma Proceedings*. 2009 Jul-Aug; 30(4):345-8.
36. Banov CH, Lieberman P, Vasomotor Rhinitis Study Groups. Efficacy of azelastine nasal spray in the treatment of vasomotor (perennial nonallergic) rhinitis. *Annals of Allergy, Asthma & Immunology*. 2001 Jan; 86(1):28-35.
37. Lieberman P, Kaliner MA, Wheeler WJ. Open-label evaluation of azelastine nasal spray in patients with seasonal allergic rhinitis and nonallergic vasomotor rhinitis. *Current Medical Research and Opinion*. 2005 Apr; 21(4):611-8.
38. Fokkens W, Hellings P, Segboer C. Capsaicin for Rhinitis. *Current Allergy and Asthma Reports*. 2016 Aug; 16(8):60.
39. Gevorgyan A, Segboer C, Gorissen R, van Drunen CM, Fokkens W. Capsaicin for non-allergic rhinitis. *Cochrane Database of Systematic Reviews*. 2015 Jul 14; 2015(7):CD010591.
40. Grossman J, Banov C, Boggs P, Bronsky EA, Dockhorn RJ, Druce H, et al. Use of ipratropium bromide nasal spray in chronic treatment of nonallergic perennial rhinitis, alone and in combination with other perennial rhinitis medications. *The Journal of Allergy and Clinical Immunology*. 1995 May; 95(5):1123-7.
41. Bronsky EA, Druce H, Findlay SR, Hampel FC, Kaiser H, Ratner P, et al. A clinical trial of ipratropium bromide nasal spray in patients with perennial nonallergic rhinitis. *The Journal of Allergy and Clinical Immunology*. 1995 May; 95(5):1117-22.
42. Braun T, Gurkov R, Kramer MF, Krause E. Septal injection of botulinum neurotoxin A for idiopathic rhinitis: a pilot study. *American Journal of Otolaryngology*. 2012 Jan-Feb; 33(1):64-7.
43. Marshak T, Yun WK, Hazout C, Sacks R, Harvey RJ. A systematic review of the evidence base for vidian neurectomy in managing rhinitis. *The Journal of Laryngology & Otolaryngology*. 2016 Jul; 130(4):7-28.
44. Halderman A, Sindwani R. Surgical management of vasomotor rhinitis: a systematic review. *American Journal of Rhinology & Allergy*. 2015 Mar-Apr; 29(2):128-34.
45. Bansal M. *Diseases of Ear, Nose & Throat with Head & Neck Surgery*, 2nd edition: pages 358-360.

Branchial Cleft Cyst with Papillary Carcinoma of Ectopic Thyroid: A Case Report

AWAL N¹, RAHMAN MS², AFROZ F³, RAHMAN MR⁴, ALAM MS⁵

Abstract

The most frequent lateral neck lesions are branchial cleft cysts, which typically develop in the second to third decades of life. The ectopic thyroid tissue within a branchial cleft cyst is an unusual and papillary thyroid carcinoma arising from this tissue is a very uncommon occurrence. In this report, we present a case of a 39-year-old woman with papillary carcinoma originating from the right lateral branchial cleft cyst without any evidence of papillary carcinoma in the thyroid gland. The carcinoma was discovered incidentally by histopathological examination followed by surgical excision.

Keywords: Branchial Cleft Cyst, Papillary Carcinoma, Ectopic Thyroid

Journal of Green Life Med. Col. 2022; 7(2): 23-26

Introduction:

Branchial cleft cysts are a congenital and benign mass that can arise from the failure of obliteration of the 2nd branchial cleft during embryogenesis. They may not be symptomatic until late childhood or early adulthood, although an infected branchial cleft cyst can cause symptoms during an upper respiratory tract illness. This is because the presence of lymphoid tissue beneath the epithelium of the cyst is assumed to be the outcome of chronic inflammation. However, ectopic thyroid tissue within this cyst is a rare abnormality.

Any thyroid tissue that is not located in its normal position is called ectopic thyroid tissue. Ectopic thyroid tissue can be seen anywhere from foramen cecum to the lower neck region, but rarely seen in branchial cleft cyst. Like normal thyroid tissue, ectopic thyroid tissue can go through

pathogenic processes. They can harbor primary papillary thyroid carcinoma.

