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CONTENTS

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INSTRUCTION TO AUTHORS

A. Introduction

Bangladesh Heart Journal is the official journal of Bangladesh Cardiac Society, and accepts articles for publication from home and abroad. This is a biannual, peer-reviewed journal and aims to publish work of the highest quality from all sub-specialties of cardiology and cardiovascular surgery. The aim of the publication is to promote research in Bangladesh and serve as platform for dissemination of scientific information in cardiology.

B. Categories of Articles

The journal accepts original research, review articles, case reports, cardiovascular images and letters to the editor, for publication.

Original Research:

Original, in-depth research article that represents new and significant contributions to medical science. Each manuscript should be accompanied by a structured abstract of up to 250 words using the following headings: Objective, Methods, Results, and Conclusions. Three to 5 keywords to facilitate indexing should be provided in alphabetical order below the abstract. The text should be arranged in sections on INTRODUCTION, METHODS, RESULTS and DISCUSSION. The typical text length for such contributions is up to 3000 words (including title page, abstract, tables, figures, acknowledgments and key messages). Numberof references should be limited to 50.

Review Articles:

Generally review articles are by invitation only. But unsolicited reviews will be considered for publication on merit basis. Following types of articles can be submitted under this category: Newer drugs, new technologies and review of a current concept.The manuscript should not exceed 5000 words (including tables and figures). A review article should include an abstract of up to 250 words describing the need and purpose of review,methods used for locating, selecting, extracting and synthesizing data, and main conclusions. The number of references should be limited to 50.

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Only case reports of exceptional quality will be published in the case report format. The text should not exceed 1500 words and is arranged as introduction, case report and discussion. Include a brief abstract of about 150 words. Number of tables/figures should be limited to 3. Include up to 10 most recent references. The patient's written consent, or that of the legal guardian, to publication must be obtained.

Cardiovascular Images:

Only clinical photographs with or without accompanying skiagrams, pathological images, echocardiographic images, angiographic images etc. are considered for publication. Image should clearly identify the condition and have the classical characteristics of the clinical condition. Clinical photographs of condition which are very common, where diagnosis is obvious, or where diagnosis is not at all possible on images alone would not be considered. Photographs should be of high quality, usually 127 × 173 mm (5 × 7 in) but no larger than 203 × 254 mm (8 × 10 in). A short text of up to 250 words depicting the condition is needed. Figures should be placed exactly at a logical place in the manuscript. The submitted images should be of high resolution (>300 dpi). The following file types are acceptable: JEPG and TIFF. The number of authors should not exceed 3. The authors should ensure that images of similar nature have not been published earlier. Authors must obtain signed informed consent from the patient, or the legal guardian.

Letter to the Editor:

Letters commenting upon recent articles in Bangladesh Heart Journal are welcome.Such letters should be received within 16 weeks of the article's publication. Letters should be up to 250 words; should contain no more than 1 figure/table and upto 5 most recent references. The text need not be divided into sections. The number of authors should not exceed 3.

C. Criteria for Acceptance

All manuscripts should meet the following criteria: the material is original, study methods areappropriate, data are sound, conclusions are reasonable and supported by the data, and the information is important; the topic has general cardiology interest; and that the article is written in reasonably good English. Manuscripts which do not follow the guidelines of Bangladesh Heart Journal are likely to be sent back to authors without initiating the peer-review process. All accepted manuscripts are subject to editorial modifications to suit the language and style of Bangladesh Heart Journal and suggestions may be made to the authors by the Editorial Board to improve the scientific value of the journal.

D. Editorial Process

The Bangladesh Heart Journal commits to high ethical and scientific standards. Submitted manuscripts are considered with the understanding that they have not been published previously in print or electronic format (except

in abstract or poster form) and are not under consideration by another publication or electronic medium. Statements and opinions expressed in the articles published in the Journal are those of the authors and not necessarily of the Editor. Neither the Editor nor the Publisher guarantees, warrants, or endorses any product or service advertised in the Journal. Bangladesh Heart Journal follows the guidelines on editorial independence produced by the International Committee of Medical Journal Editors (ICMJE). All manuscripts correctly submitted to the Bangladesh Heart Journal are first reviewed by the Editors. Manuscripts are evaluated according to their scientific merit, originality, validity of the material presented and readability. Some manuscripts are returned back to the authors at this stage if the paper is deemed inappropriate for publication in the Bangladesh Heart Journal, if the paper does not meet the submission requirements, or if the paper is not deemed to have a sufficiently high priority. All papers considered suitable by the Editors for progress further in the review process, undergo peer review by at least two reviewers. If there is any gross discrepancy between the comments of two reviewers, it is sent to a third reviewer. Peer reviewers' identities are kept confidential; authors' identities are also not disclosed to the reviewers. Accepted articles are edited, without altering the meaning, to improve clarity and understanding. Decision about provisional or final acceptance is communicated within 8 weeks.

E. Cover Letter

The cover letter should outline the importance and uniqueness of the work. It should include the signed declaration from all authors on:

1. Category of manuscript (original research, review article, case report, cardiovascular image, letter to the Editor)
2. Statement that the material has not been previously published or submitted elsewhere for publication (this restriction does not apply to abstracts published in connection with scientific meetings.)
3. Transfer of copyright to the Bangladesh Heart Journal upon the acceptance of the manuscript for publication
4. All authors have reviewed the article and agree with its contents
5. Information of any conflicts of interest (of any) of the authors.
6. Sources of research support, if any, including funding, equipment, and drugs.

The cover letter should also include the mailing address, telephone and fax numbers, and e-mail address of the corresponding author.

F. Manuscript Preparation

The manuscripts should comply with the prescribed guidelines. It should be well organized and written in simple and correct English under appropriate headings. The abbreviations and acronyms should be spelled out when they occur first time.

The Introduction should address the subject of the paper. The Methods section should describe in adequate detail the laboratory or study methods followed and state the statistical procedures employed in the research. This section should also identify the ethical guidelines followed by the investigators with regard to the population, patient samples or animal specimens used. A statement should be made, where applicable, that their study conforms to widely accepted ethical principles guiding human research (such as the Declaration of Helsinki) AND also that their study has been approved by a local ethics committee.The Results section should be concise and include pertinent findings and necessary tables and figures.The Discussion should contain conclusions based on the major findings of the study, a review of the relevant literature, clinical application of the conclusions and future research implications. Following the Discussion, Acknowledgements of important contributors and funding agencies may be given.

a. Title page information

- Title. Concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations where possible.
- Author names and affiliations. Please clearly indicate the given name(s) and family name(s) of each author and check that all names are accurately spelled. Present the authors' affiliation addresses (where the actual work was done) below the names. Indicate all affiliations with a lower case superscript letter immediately after the author's name and in front of the appropriate address. Provide the e-mail address of each author.
- Corresponding author. Clearly indicate who will handle correspondence at all stages of refereeing and publication, also post-publication. Ensure that the e-mail address is given and that contact details are kept up to date by the corresponding author.

b. Abstract

A concise and factual abstract is required. The abstract should state briefly the purpose of the research, the principal results and major conclusions. An abstract is often presented separately from the article, so it must be able to stand alone. References should be avoided. Also, non-standard or uncommon abbreviations should be

avoided, but if essential they must be defined at their first mention in the abstract itself.

c. Keywords

Immediately after the abstract, provide a maximum of 5 keywords. Keywords should be the listed terms in the Medical Subject's Headings (MeSH) of the National Library of Medicine (NLM), available at https://www.nlm.nih.gov/mesh.

d. Abbreviations

Define abbreviations that are not standard in this field in a footnote to be placed on the first page of the article. Such abbreviations that are unavoidable in the abstract must be defined at their first mention there, as well as in the footnote. Ensure consistency of abbreviations throughout the article.

e. Acknowledgements

Collate acknowledgements in a separate section at the end of the article before the references. List here those individuals who provided help during the research (e.g., providing language help, writing assistance or proof reading the article, etc.).

f. Units

Follow internationally accepted rules and conventions: use the international system of units (SI). If other units are mentioned, please give their equivalent in SI. Generic rather than trade names of drugs should be used.

g. Figures and graphics

- For graphics, a digital picture of 300 dpi or higher resolution in JPEG or TIFF format should be submitted.
- Figures should be numbered consecutively according to the order in which they have been first cited in the text, if there is more than 1 figure. Each figure should be cited in the text.
- Each figure/illustration should be provided with a suitable legend that includes enough information to permit its interpretation without reference to the text.
- All photomicrographs should indicate the magnification of the prints.
- When symbols, arrows, numbers or letters are used to identify parts of the illustrations, each one should be explained clearly in the legend.

h. Tables

Tables should be placed next to the relevant text in the article.