Papillary thyroid cancer of branchial cleft cyst is typically diagnosed incidentally and may clinically be difficult to distinguish from benign branchial cleft cyst. It is discovered during surgical resection followed by histopathological examination. We present a rare case of ectopic papillary thyroid carcinoma that has developed from a branchial cleft.

Case report:

This is a case report of a 39-year-old woman who presented with who had a painless swelling on the right side of her neck. Painless right-sided neck swelling for the last 3 months. The swelling gradually increased in size with no obstructive symptoms. No history of previous head neck radiation exposure was reported. On physical examination, a 6x4 cm palpable, soft, non-tender, movable mass was seen on the right anterior side of the neck. The rest of the physical examination including palpation of thyroid gland was unremarkable. The neck ultrasonography revealed a solitary nodule measuring 0.5 cm in the right lower pole of thyroid gland and a big cystic mass of 5 cm in the right anterior aspect of the neck containing a solid component measuring about 18 x 10 cm with calcified changes.

Fine needle aspiration cytology from the right side of the neck yielded 15 cc thick straw-colored fluid. Aspirate material contains amorphous eosinophilic substances with

1. Naila Awal, Assistant Professor, Department of Pathology, Green Life Medical College, Dhaka. nailaawal@gmail.com
2. Md Saidur Rahman, Professor and Head, Department of Pathology, Sher-e-Bangla Medical College, Barishal.
3. Farhana Afroz, Associate Professor, Department of Pathology, Green Life Medical College, Dhaka
4. Md. Rifayet Rahman, Associate Professor, Department of Pharmacology, Green Life Medical College, Dhaka
5. Md. Shafiqul Alam, Associate Professor, Radiology and Imaging Department, Monno Medical College, Manikgonj.

Address of Correspondence: Naila Awal, Assistant Professor, Department of Pathology, Green Life Medical College, Dhaka, email: nailaawal@gmail.com

Received: 10.10.2024

Accepted: 06.07.2024

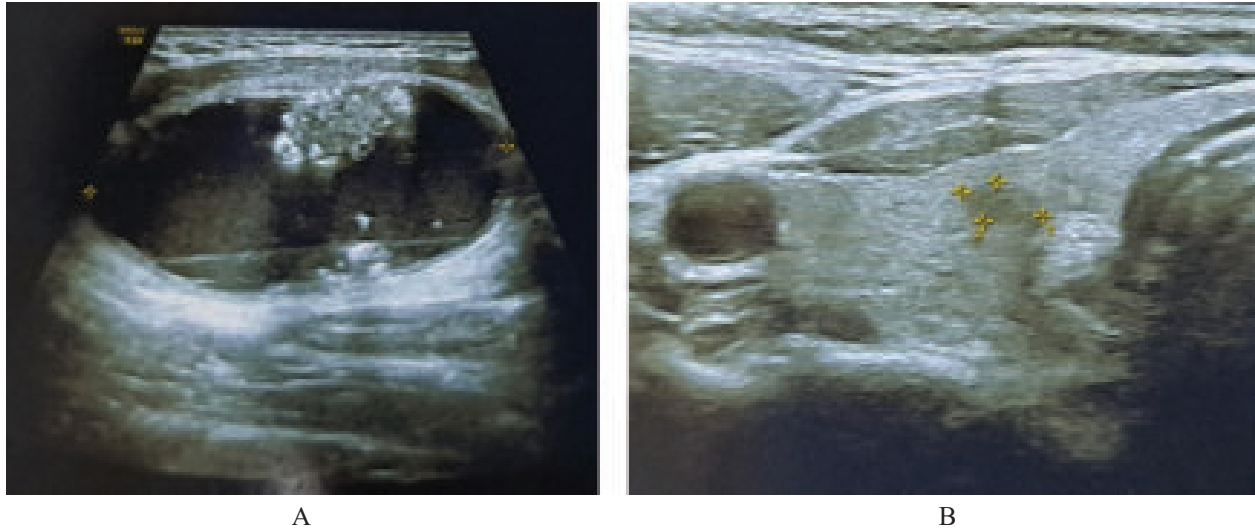


Figure 1. A. Neck ultrasound evaluation showed a) large cystic lesion (51x34mm) containing a solid component measuring about 18x10 mm at the right side of the neck, completely separated from thyroid gland b) small nodule (5.0x3.7mm) at right lobe of thyroid