- Number tables consecutively in accordance with their appearance in the text. Each table should be cited in the text in Arabic numerals.
- Titles should be brief and a short or abbreviated heading for each column should be given.
- Explanatory matter should be placed in footnotes and not in the heading.
- Abbreviations in each table should be explained in footnotes.
- The data presented in a table should not be repeated in the text or figure.

i. References

References should follow the standards summarized in the NLM's International Committee of Medical Journal Editors (ICMJE) Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals (ICMJE recommendations), available at: http://www.icmje.org/recommendations/. The titles of journals should be abbreviated according to the style used for MEDLINE (www.ncbi.nlm.nih.gov/nlmcatalog/journals). Journals that are not indexed should be written in full.

- References should be numbered consecutively in the order in which they are first mentioned in the text.
- References in text, tables and legends should be identified by superscript Arabic numerals at the end of the sentence outside any punctuation. If several different studies or papers are cited within one sentence, the number should be placed where it will accurately identify the correct study.
- The names of authors in the text should concur with the reference list.
- References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration.
- Abstracts as references may be used; "unpublished observations" and "personal communications" may not be used as references, although references to written, not oral, communications may be inserted (in parentheses) in the text.
- Papers accepted but not yet published may be included as references by adding "In press" after the journal name. Information from manuscripts submitted but not yet accepted should be cited in the text as "unpublished observations" (in parentheses).
- In general: All authors/editors should be listed unless the number exceeds six, when you should give six followed by "et al."

Examples of correct forms of references are given below:

Articles in Journals (see also *Journal article on the Internet*)

1. *Standard journal article*

List the first six authors followed by et al.

Halpern SD, Ubel PA, Caplan AL. Solid-organ transplantation in HIV-infected patients. N Engl J Med. 2002 Jul 25;347(4):284-7.

More than six authors:

Rose ME, Huerbin MB, Melick J, Marion DW, Palmer AM, Schiding JK, et al. Regulation of interstitial excitatory amino acid concentrations after cortical contusion injury. Brain Res. 2002;935(1-2):40-6.

2. *Organization as author*

Diabetes Prevention Program Research Group. Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance.Hypertension. 2002;40(5): 679-86.

3. *Both personal authors and organization as author* (List all as they appear in the byline.)

Vallancien G, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1,274 European men suffering from lower urinary tract symptoms. J Urol. 2003;169(6):2257-61.

4. *Volume with supplement*

Geraud G, Spierings EL, Keywood C. Tolerability and safety of frovatriptan with short- and long-term use for treatment of migraine and in comparison with sumatriptan. Headache. 2002;42Suppl 2:S93-9.

5. *Issue with supplement*

Glauser TA. Integrating clinical trial data into clinical practice.Neurology. 2002;58(12 Suppl 7):S6-12.

6. *Type of article indicated as needed*

Tor M, Turker H. International approaches to the prescription of long-term oxygen therapy [letter]. Eur Respir J. 2002;20(1):242.

Lofwall MR, Strain EC, Brooner RK, Kindbom KA, Bigelow GE. Characteristics of older methadone maintenance (MM) patients [abstract]. Drug Alcohol Depend. 2002;66Suppl 1:S105.

7. *Article published electronically ahead of the print version*

Yu WM, Hawley TS, Hawley RG, Qu CK. Immortalization of yolk sac-derived precursor cells. Blood. 2002 Nov 15;100(10):3828-31. Epub 2002 Jul 5.

Books and Other Monographs

1. *Personal author(s)*

Murray PR, Rosenthal KS, Kobayashi GS, Pfaller MA. Medical microbiology. 4th ed. St. Louis: Mosby; 2002.

2. *Editor(s), compiler(s) as author*

Gilstrap LC 3rd, Cunningham FG, VanDorsten JP, editors.Operative obstetrics. 2nd ed. New York: McGraw-Hill; 2002.

3. *Organization(s) as author*

Advanced Life Support Group. Acute medical emergencies: the practical approach. London: BMJ Books; 2001. 454 p.

4. *Chapter in a book*

Meltzer PS, Kallioniemi A, Trent JM. Chromosome alterations in human solid tumors. In: Vogelstein B, Kinzler KW, editors. The genetic basis of human cancer. New York: McGraw-Hill; 2002. p. 93-113.

5. *Conference proceedings*

Harnden P, Joffe JK, Jones WG, editors.Germ cell tumours V. Proceedings of the 5th Germ Cell Tumour Conference; 2001 Sep 13-15; Leeds, UK. New York: Springer; 2002.

6. *Dissertation or thesis*

Borkowski MM. Infant sleep and feeding: a telephone survey of Hispanic Americans [dissertation]. Mount Pleasant (MI): Central Michigan University; 2002.

Other Published Material

Newspaper article

Tynan T. Medical improvements lower homicide rate: study sees drop in assault rate. The Washington Post. 2002 Aug 12;Sect. A:2 (col. 4).

Unpublished Material

In press or Forthcoming

Tian D, Araki H, Stahl E, Bergelson J, Kreitman M. Signature of balancing selection in Arabidopsis. ProcNatlAcadSci U S A. Forthcoming 2002.

Electronic Material

1. *Journal article on the Internet*

Abood S. Quality improvement initiative in nursing homes: the ANA acts in an advisory role. Am J Nurs. 2002 Jun [cited 2002 Aug 12];102(6):[about 1 p.]. Available from: http://www.nursingworld.org/AJN/2002/june/Wawatch.htmArticle

Article published electronically ahead of the print version:

Yu WM, Hawley TS, Hawley RG, Qu CK. Immortalization of yolk sac-derived precursor cells.Blood. 2002 Nov 15;100(10):3828-31. Epub 2002 Jul 5.

Article with document number in place of traditional pagination:

Williams JS, Brown SM, Conlin PR. Videos in clinical medicine.Blood-pressure measurement. N Engl J Med. 2009 Jan 29;360(5):e6. PubMed PMID: 19179309.

Article with a Digital Object Identifier (DOI):

Zhang M, Holman CD, Price SD, Sanfilippo FM, Preen DB, Bulsara MK. Comorbidity and repeat admission to hospital for adverse drug reactions in older adults: retrospective cohort study. BMJ. 2009 Jan 7;338:a2752. doi: 10.1136/bmj.a2752. PubMed PMID: 19129307; PubMed Central PMCID: PMC2615549.

2. *Monograph on the Internet*

Foley KM, Gelband H, editors. Improving palliative care for cancer [Internet]. Washington: National Academy Press; 2001 [cited 2002 Jul 9]. Available from: http://www.nap.edu/books/0309074029/html/.

3. *Homepage/Web site*

Cancer-Pain.org [Internet]. New York: Association of Cancer Online Resources, Inc.; c2000-01 [updated 2002 May 16; cited 2002 Jul 9]. Available from: http://www.cancer-pain.org/.

G. Submission Preparation Checklist

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

1. The submission has not been previously published elsewhere, is original and has been written by the stated authors.
2. The article is not currently being considered for publication by any other journal and will not be submitted for such review while under review by the Bangladesh Heart Journal.
3. The submission file is in Microsoft Word file format, and the figures are in JEPG or TIFF format.
4. The text is single-spaced; uses a 12-point font; employs italics, rather than underlining (except with URL addresses); and all illustrations, figures, and tables are placed within the text at the appropriate points, rather than at the end.
5. The text adheres to the stylistic and bibliographic requirements outlined in the Instruction to Authors. Make sure that the references have been written according to the ICMJE Recommendations Style.
6. Spell and grammar checks have been performed.
7. All authors have read the manuscript and agree to publish it.

H. Submission

Papers should be submitted to the Editor. Three copies of manuscript should be submitted duly signed by all authors with a copy of CD, to:

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Bangladesh.

Papers can also be submitted via the email using the following address:

Email: bangladeshheartj@yahoo.com

Original Article

Association of free Tri-iodothyronine Level with Cardiogenic Shock and Prognosis in Patients Hospitalized with Acute ST-elevation Myocardial Infarction Treated with Streptokinase Therapy

S M Nazmul Huda[1], Amal Kumar Choudhury[2], Jafrin Jahan[3], Masuma Tabassum[4], Atikur Rahman[5], Deb Dulal Debnath[6]

Abstract:

Background: Cardiogenic shock is the leading cause of death in patients with Acute ST-segment Elevation Myocardial Infarction (STEMI). Low free Tri-iodothyronine (FT3) levels are generally associated with poor prognosis in STEMI patients. This study was done to assess the association between FT3 levels and Cardiogenic shock in patients hospitalized with STEMI treated with streptokinase therapy.

Methods: This was an observational study of 140 patients of STEMI treated with streptokinase therapy in the department of cardiology, NICVD, Dhaka, Bangladesh from October 2018 to September 2019. The patients were divided into low FT3 (FT3 <3.5pmol/L; n = 70) and normal FT3 (FT3 e'' 3.5pmol/L; n = 70) groups according to FT3 levels measured within 24 hours after admission.

Results: During the index hospitalization period, 30 patients developed cardiogenic shock, 23(32.9%) in low FT3 group and 7(10.0%) in normal FT3 group. There were 17 deaths with 18.6% in low FT3 group and 5.7% in normal FT3 group (p=0.01). MACE occurred 45.7% in low FT3 group and 18.6%in normal FT3 group (p=0.001). The mortality in patients with cardiogenic shock was 43.3% compared to 3.6% in patients without cardiogenic shock. Multivariate logistic regression analysis showed FT3 level was an important predictor for cardiogenic shock in patients hospitalized with STEMI (p=0.01).

Conclusion: Low FT3 levels were strongly associated with cardiogenic shock in patients with STEMI. The FT3 level screening may be a simple and valuable way to predict Cardiogenic shock after STEMI.

Keywords: FT3, Cardiogenic shock, STEMI, MACE.

Conflict of Interest: There is no conflict of Interest.

(Bangladesh Heart Journal 2023; 38(1): 1-7)

Introduction:

Thyroid hormones have a broad range of effects on the heart and vascular system. The precise mechanism of the thyroid hormones at cellular and molecular level on heart have been investigated and well characterized.[1]

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Free Tri-iodothyronine (FT3) is the only thyroid hormone transported into the myocyte and thereby plays a major role in cardiovascular haemodynamics, cardiac filling, and systolic contractility.[2] The observation that even minor sub-clinical alterations in thyroid hormone levels can lead to adverse effects on the cardiovascular system is now recognized.[3]

Thyroid hormones metabolism is altered in severe illnesses and is characterized by low FT3 levels and normal-to-low free Thyroxine (FT4) and Thyroid Stimulating Hormone (TSH) levels which is known as the low-T3 syndrome or euthyroid sick syndrome.[4] The decline of FT3 level is caused either by increasing T3 catabolism or decreased conversion of the pro-hormone T4 into T3.[2] Inflammation, hypoxia, and oxidative stress modulate the activity of de-iodinase and thereby involve in the reduction of FT3 level.[5]

In acute myocardial infarction (AMI), the thyroid hormone system is rapidly down-regulated and it has tradi-tionally been interpreted as an adaptive process that reduces catabolism and conserve energy.[6] However, this theory is now challenged by clinical outcome data on baseline thyroid hormone dysregula-tion in AMI patients. Abnormal thyroid function tests, especially decreased FT3 have been shown to correlate with the severity of the disease and mortality in critically ill patients without prior known intrinsic thyroid diseases.[7,8] Furthermore, animal model studies showed that thyroid hormones have an important therapeutic role in limiting infarct size and improving myocardial function after AMI.[9]

Few clinical studies involving patients with AMI have addressed the possible correlation of thyroid hormone with the extent of myocardial injury. The Lower serum FT3 has been associated with increased serum levels of indicators of myocardial injury (troponin T and N-terminal pro-brain natriuretic peptide), as well as with lower left ventricular ejection fraction.[10] Lymvaios et al. (2011) also found a strong correlation of low FT3 with impaired left ventricular systolic function among AMI patients and describes FT3 levels as a predictor of ventricular functional recovery.[11]

Cardiogenic shock is the leading cause of death in patients with AMI and has a frequency of about 7-10%.[12,13] The incidence of CS has increased in recent years, while the exact reason is unclear, improved diagnosis and better access to healthcare are both likely contributory.[14] Cardiogenic shock caused by impairment of myocardial functions resulting in reduced cardiac output, end-organ hypoperfusion, and hypoxia which is evident clinically by presence of hypotension refractory to volume resuscitation and features of end-organ hypoperfusion. Despite all the advances in pharmacological, mechanical and reperfusion endeavors, in-hospital mortality in patients with cardiogenic shock continues to be as high as 50% to 80%.[15]

Although primary Percutaneous coronary intervention (PCI) strategy is recommended over fibrinolysis within indicated timeframe yet fibrinolysis is used widely as a means of revascularization in patients with acute STEMI especially in centres not offering primary PCI facilities in Bangladesh.[16] Cardiogenic shock occurs more amongst patients with ST-segment elevation myocardial infarction (STEMI vs NSTEMI, 7.5% vs 2.5%). It was observed that shock developed in 7.5% of patients with STEMI and in 2.5% of patients with non-ST-segment elevation myocardial infarction.[12]

Early identification of high risk patients helps to ensure appropriate therapies for their level of risk. Despite emerging innovative treatments, in-hospital mortality in patients with cardiogenic shock continues to be very high. Reduced FT3 along with other risk score could aid physicians to estimate risk of development of cardiogenic shock after acute STEMI more efficiently, improve their management and therefore contribute to reduce mortality and morbidity in STEMI patients.

Methodology

Study population

This prospective observational study was conducted at the department of Cardiology, NICVD, Dhaka, Bangladesh, from October 2018 to September 2019. A total of 140 cases were included in the study after considering the inclusion and exclusion criteria. The inclusion criterion was Patients with STEMI admitted in NICVD and treated with streptokinase therapy. The exclusion criteria were (1) History of Thyroid disease, overt Hypothyroidism or Hyperthyroidism, Amiodarone use within one month, (2) Previous myocardial infarction, Valvular, Congenital heart diseases and Cardiomyopathy, (3) Any severe co-morbidities (renal disease, previous stroke, COPD, anemia, malignancy, bleeding disorder. The study protocol was approved by the Hospital Ethics Committee. Informed written consent was obtained from the patients or a legal representative. Similar type of patient regarding age, sex, risk factors type of STEMI were selected to match both the groups as close as possible. Blood samples were collected within the first 24 h after admission. The serum levels of thyroid hormone (including FT3, FT4 and TSH) levels were measured by

Automatic Immune Assay System (Immunology Machine, Model Access 2, Beckman Coulter). The patients were divided into the low FT3 (FT3 <3.5 pmole/L; n=70) and the normal FT3 (FT3 e"3.5 pmol/L n=70) groups according to the FT3 levels. Baseline investigations (12 lead ECG, RBS, Serum Creatinine) and Echocardiography were carried out and noted.

Definition

Cardiogenic Shock was diagnosed as (1) systolic blood pressure <90 mmHg for >30 minutes or needed support to maintain blood pressure e"90 mmHg and evidence of end-organ hypoperfusion (decreased urine output, altered mental status and peripheral vasoconstriction).

Follow up

Routine follow-up was done every day during hospitalization period. The primary endpoint of this study was the development of cardiogenic shock and the secondary endpoint was all-cause death during hospitalization period.

Results:

The mean age of the patients was 61.8±12.5 years in low FT3 group and 58.6±12.7 years in normal FT3 group. The mean age of Low FT3 group was higher than Normal FT3 group which was not statistically significant (p=0.13). Regarding sex distribution, in Low FT3 group, 51 (72.9%) patients were male and 19 (27.1%) were female. In Normal FT3 group patients, 55 (78.6%) patients were male and 15 (21.4%) patients were female. Male female ratio was around 3:1. Statistically no significant association was seen in term of sex among the study groups (p=0.59) (Table I).

The mean RBS level was observed insignificantly higher in Low FT3 group and Normal FT3 group 8.1±3.4 vs. 7.3±2.9 mg/dl respectively (p=0.18). The mean serum creatinine was higher in Low FT3 group than Normal FT3 group (1.21±0.18 vs. 1.15±0.16 mg/dl) with statistical insignificant difference (p=0.06). The mean serum troponin I was higher in Low FT3 group than Normal FT3 group (28.2±19.6 vs. 19.8±16.1, p=0.007) with significant difference. It was observed that the mean LVEF% was significantly less in Low FT3 group than Normal FT3 group (43.45±4.83 vs 47.03±4.92, p=0.03) (Table I).

Distribution of study patients in respect to the site of MI showed similar frequencies in both group with no statistically significant association (Table II).

The mean level of FT3 was significantly lower in Low FT3 group than Group II (2.97±0.37 vs 4.25±0.48 pmol/L, p<0.001). The mean FT4 level was observed 13.22 ± 4.32 pmol/L in Low FT3 group and 14.64 ± 2.53pmol/L in Normal FT3 group with no statistical significance (p=0.12). TSH level is higher in Low FT3 group (2.26 ± 4.62 vs 1.45 ± 0.96) but without statistical significance (p=0.328) (Table III).

During the index hospitalization period, 30 patients developed Cardiogenic shock. Among them 23(32.1%)

Table-I

Baseline clinical, Biochemical and Procedural characteristics of study participants.

Characteristics	Group I (n= 70)	Group II (n=70)	p-value
Demographics			
Male – No. (%)	51 (72.9%)	55 (78.6%)	0.41^{ns}
Female – No. (%)	19 (27.1%)	15 (21.4%)	
Age (years)- mean±SD	61.8±12.5	58.6±12.7	0.002^{s}
CAD risk factors			
Diabetes mellitus– No. (%)	20 (28.6%)	16 (22.9%)	0.43^{ns}
Hypertension- No. (%)	37 (52.9%)		28 (40.0%)
0.12^{ns}			
Smoking- No. (%)	26 (37.1%)	35 (50.0%)	0.13^{ns}
Biochemical status			
RBS mmol/L (Mean ± SD)	7.3±2.9	8.1±3.4	0.18^{ns}
Serum creatinine mg/dl (Mean ± SD)	1.15±0.16	1.21±0.18	0.06^{ns}
Serum troponin I ng/ml (Mean ± SD)	19.8±16.1	28.2±19.6	0.007^{s}
Echocardiography			
LVEF (Mean ± SD)	47.03±4.92	43.45±4.83	0.003^{s}

p-value reached from Chi-square test and unpaired t-test.