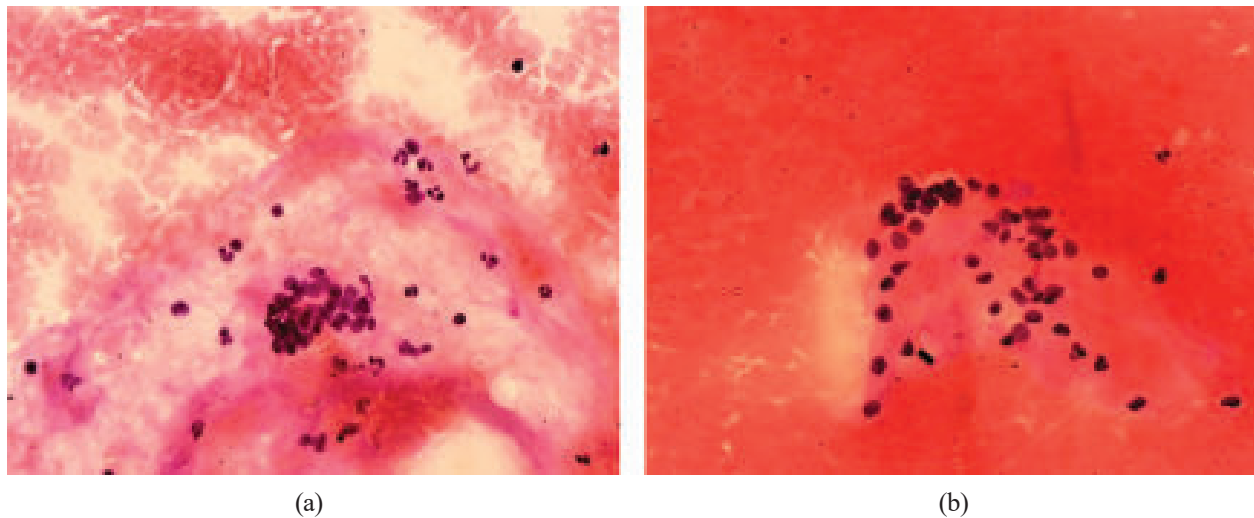


Figure 2. (a) and (b): US guided Fine needle aspiration shows follicular epithelial cells arranged in small clusters and singly in a background of colloid. (hematoxylin-eosin [H&E] staining, original magnification $\times 20$)

a small number of lymphocytes mixed with many foamy macrophages indicating a branchial cleft cyst. The USG-guided FNAC performed from the right lobe of the thyroid suggested a nodular goiter.

The surgical excision of the cyst was performed, and grossly, there is a large collapse cystic structure measuring 4.5X3.5 cm. The inner surface shows a grey, brown granular tumorous lesion measuring 2x1.5 cm and the outer surface of the cyst is smooth.

Microscopically, the cyst wall is partially lined by flattened epithelium with a stroma containing lymphoid follicles, but other areas show typical features of papillary thyroid carcinoma within cyst wall and broad-based papillary structure lined by columnar epithelium. The nuclear characteristics of papillary thyroid carcinoma were seen in the resected sample. The normal thyroid tissue is discovered within the cyst wall adjacent to the tumor on histopathological examination. Areas of calcification are also seen.

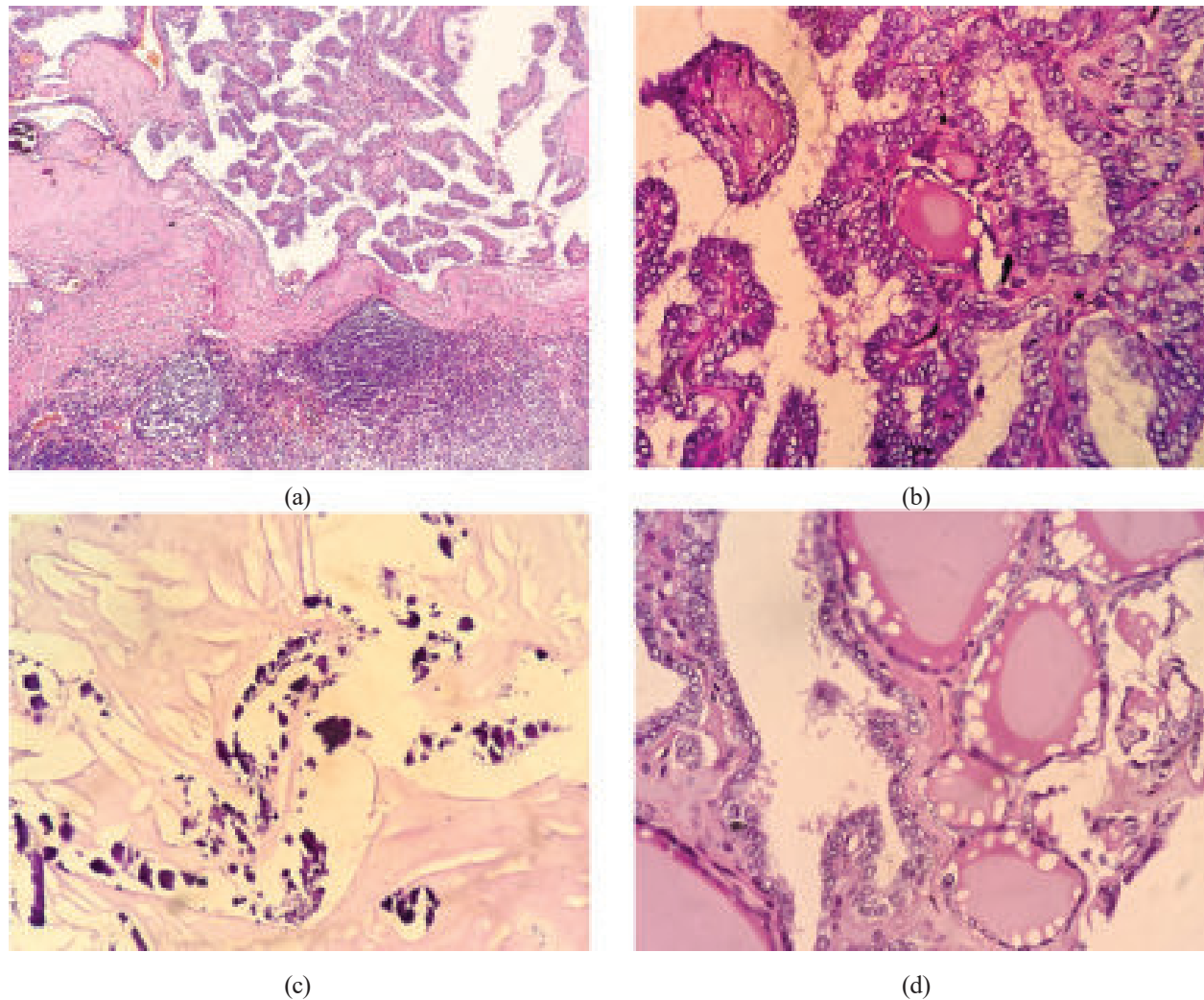


Fig.3a. Microscopic slide (hematoxylin-eosin [H&E] staining, original magnification $\times 10$) showing thick wall of branchial cyst with papillary thyroid carcinoma 3b. High magnification of papillae (hematoxylin-eosin [H&E] staining, original magnification $\times 20$) showing single or stratified cuboidal cells with clear, ground glass, overlapping nuclei and occasional nuclei with nuclear groove lining the papillae 3c. Microscopic slide (hematoxylin-eosin [H&E] staining, original magnification $\times 20$) showing psammoma bodies 3d. Microscopic slide (hematoxylin-eosin [H&E] staining, original magnification $\times 40$) showing normal thyroid tissue within the cyst wall adjacent to the tumor.

Discussion:

Ectopic thyroid may be defined as functional thyroid tissue outside the normal anatomical location. Autopsy studies suggest that 7 - 10% of adults may have thyroid tissue remnants along the path of thyroid descent¹ or along its embryonic course. Ectopic thyroid tissue is frequently seen within thyroglossal duct cysts. The anterior tongue, larynx, trachea, esophagus, superior mediastinum, pericardium, diaphragm, salivary glands, retropharyngeal space, cervical lymph nodes and neck branchial cyst are the other possible sites of ectopic thyroid.²

The papillary thyroid carcinoma arising from branchial cleft cyst is extremely rare, a total of only 14 cases have been reported in the literature.