Table-II

Distribution of the study patients according to site of MI (N=140).

Site of MI	Group I (n= 70)		Group II (n=70)		Total(n=140)		p value
	Number	%	Number	%	Number	%	
Anterior	35	50.0	33	47.1	68	48.6	0.73^{ns}
Inferior	14	20.0	20	28.6	34	24.3	0.23^{ns}
Extensive	14	20.0	11	15.7	25	17.9	0.51^{ns}
Inferior+RVI	7	10.0	6	8.6	13	9.3	0.77^{ns}

Group I: Acute ST

EMI patients with low FT3
Group II: Acute STEMI patients with normal FT3
Data were analyzed using Chi-Square Test.
ns = Not significant (p>0.05).

Table-III

Thyroid hormone status of the study patients (N=140).

Variable	Group I (n=70) Mean±SD	Group II (n= 70) Mean±SD	p value
FT3 (pmol/L)	2.97±0.37	4.25±0.48	<0.001^{s}
FT4 (pmol/L)	13.22±4.32	14.64±2.53	0.12^{ns}
TSH (mIU/L)	2.26±4.62	1.45±0.96	0.328^{ns}

Group I: Acute STEMI patients with low FT3
Group II: Acute STEMI patients with normal FT3
s = Significant (p<0.05)
ns = Not significant (p>0.05)
P value reached from unpaired t test.

in Low FT3 group and 7(10.0%) in Normal FT3 group. Cardiogenic shock occurred significantly more in Low FT3 group (p=0.001). There were 13 deaths with 18.6% in Low FT3 group and 5.7% in Normal FT3 group (p=0.01) (Table IV). Regarding Major Adverse Cardiac Event (MACE) which includes acute heart failure, significant arrhythmia, cardiogenic shock and cardiovascular death were higher in low FT3 group than normal FT3 group with statistically significant association (45.7% vs. 18.6%, p=0.001) (Table V).

The mortality in patients with cardiogenic shock was 43.3% compared to 3.6% in patients without cardiogenic shock. Mean hospital stay was observed more in patients with cardiogenic shock than without cardiogenic shock (5.29±1.65 vs. 3.67±1.01, p=0.001). (Table VI). The binary logistic regression analysis of Odds Ratio for characteristics of the subjects likely to cause Cardiogenic shock showed the low LVEF, elevated troponin I and low FT3 revealed to be significantly associated to develop Cardiogenic shock with the ORs being 1.89, 1.09 and 6.14 respectively (Table VII).

Table-IV

Comparison of FT3 by cardiogenic shock (n=140).

Variable	Cardiogenic shock (n=30) Mean±SD	No cardiogenic shock (n= 110) Mean±SD	p value
FT3 (pmol/L)	2.91±0.57	3.81±0.71	<0.001^{s}

s = Significant (p<0.05)
p value reached from unpaired t test.

Table-V

Comparison of patients by Major Adverse Cardiac Event (N=140).

MACE	Group I (n=70)		Group II (n= 70)		P value
	Number	%	Number	%	
Present	32	45.7	13	18.6	0.001[s]
Absent	38	54.3	57	81.4	

Group I: Acute STEMI patients with normal FT3
Group II: Acute STEMI patients with low FT3
p value reached from Chi Square test
s = Significant (p<0.05)

Table-VI

Multivariate regression analysis of predictors for Cardiogenic Shock (N=140).

Variables of interest	Regression coefficient (β)	Odds Ratio (OR)	95% CI of OR	p value
Agee"60 years	0.024	0.98	0.305 - 3.121	0.76[ns]
Smoking	0.245	1.42	0.242 - 8.182	0.11[ns]
Heart rate	1.378	2.78	1.783 – 55.107	0.03[s]
Serum creatinine	0.301	1.49	0.349 - 10.101	0.16[ns]
Low LVEF% <50%	0.402	1.89	1.211 – 11.913	0.02[s]
Elevated Troponin I	0.116	1.14	1.057 – 1.697	0.03[s]
Low FT3	1.332	3.43	1.008 – 11.644	0.01[s]

s = Significant (p<0.05)
ns = Not significant (p>0.05)

Table-VII

Comparison of outcomes by cardiogenic shock (n=140).

Variables	Cardiogenic shock (n=30)	No cardiogenic shock (n=110)	p value
Death No. (%)	13 (43.3)	4 (3.6)	<0.001[s]
Hospital stay (days)Mean ± SD	5.29±1.65	3.67±1.01	0.001[s]

s = Significant (p<0.05)
p value reached from Fisher's exact test and unpaired t test.

Discussion:

The mean age of study subjects was found 60.2±12.7 years. In the study by Zhang et al. (2012), the mean age 68.6±3.2 years was higher than that of our study. This disparity in distribution of age may be explained by the fact that Bangladeshis are prone to develop IHD which is often early in onset, follows a rapidly progressive course and having 5 -10 years earlier onset of first myocardial infarction.[16]

Regarding sex, the ratio of male and female patients was 3:1, however no statistically significant difference was seen between two groups. In Bangladesh, almost all of the studies related to coronary artery disease reported an overwhelming majority of male patients. This is probably due to fact that the females have less chance of CAD and less access to tertiary level care due to social issues.[17,18]

The mean serum troponin I was significantly higher in low FT3 group than normal FT3 group with significant difference (28.2±19.6 vs. 19.8±16.1, p=0.007). Troponin I correlate with severity of myocardial infarction and it is a traditional predictor of poor prognoses in patients with AMI.[19]

Regarding left ventricular ejection fraction, it is considered to be one of the major end points after treatment of

myocardial infarction and is strongly related to short and long-term survival. The mean left ventricular ejection fraction was significantly less in Low FT3 group than Normal FT3 group (43.45±4.83 vs 47.03±4.92, p=0.03). The results showed that FT3 was positively correlated with LVEF and revealed that the cardiac function is impaired in the low FT3 state in STEMI patients. This finding in our study was supported by previous studies done by Lymvaios et al. (2011) and Wang et al. (2013).[10.11]

In our study, we observed significantly more patients developed Cardiogenic shock in Low FT3 group than Normal FT3 group (37.1% vs 10.0%, p= 0.001). We found FT3 was independently associated with the increased development of Cardiogenic shock in hospitalized STEMI patients. Although multiple studies have established the risk profiles of cardiogenic shock, the associations of thyroid hormones with cardiogenic shock haven't been reported but multiple studies showed a low FT3 level is associated with the severity of STEMI.[19,20]

Considering the in-hospital death, 18.6% patients with Low FT3 died during their hospital stay, on the contrary 5.7% patients with normal FT3 were died. There was statistically significant difference in terms of mortality between two groups of patients (p=0.01). We found Major Adverse Cardiac Event (MACE) were higher in Low FT3 group than Normal FT3 group with statistically significant association (45.7% vs. 18.6%, p=0.001). We found more endpoint events, including cumulative deaths and MACE in the low FT3 group. These findings are consistent with relevant studies done by song et al. (2018), Lazzeri et al. (2012) and Zhang et al. (2012).[7,19.20,]

Among the patients with cardiogenic shock, the mortality was 43.3% compared to 3.6% in patients without cardiogenic shock. This high mortality rate in patients with cardiogenic shock following STEMI is consistent with previous studies.[21,22]

Thyroid hormones exert direct effects on hemodynamics, including increasing cardiac contractility, decreasing vascular resistance and so on. A low FT3 state after AMI changes the transcription of many cardiac structural and functional genes.[23] These changes in the expression of cardiac genes are also characteristic of pathological cardiac remodeling after myocardial infarction.[24] In addition, thyroid hormones are powerful regulators of vasculature in the adult myocardium; therefore, a low FT3 state would inhibit neovascularization in cardiac tissue after AMI, which would accelerate cardiac pathologic remodeling and heart failure.[25] These changes in a low FT3 state would accelerate pathological cardiac remodeling and worsen the cardiac function, which would lead to short and long-term adverse cardiac events. In this study we found that Low FT3 level is associated with increased development of Cardiogenic shock in comparison to normal FT3 level in Streptokinase treated STEMI patients.

This study has several limitations. Firstly, it's a single centre, small scale study with limited sample size. Secondly, FT3 levels were not measured at various time points and the changes of thyroid hormone over time after AMI are unknown.

Conclusion:

Low free Tri-iodothyronine (FT3) levels are strongly associated with cardiogenic shock development in Streptokinase treated STEMI patients. Measurement of FT3 levels may be a valuable and simple way to identify high-risk STEMI patients. These results provide useful insights into the management of patients with STEMI.