The development of branchial cleft cysts has been explained by two major theories: the congenital theory and the acquired theory. The congenital theory suggests that branchial cysts develop as a result of incomplete obliteration of the second, third, and fourth pharyngeal pouches, whereas the acquired theory suggests that they develop as a result of cystic transformation of cervical lymph nodes. The existence of ectopic thyroid tissue within

a branchial cleft cyst can be explained by either theory. The thyroid gland develops from one median anlage and two lateral anlages. Some thyroid cells may become trapped in a lateral branchial cleft cyst and possibly this can lead to thyroid carcinoma in branchial cleft cysts.⁴

Preoperative diagnosis of BCC containing papillary thyroid cancer is challenging. All published cases in literature were detected incidentally after surgical resection of a cystic mass in the lateral neck. Sidhu's criteria for papillary thyroid carcinoma in branchial cleft cysts suggest that if normal thyroid tissue is present alongside papillary thyroid carcinoma within the wall of the branchial cleft cyst and there is no evidence of papillary carcinoma in the thyroid, it points to primary papillary thyroid carcinoma rather than secondary metastases.⁵ In our case, there was normal thyroid tissue adjacent to the papillary carcinoma within the cyst wall and ultrasound guided fine-needle aspiration cytology from solid part of right thyroid revealed nodular goiter, which suggested that papillary thyroid carcinoma arise from ectopic thyroid tissue from branchial cleft cyst.

Conclusion:

In conclusion, the causes of lateral cystic masses in the neck can be complex. Branchial cleft cyst and ectopic thyroid tissue can both be differential diagnoses of a lateral neck mass in middle-aged population. However, primary ectopic thyroid carcinoma on the other hand, is a rare finding. There are no clinical, biochemical, or imaging parameters that can help to determine the nature of the lesion; the nature of the lesion, only histopathological examination can determine the definitive diagnosis.

Therefore, early, and thorough diagnosis is essential for proper and the least burdensome treatment for the patient.

The likelihood of primary papillary cancer in the branchial cleft cyst must be taken into consideration by surgeons.

References:

1. Adelchi C, Mara P, Melissa L, De Stefano A, Cesare M. Ectopic thyroid tissue in the head and neck: a case series. *BMC Res Notes* [Internet]. 2014 [cited 2023 Aug 31];7(1). Available from: <http://dx.doi.org/10.1186/1756-0500-7-790>
2. Fumarola A, Trimboli P, Cavaliere R, Coletta I, Veltri A, Di Fiore A, et al. Thyroid papillary carcinoma arising in ectopic thyroid tissue within a neck branchial cyst. *World J Surg Oncol* [Internet]. 2006 [cited 2023 Aug 31];4(1):24. Available from: <http://dx.doi.org/10.1186/1477-7819-4-242>
3. Tsung SH. Papillary thyroid carcinoma occurring in branchial cleft cyst: Case report and review of literature. *European Journal of Medical and Health Sciences* [Internet]. 2021 [cited 2023 Aug 31];3(2):6–8. Available from: <https://www.ejmed.org/index.php/ejmed/article/view/734>
4. Sagit M, Gokler A, Akin I, Han U. Wrong egg in the usual nest: Thyroid papillary carcinoma within a branchial cleft cyst. *Ear Nose Throat J* [Internet]. 2013;92(7):E31–4. Available from: <http://dx.doi.org/10.1177/014556131309200719>
5. Sidhu S, Lioe TF, Clements B. Thyroid papillary carcinoma in lateral neck cyst: missed primary tumour or ectopic thyroid carcinoma within a branchial cyst? *J Laryngol Otol* [Internet]. 2000 [cited 2023 Aug 31];114(09). Available from: <https://pubmed.ncbi.nlm.nih.gov/11091840/>
6. Gur H, Bozdogan Arpacı R, Ismi O, Dag A, Vayisoglu Y, et al. Papillary thyroid carcinoma spreading into branchial cleft cyst. *Turk Arch Otorhinolaryngol* [Internet]. 2019 [cited 2023 Aug 31];57(2):95–8. Available from: <http://dx.doi.org/10.5152/tao.2019.4151>
7. Karras S, Anagnostis P, Noussios G, Pontikides N. Thyroid papillary carcinoma arising in ectopic thyroid tissue within a branchial cleft cyst. *BMJ Case Rep* [Internet]. 2013 [cited 2023 Aug 31];2013(apr22 1):bcr2013009312–bcr2013009312. Available from: <https://pubmed.ncbi.nlm.nih.gov/23608867/>

Continuing Medical Education (CME)*Journal of Green Life Med. Col. 2023; 8(1): 27*