Death rate was significantly higher in patients with cardiogenic shock than without cardiogenic shock (43.3% vs. 3.6%, p<0.001). Mean hospital stay was observed more in patients with cardiogenic shock than without cardiogenic shock (5.29±1.65 vs. 3.67±1.01, p=0.001).

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Original Article

Effect of Open Heart Surgery on Lung Complications with their Incidence and Fate – a Single Center Study

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Abstract :

This prospective study was designed to determine the incidence of pulmonary complications after open cardiac surgery as well as to identity predisposing factors of these complications at NICVD, Dhaka, Bangladesh. The cumulative incidence of pulmonary complications after open heart surgery was 16.85% (15of 89) with a mortality rate of 33.33% (5 of 15) and the overall mortality among all patients was 5.62% (5 of 89). Pulmonary complications occurred in 15.9% of patients with coronary artery revascularization, 11.53% in patients with valvular replacement and 26.31% in patients with congenital heart disease.

ARDS occurred in 2.25% of patients with a mortality rate of about 100%,, pneumonia in 5.62%, atelectasis in 2.25%, pleural effusion in 5.62% and pneumothorax in 3.37%. The most predisposing factors were massive blood transfusion, re-exploration for control of post-operative bleeding, cardiopulmonary resuscitation and prolonged length stay in the intensive care unit.

Keywords:

(Bangladesh Heart Journal 2023; 38(1): 8-12)

Introduction:

The incidence of postoperative pulmonary complications (PPC) after open heart surgery varies from 6% to 70% depending upon the criteria used to define pulmonary complications 1 . The various components of the respiratory system; airways,lungs, chest wall, intercostals muscles; diaphragm and neural pathways, to and from these various components are subjected to damage caused by a variety of processes associated with cardiac surgery and cardio pulmonary bypass (CPB). Popular believe is that general anesthesia, surgical incision, cardiopulmonary bypass (CPB), ischemia time, intensity of surgical manipulation and number of drains may predispose patients to pulmonary function changes, which are highly relevant on the onset of pulmonary complications in cardiac surgery postoperative Cardiac surgery through either a sternotomy or thoracotomy has deleterious effects on the function of the muscle pump and the chest wall. Additionally, phrenic nerve damage resulting from cold topical solution applied inside the pericardium may cause mechanical problems. Left side cardiac distension or elevated pressure may cause alveolar edema, and transfusion reaction or allergic reaction to drugs (e.g. protamin) may increase capillary permeability leading to alveolar flooding. Mechanical alterations in lung function.2

(A) Atelectasis

It is the most common pulmonary complication after cardiac surgery occurring in about 70% of cases. During (CPB), the lungs are not perfused and they are allowed

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to collapse to functional residual capacity. When the lungs are subsequently re-expanded then variable degree of pulmonary atelectasis remains .

Etiology of pulmonary atelectasis

Preoperative factors : (1) Smoking, chronic bronchitis.(2) Obesity [decreased functional residual capacity (FRC)]. (3) Cardiogenic pulmonary edema.

(B) Acute lung injury [ARDS] and cardio-pulmonary by

Pass Activation of complement and neutrophils causes sequestration of neutrophils in the pulmonary microvasculature and an increase in the pulmonary capillary permeability .On the other hand reperfusion injury after ischemia generates oxygen free radicals and may also contribute to lipid peroxidationat this time . There is an increased systemic level of thromboxane during (CPB). Thromboxane released from platelets activated by extra corporeal circuit, and its profound effects on vasoconstriction and platelet aggregation could further injure the microcirculation .

(C) Postoperative pneumonia

Pneumonia remains the greatest threat to survival that exists in a surgical patient. Patients are often debilitated by the effects of the disease process, intravenous lines, bladder catheters, endotracheal tube and surgical wound. The incidence of pneumonia is about 5–19%. More than 90% of nosocomial pneumonia is bacterial and in 50–70% of cases the responsible organisms are gram negative bacilli. The most important causative gram negative organisms include Klebsiella species, Escherichia coli and Pseudomonas aeruginoza .2

Very few studies have focused on the intraoperative and postoperative risk factors responsible for the development of pulmonary complications in patients undergoing cardiac surgery using cardiopulmonary bypass. In present study we determined the frequency of post-operative pulmonary complications (PPCs) and inter-operative factors associated with PPCs in patients undergoing cardiac surgery using cardio-pulmonary bypass.3

Methods:

This prospective observational study was arranged in National Institute of cardiovascular Diseases Dhaka Bangladesh . A total number of 89 patients undergoing cardiac surgery using cardiopulmonary bypass (CPB) from Jan 2015 to August 2016 in a single cardiac surgical unit were included. Patients undergoing coronary artery bypass grafting (CABG) and valvular operations and Patients undergoing congenital cardiac procedures were selected. In all patients standard procedures were used for surgery. All procedure was done through median skin incision. Standard cardiopulmonary bypass apparatus was used in all patients incorporating membrane oxygenator and arterial line filters. Venus cannulation was done through right atrium using either two single venous cannulas or a two stage single venous cannula. And arterial cannulation was done through ascending aorta using either angles tip or straight tip aortic perfusion cannula. Lactated ringer was used to prime CPB circuit. After establishing CPB, temperature was lowered to 30 to 28 degree Celsius to maintain moderate hypothermia during surgery. Cold blood cardioplegia was used to arrest and protect the heart of patients undergoing CABG and tepid blood cardioplegia was used for valvular procedures after application of aortic cross-clamp. All patients were weaned off from CPB after re-warming the patient to 36.5 to 37 degree Celsius. All patients were shifted to intensive care unit (ICU) in stable condition. And weaned off from mechanical ventilation in ICU. The senior anesthetist and the pulmonologist of the hospital noted data regarding postoperative pulmonary complications. Duration of mechanical ventilation > 48 hours or need for re-intubation of patients was considered as respiratory failure. Pneumonia was labeled by the presence of fever

and sputum along with presence of findings of pneumonia on chest X-rays and on laboratory reports weaned off from mechanical ventilation in ICU. The senior anesthetist and Any death during hospital stay or within one month after surgery due to the results of surgical complications was considered operative mortality. Data analysis was done using SPSS v23 software. Chi-square tests were used to determine the effect of post-operative pulmonary complications on operative mortality and to compare the frequency of PPCs in valvular and CABG patients. Logistic

regression analysis was used to determine the association of pre-operative and intra-operative variables on the incidence of PPCs after surgery. P-value d” 0.05 was taken as significant effect.

Results:

The study included 89 patients (65 males and 24 females) the average age was 41 years that ranged from 15 to 68 years. The patients were classified into three groups:

(1) Coronary artery revascularization includes 44 patients (36 males and 8 females with mean age of 51.9 years.

(2) Valve replacement includes 26 patients (18 males and 8 females) with mean age of 15–53 years. (3) Congenital heart disease includes 19 patients (11 males and 8 females) with mean age of 4.9 years.

The cumulative incidence of pulmonary complications after open heart surgery was 16.85% (15of 89) with a mortality rate of 33.33% (5of 15) and the overall mortality among all patients was 5.62% (5 of 89).

Acute respiratory distress syndrome (ARDS) developed in; one patient with congenital heart diseases, and 1patient with coronary artery revascularization. The first patient with coronary artery revascularization presented with dyspnea, tachypnea, hypoxemia, and bilateral diffuse pulmonary infiltration. Mechanical ventilation was done, the patient underwent mechanical ventilation for 5 days with no response to supportive therapy and died due to multiorgan failure. ARDS occurred in a female patient 3 years old after closure of ventricular sepal defect, after estubation by 8 h she developed tachypnea, hypoxemia, and diffuse pulmonary infiltrations, and intubation and mechanical ventilation was done for 3 days. with no response to supportive therapy and died. The incidence found in this study was 2.25% (2 out of 89)

Right lower lobe atelectasis developed in one patient with coronary arteries revascularization during the first 24 h postoperative and resolved by physiotherapy. Mechanical ventilation for 7 days was required in another patient with congenital heart disease and deteriorated further to death after lower lobe inflation guided by repeated endotracheal intubations. The incidence of early postoperative atelectasis was 2.25% (2 of 89).

Right lower lobe pneumonia occurred in one patient with CABG surgery in elderly age, one patient with mitral valve replacement, and another patient with congenital heart disease, they presented with fever, leucocytosis, tachypnea, hypoxemia, and right lower lung zone heterogeneous opacity, the causative organisms were Klebsiella spp. And streptococcal pneumonia, respectively. However its very unfortunate to mention that the poor girl of 4 years of age of VSD died in 4th post operative day due to pneumonia with high fever from gm negative bacteria Pseudomonas aeruginosa resistant to all antibiotics including meropenum with concomitant wound dehiscence and mediastinitis .The incidence of pneumonias in early postoperative period was 3.37% (3 of 89).The total incidence of early postoperative pulmonary complications after open heart surgery was 16.85% (15 of 89), while the incidence of mortality was 5.62% (5 of 89).

A total of three patients one being 45 years old in CABG surgery one patient of 30 years old with AVR and one patient of Incomplete AV canal defect developed post operative pneumothorax on early post operative days and recovered after tube thoracotomy who had finding of intact pleura on per operative aspect on that side without any chest drain tube insitu. The incidence was 3.37% (3 out of 89)

Finally, five patients developed pleural effusion in late post operative period on left side in late postoperative period after discharge on subsequent follow up. The incidence was 5.62% (5 out of 89)

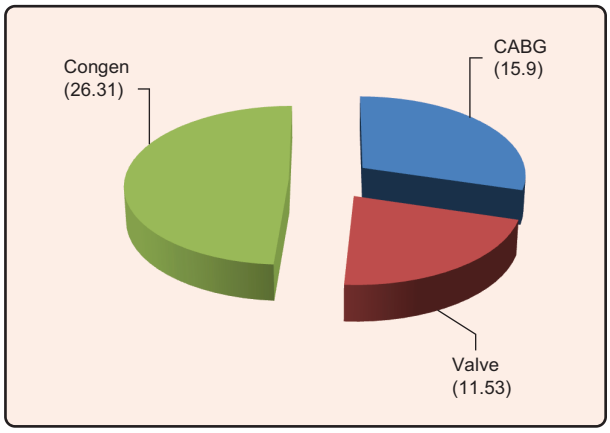

Fig.-1: *Percentage of PPC in CABG, Valve and Congenital cases*

Table-I

showing different types of PPC and Outcome

	A	R	DS	Ate	lec	tasis	Pne	umo	thorax	PL	EF		P	N	M
	Age	Sex	Outcome	Age	Sex	Outcome	Age	sex	outcome	Age	sex	outcome	A	S	O
CABG	60	M	Died	50	M	S	45	M	S	55 62	F M	S S	65	M	D
Valve							30	F	S	39 25	M M	S S	37	F	S
Congen	03	F	Died	04	M	Died	06	M	S	08	M	S	04	F	D
Total	2			2			3			5			3		

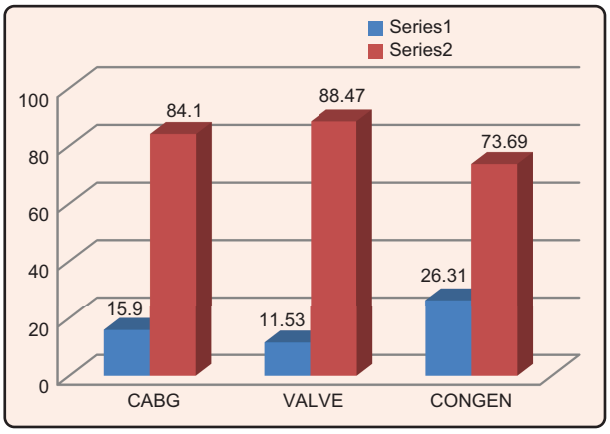

Fig.-2: *Percentace of PPC and without PPC*

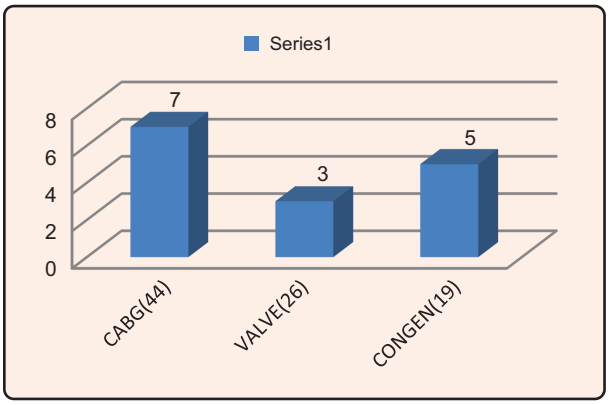

Fig.-3: *number of PPC*

Discussion:

Pulmonary complications are less after cardiopulmonary bypass but can be life threatening in some cases. The documented incidence of PPCs ranges from 3% to 16% after CABG and 5%-7% after valvular heart surgery and 6-76% after congenital heart surgery. 3

The aim of this study was to measure the incidence of pulmonary complication after open heart surgery, 89 patients (65 males and 24 females) were included for this study, open heart surgery was done using Cardiopulmonary bypass (CBP) in all 89 patients . It should come as no surprise that cardiac surgery can have pronounced effects on lung function. The anesthetic agents, chest wall alteration, and direct lung manipulation can all affect pulmonary parameters. Functional residual capacity (FRC) can decrease by up to 20% with anesthesia (Szelowski LA, et al. Curr Probl Surg. 2015;52[1]:531), and the thoracic manipulation and alteration of rib cage mechanics with a classic median sternotomy approach can lead to decreases in forced vital capacity (FVC) and expiratory volume in the first second of forced expiration (FEV1) that can last for months after surgery. Use of the cardiopulmonary bypass circuit can also lead to bronchoconstriction.4

The most frequent pulmonary consequence of cardiac surgery is atelectasis, seen on postoperative chest radiographs in approximately 50% to 90% of patients (Szelowski LA, et al. Curr Probl Surg. 2015;52[1]:531). In our study the incidence was found in atelectasis was 2.25%. in another study in Pakistan it was found 3.86% (Naveed A et al.2017) Incidence of atelectasis in our study was comparable to this study. The reduction in incidence of atelectasis in our study may be due to the practice of pre-operative incentive spirometry practice in all patients before surgery and the use of positive end expiratory pressure (7-8 cmH2O) during mechanical ventilation after surgery. Induction, apnea during cardiopulmonary bypass, manual compression of the lungs for surgical exposure, internal mammary harvesting, and pleurotomy can lead to atelectasis in the intraoperative setting while weak cough, poor inspiratory efforts, interstitial edema, and immobility further contribute postoperatively (Weissman 2004). While frequently seen, clinically significant pulmonary consequences from this radiographic finding alone are rare (Weissman 2004).4

Pleural effusions are seen on immediate postoperative chest radiographs in the majority of patients. Additionally, 10% to 40% of patients develop pleural effusions 2 to 3 weeks after surgery secondary to postpericardiotomy syndrome. In our study pleural effusion was noted as late PPC, the incidence of which is 5.62%. While some effusions require drainage and further intervention (eg, hemothorax), most effusions require no specific treatment and resolve over time (Weissman 2004).4

The prevalence of pneumonia following cardiac surgery varies based on differences in study populations and diagnostic criteria, but it remains an important source of morbidity and mortality. In our study, the incidence of pneumonias in early postoperative period was 3.37% In one series, postoperative pneumonia occurred in 3.1% of patients, with higher rates observed in patients who were older, had worse left ventricular ejection fraction, had COPD, experienced longer bypass times, and received more red blood cell transfusions in the operating room (Allou N, et al. Crit Care Med. 2014;42[5]:1150). This is very much compatible with findings of our study. A meta-analysis found that an average of 6.37% of patients developed ventilator-associated pneumonia (VAP), and

this rose to 35.2% in those receiving ventilation for greater than 48 hours. Those who developed VAP had an odds ratio of dying of 15.18 (95% CI 5.81-39.68) compared with those who did not (He S, et al. J Thorac Cardiovasc Surg. 2014;148[6]:3148).4

A small proportion of patients go on to develop ARDS. While relatively uncommon, ARDS carries a high mortality rate. In our study ARDS incidence was found 2.25% with high mortality. Many possible etiologies for ARDS in cardiac surgery patients have been proposed, including an inflammatory response related to the cardiopulmonary bypass circuit, reperfusion injury secondary to reduced pulmonary blood flow during bypass, protamine administration, transfusion, hypothermia, and lack of ventilation during bypass (Weissman 2004); (Stephens RS, et al. Ann Thorac Surg. 2013;95[3]:1122). Type of surgery may also play a role, as patients who undergo aortic surgery are at an even greater risk (Stephens 2013). As with other cases of ARDS, treatment is supportive: low tidal volume ventilation and careful management of fluid balance, as well as paralysis, prone positioning, and consideration for extracorporeal membrane oxygenation (ECMO), as appropriate (Stephens 2013).4

Conclusion:

The cumulative incidence of pulmonary complication after open heart surgery was 16.85%, with an overall mortality 33.33% (5 of 15) and mortality among all patients was 5.62% (5 of 89). ARDS occurred in 2.25%, pneumonia occurred in 2.79% atelectasis occurred in 3.37%, pleural effusion occurred in 3.37%.

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Original Article

Wave-Wise Comparison of Demographics, Clinical Characteristics & In-Hospital Outcome of COVID-19 Pandemic in Bangladesh: Single Centre Study

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Abstract:

Background: Most countries in the world faced two waves of Corona virus disease-19 (COVID-19). But there is a lack of data regarding the wave-wise comparison of epidemiological and clinical characteristics of the COVID-19 outbreak. This study aimed to compare the demographics, clinical characteristics, and in-hospital outcomes of two waves of the COVID-19 pandemic in Bangladesh.