27.07.2022	Adolescent Health: A Challenging and Burning Issue, Nowadays	Department of Community Medicine
24.08.2022	Roadmap for Implementing Antimicrobial Stewardship	Department of Microbiology
31.08.2022	Management of Hyperglycemia in Hospitalized Patients	Department of Endocrinology and Metabolism
07.09.2022	Anatomy of the Liver & Beyond	Department of Anatomy
14.09.2022		Department of ENT
28.09.2022	Stormy Journey of a Pregnant Woman	Department of Obstetrics and Gynecology
12.10.2022	Hanging- A Method of Suicide Toxicology	Department of Forensic Medicine &
19.10.2022	A 50yr Old Lady with a Breast Lump	Department of Surgery
26.10.2022		Department of Pathology
02.11.2022	Updated Management of Asthma	Department of Medicine
09.11.2022	Hemostasis	Department of Physiology
16.11.2022		Department of Dentistry
23.11.2022	Stress Management for Medical Students	Department of Community Medicine
30.11.2022	Antimicrobials Resistance Containment in Bangladesh- Opportunity & Challenges	Department of Microbiology
07.12.2022	Refractive Error & It's Management	Department of Ophthalmology
14.12.2022	Insomnia: Pathway of Managements	Department of Pharmacology
21.12.2022	A 27 Months old Child with Cough, Fever, Diarrhea	Department of Pediatrics
28.12.2022	Medication Errors in Anaesthesia	Department of Anaesthesia

link: <https://greenlife.edu.bd/campus-life/cme-2022/>

GREEN LIFE MEDICAL COLLEGE JOURNAL

Reviewers Panel

1. **Prof. Dr. A.B.M Bayezid Hossain**
Professor & Head
Department of Surgery
Green Life Medical College
2. **Professor Dr. Aftab Uddin Ahmed**
Professor & Head
Department of E.N.T
Green Life Medical College
3. **Prof. Dr. Ehsamul Azim**
Professor & Head
Department of Community Medicine
Green Life Medical College
4. **Prof. Dr. Fahmida Kabir**
Professor & Head
Department of Biochemistry
Green Life Medical College
5. **Dr. Helal Uddin Ahmed**
Associate Professor
National Institute of Mental Health, Dhaka
6. **Prof. Dr. Homayera Rahman**
Professor & Head
Department of Physiology
Green Life Medical College
7. **Prof. Dr Humaira Naushaba**
Professor & Head
Department of Anatomy
Green Life Medical College
8. **Prof. Joya Sree Roy**
Professor
Department of Gynecology and Obstetrics
Green Life Medical College
9. **Prof. M.A. Azhar**
Professor
Department of Medicine
Green Life Medical College
10. **Prof. Dr. Md Manjur Alam**
Professor
Department of Surgery
Green Life Medical College
11. **Dr. Md. Rifayet Rahman**
Associate Professor & Head
Department of Pharmacology
Green Life Medical College
12. **Prof. Dr. Md. Zahidur Rahman**
Professor & Head
Department of Orthopedic Surgery
Green Life Medical College
13. **Prof. Dr. Md. Rezaul Karim Dewan**
Professor & Head
Department of Pathology
Green Life Medical College
14. **Prof. Dr. Mohammad Asifuzzaman**
Professor & Head
Department of Dermatology and Venereology
Green Life Medical College
15. **Prof. Dr. Quazi Rakibul Islam**
Professor & Head
Department of Paediatrics
Green Life Medical College
16. **Prof. Dr. Rabeya Begum**
Professor & Head
Department of Anesthesiology
Green Life Medical College
17. **Dr. Salma Parvin**
Associate Professor & Head
Department of Ophthalmology
Green Life Medical College
18. **Prof. Dr. Salma Rouf**
Professor & Head
Department of Obstetrics & Gynaecology
Green Life Medical College
19. **Dr. Sanjida Akhter**
Associate Professor & Head
Department of Forensic Medicine
Green Life Medical College
20. **Prof. Dr. Sanya Tahmina Jhora**
Professor & Head
Department of Microbiology
Green Life Medical College
21. **Prof. Dr. SK. Abdul Fattah**
Professor & Head
Department of Medicine
Green Life Medical College
22. **Prof. Soofia Khatoon**
Professor and Head
Department of Pediatrics
Centre for Women & Child Health, Dhaka
23. **Prof. Syed Atiqul Haq**
Professor
Department of Rheumatology
Green Life Medical College
23. **Dr. Sudhakar Sarker**
Assistant Professor
Department of Cardiology
Green Life Medical College

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