Methods: This prospective cross-sectional study was carried out at the National Heart Foundation Hospital & Research Institute. From April 3, 2020, to January 28, 2021, was considered the first wave, and from February 27, 2021, to September 25, 2021, was considered the second wave. COVID-positive patients and all admitted patients who became COVID-19 positive during these periods were included in this study for comparison.

Results: The first wave included 727 patients, and the second wave included 858 patients. The mean age of the patients in the first wave was 48.11 15.75 years, and in the second wave it was 50.65 16.63 years. Males were predominant in both waves. Healthcare personnel were less affected during the second wave (11.9% vs. 30.7%; p=0.001). Hypertension, chronic kidney disease, and cardiovascular disease were more prevalent in the second wave (p 0.05), and dyslipidemia and obesity in the first wave (p<0.05). During the second wave, 80.5% of patients were unvaccinated. Asymptomatic patients were predominant in the second wave (26.9% vs. 17.5%; p=0.001). COVID-19-related symptoms (fever, body ache, headache, anosmia, sore throat, shortness of breath, and diarrhea) were less prominent during the second wave (p<0.05). Oxygen requirements and IV antibiotic use were higher during the second wave (p<0.05).

Asymptomatic & severe disease form were prevalent in second wave (p<0.05). Mortality rate was more during second wave (5.1% vs 3.4%; p=0.1). Age > 50 years, severe left ventricular dysfunction, severe and critically ill patients were the independent predictor of mortality.

Conclusion: In comparison to the first wave, during the second wave symptoms were less prominent, asymptomatic and severe disease forms were more prevalent & mortality rate was high. Unvaccinated persons are more prone to affected by COVID-19.

Key wards: *COVID-19, first wave, second wave, vaccine status, clinical features, in-hospital outcome.*

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Introduction:

Corona virus disease-19 (COVID-19) caused by severe acute respiratory syndrome corona virus-2 (SARS-CoV-2) is the most devastating epidemic which affected 1550371 people and 27,393 deaths in Bangladesh till

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25th September 2021[1]. For a developing country like Bangladesh, COVID-19 has appeared as a challenging catastrophe. Bangladesh faced first wave of COVID-19 without preparedness like other countries. During first wave, the government took numerous measures to fight the COVID-19 pandemic in Bangladesh such as increasing healthcare facilities, screening, rescuing, and lockdown, increasing social awareness regarding the spread of the disease and its probable impact, usefulness of using mask, restriction on local and international air travels, and switch to online educational activities for students instead of on campus activities[2]. Vaccination, recruitment of more HCP, addition of more hospitals & ICU facilities lead to proper preparedness against COVID-19 during second wave in Bangladesh. The increased number of cases in the second wave may be due to genetic mutation of virus, widespread disregard to the 'COVID appropriate behaviours' by the public, using highly variable quality of masks, increased number of asymptomatic patients, and the higher testing[3]. It is observed that the mutant virus has more effective transmission capability and lesser incubation period[3]. In Japan, in comparison to first wave data from the second wave indicated a demographic shift toward a younger population with fewer comorbidities, a lower proportion of severe patients at admission, and decreased mortality[4].

Like other countries, Bangladesh also experienced two waves of COVID-19. There is a lack of data regarding comparison of epidemiological and clinical characteristics of the first wave and second wave of COVID-19 outbreak. This study aimed to compare the demographics, clinical characteristics and in-hospital outcome of two waves of COVID-19 pandemic in Bangladesh.

Material and Methods:

This prospective cross-sectional study was carried out at National Heart Foundation Hospital & Research Institute. From 3rd April, 2020 to 28th January, 2021 was considered as the first wave and from 27th February, 2021 to 25th September, 2021was considered as second wave (Figure 1)[5]. WHO defined a pandemic, consider better control if the infection below 5%[6]. The start points for both the first and second wave were defined during the time of infection rate above 5% & end points were defined during the time of infection rate below 5%. COVID-positive patients & all admitted patients who become COVID-19 positive during these periods were included in this study for comparison.

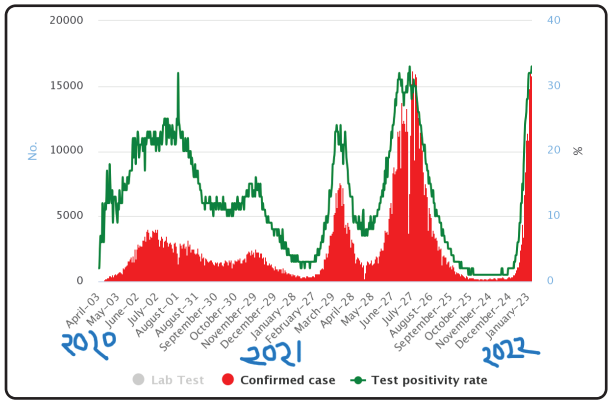

Fig.-1: *Infection rate of COVID-19 patients according to time-frame.*
Abbreviation: COVID-19: coronavirus disease 2019.

Demographic information included gender, age, risk factors and co-morbidities (diabetes mellitus, hypertension, smoking, dyslipidemia, obesity, cardiovascular disease, cerebro-vascular disease, chronic obstructive pulmonary disease /asthma, chronic kidney disease, pregnancy). The degrees of severity of COVID-19 were classified as mild, moderate, severe, and critical ill[7,8]. Mild type was defined as mild clinical symptoms without imaging findings of pneumonia. Moderate type was defined as clinical symptoms (fever or other respiratory symptoms) with imaging findings of pneumonia. Patients with severe type had any of the following parameters: (I) respiratory distress, respiratory rate e"30 times/min; (II) oxygen saturation d"93% at rest. Also patients showing a rapid progression (>50%) on chest imaging within 24–48 hours was regarded as severe type. Patients with critical ill type had to meet any of the following standards: (I) respiratory failure requiring mechanical ventilation; (II) shock; (III) complicated extrapulmonary organ failure requiring care in the intensive care unit.

Continuous variables are described using the mean and standard deviation (SD), and compared using unpaired Student's't' test. Discrete variables are expressed as number of cases and percentage. Comparison between variables was performed using the two-sided chi-square tests for discrete variables, or Fisher's exact tests (expected frequency <5). Binary logistic regression was used to identify the predictors of mortality. Variables significantly related to in-hospital outcome such as mortality in univariate analysis were included in a binary logistic regression model to identify independent predictors of the mortality. A two-sided p value <0.05 was

considered statistically significant. All analyses were performed using SPSS statistical software version 16.0 (SPSS Inc., Chicago, IL, USA).

Results:

First wave included 727 patients and second wave included 858 patients. The mean age of the patients in first wave was 48.11 ±15.75 years and in second wave was 50.65 ± 16.63years. In the first wave the number of positive cases between 1 and 20 years, 21 and 40 years, 41 and 60 years, and 81 and 100 years were 1.7%, 32.2%, 43.5%, 22.0%, and 0.6%, respectively. The number of positive cases between the age 1 and 20 years, 21 and 40 years, 41 and 60 years, 61 and 80 years, and 81 and 100 years were 3.0%, 23.0%,45.2%, 27.1%, and 1.7%, respectively in the second wave.

Male were predominant in both waves. Healthcare personnel were less affected during second wave (11.9% vs 30.7%; p=0.001). Hypertension, chronic kidney disease and cardiovascular disease were more prevalent in second wave (p<0.05) & dyslipidemia and obesity in first wave (p<0.05). Comparison of baseline characteristics between the first and second wave COVID-19 are outlined in table 1. During first wave patients without co-morbidities were more prevalent than second wave. During second wave patients with <4 co-morbidities were more prevalent than first wave.

During first wave vaccine was unavailable and all COVID-19 patients were unvaccinated. During second wave, 80.5% COVID-19 patients were unvaccinated, 14.6% patient received first dose vaccine and 4.9% patients received first & second dose vaccine (Figure 2).

Table-I

Comparison of baseline characteristics between the first and second wave COVID-19 in Bangladesh

Variables	First wave (n=727)	Second wave (n=858)	P value
Age (Mean age ±SD)	48.11 ±15.75 years	50.65 ± 16.63years	0.7#
<20 years	12(1.7%)	26(3.0%)	0.074
21-40 years	234(32.2%)	197(23.0%)	0.001
41-60 years	317(43.5%)	390(45.2%)	0.46
61-80 years	160(22.0%)	230(27.1%)	0.019
>80 years	4(0.6%)	15(1.7%)	0.029
Gender Male	470(64.6%)	557(64.9%)	0.9*
Female	257(35.4%)	301(35.1%)	
HCP	223(30.7%)	102(11.9%)	0.001*
Non-HCP	504(69.3%)	756(88.1%)	
Risk factors & co-morbidities			
HTN	374(51.4%)	548(63.9%)	0.001*
DM	304(41.8%)	343(40.0%)	0.458*
Smoking	229(31.5%)	293(34.1%)	0.263*
Dyslipidemia	283(38.9%)	176(20.5%)	0.001*
Cardiovascular disease	425(58.5%)	623(72. 6%)	0.001*
COPD/BA	53(7.3%)	46(5.4%)	0.114*
Obesity	249(34.3%)	230(26.8%)	0.005*
CKD	234(32.2%)	352(41.0%)	0.001*
Pregnancy	9(1.2%)	2(0.2%)	0.001*
Number of co-morbidities			
0	129(17.7%)	89(10.4%)	
<4	275(37.9%)	394(45.9%)	0.0001*
≥4	323(44.4%)	375(43.7%)	

Abbreviation: COVID-19: coronavirus disease 2019; HCP: healthcare personnel; non-HCP: non-healthcare personnel; SD: standard deviation; HTN: hypertension; DM: diabetes mellitus; COPD: chronic obstructive pulmonary disease; BA: Bronchial asthma; CKD: chronic kidney disease. *Chi square test was done to find out the significance; #Student's 't' test was done to find out the significance.

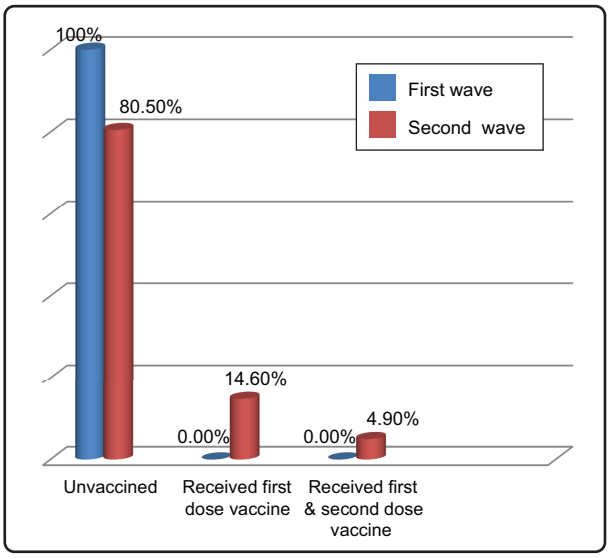

Fig.-2: *Vaccine status of the COVID-19 patients during first & second wave*

Symptomatic patients were more predominant during first wave (82.5% vs 73.1%; p=0.001) and asymptomatic patients were in second wave (26.9% vs 17.5%; p=0.001). COVID-19 related symptoms (fever, body ache, headache, anosmia, sore throat, shortness of breath and diarrhea) were less prominent during second wave (p<0.05). Comparison of clinical characteristics between the first and second wave COVID-19 are depicted in table II.

Oxygen requirement was more during second wave (45.9% vs 37.4%; p<0.05). High flow nasal cannula was unavailable during first wave but in second wave it was used in 1.6% (14) patients. About 40% patients received IV antibiotics during second wave, although 38.2% patients did not receive antibiotics. Comparison of treatment option between the first and second wave COVID-19 are detailed in table III.

Regarding disease severity, asymptomatic patients were more prevalent during second wave in comparison to first wave. Mild, moderate and critical ill forms of disease severity were more predominant during first wave and severe form during second wave respectively. Mortality rate was high during second wave.

Wave-wise comparison of in-hospital outcome of COVID-19 patients is shown in table IV.

Table V shows the univariate analysis of in-hospital outcome of study population. Age more than 50 years, non-health care personnel, presence of cardiovascular disease, EF category, disease severity, diabetes mellitus and hypertension significantly related with in-hospital mortality.

Out of 1585 patients, 69 patients died in this study. Univariate analysis factors analysis several factors were significantly related with in-hospital mortality. Based on these variables, binary logistic regression using the forward method was performed, and we found that age >50 years, severe & critically ill form and severe LV dysfunction were the independent predictor of mortality (Table VI).

Table-II

Comparison of clinical characteristics between the first and second wave COVID-19

Variables	First wave (n=727)	Second wave (n=858)	P value*
Clinical presentation			
Symptomatic	600(82.5%)	627(73.1%)	0.001
Asymptomatic	127(17.5%)	231(26.9%)	
Presenting symptoms			
Fever	481(66.2%)	425(49.5%)	0.001
Fatigue	210(28.9%)	231(26.9%)	0.38
Cough	269(37.0%)	313(36.5%)	0.83
Body ache	140(19.3%)	121(14.1%)	0.006
Headache	125(17.2%)	93(10.8%)	0.001
Anosmia	120(16.5%)	62(7.2%)	0.001
Sore throat	91(12.5%)	45(5.2%)	0.001
Shortness of breath	80(44.2%)	37(26.6%)	0.002
Diarrhea	50(6.9%)	30(3.5%)	0.002
Generalized itching	29(4.0%)	23(2.7%)	0.14
Left ventricular ejection fraction			
Good	439(60.4%)	475(55.4%)	0.076
Mild	156(21.5%)	222(25.9%)	
Moderate	113(15.5%)	128(14.9%)	
Severe	19(2.6%)	33(3.8%)	

Abbreviation: COVID-19: coronavirus disease 2019. *Chi square test was done to find out the significance.

Table-III
Distribution of treatment

Variables	First wave (n=727)	Second wave (n=858)	P value*
Oxygen therapy	272(37.4%)	394(45.9%)	0.001
HFN cannula	0(0%)	14(1.6%)	0.001
Antibiotics			
IV	112(15.4%)	336(39.2%)	0.001
Oral + IV	62(8.5%)	29(3.4%)	
Oral	482(66.3%)	165(19.2%)	
Not received	71(9.8%)	328(38.2%)	
Antibiotics			
Single	493(67.8%)	416(48.5%)	0.001
Double	163(22.4%)	114(13.3%)	
Not received	71(9.8%)	328(38.2%)	
Steroids	56(7.7%)	66(7.7%)	0.99
Favipiravir	39(5.4%)	6(0.7%)	0.001
Remdesivir	35(4.8%)	57(6.6%)	0.12
Ivermectin	539(74.1%)	283(33.0%)	0.001
Hydroxy-chloroquine	4(0.6%)	0(0%)	0.03
Enoxaparine	447(61.5%)	646(75.3%)	0.001

Abbreviation: HFN: high flow nasal; IV: intravenous. *Chi square test was done to find out the significance.

Table-IV
In-Hospital outcome

Variables	First wave (n=727)	Second wave (n=858)	P value*
Disease severity			
Asymptomatic	127(17.5%)	231(26.9%)	0.001
Mild	491(67.5%)	532(62%)	0.02
Moderate	51(7.0%)	3(0.3%)	0.001
Severe	44(6.1%)	89(10.5%)	0.002
Critical ill	14(1.9%)	3(0.3%)	0.002
Mortality	25 (3.4%)	44(5.1%)	0.1

Abbreviation: COVID-19: coronavirus disease 2019. *Chi square test was done to find out the significance.

Table-V

Univariate analysis of in hospital outcome of study population

Variables	Outcome		P value*
	In hospital deathf(%)#	Recoveredf(%)#	
Age group			
<50 Y	9 (1.1)	791 (98.9)	0.000
>50 Y	60 (7.6)	725 (92.4)	
Gender			
Male	51 (5.0)	976 (95.0)	0.122
Female	18 (3.2)	540 (96.8)	
NOHCP/HCP			
NOHCP	67 (5.3)	1193 (94.7)	0.000
HCP	2 (0.6)	323 (99.4)	
CVD			
Present	55 (5.2)	993 (94.8)	0.015
Absent	14 (2.6)	523 (97.4)	
Obesity			
Non obese	54 (4.9)	1052 (95.1)	0.117
Obese	15 (3.1)	464 (96.9)	
EF Category			
Severe LV Dysfunction	6 (11.5)	46 (88.5)	0.001
Moderate LV Dysfunction	11 (4.6)	230 (95.5)	
Mild LV Dysfunction	25 (6.6)	353 (93.4)	
Good Function	27 (3.0)	887 (97.0)	
Disease Severity			
Asymptomatic	2 (0.6)	356 (99.4)	0.000
Mild	18 (1.8)	1005 (98.2)	
Moderate	1 (1.9)	53 (98.1)	
Severe	37 (27.8)	96 (72.2)	
Critically ill	11 (64.7)	6 (35.3)	
Diabetes Mellitus			
Diabetic	44 (6.8)	603 (93.2)	0.001
Non diabetic	25 (2.7)	913 (97.3)	
Blood Pressure			
Hypertensive	51 (5.5)	871 (94.5)	0.007
Normotensive	18 (2.7)	645 (97.3)	

Abbreviation: HCP: healthcare personnel; non-HCP: non-healthcare personnel; CVD: cardiovascular disease; EF: ejection fraction; LV: left ventricular. # Value in the parenthesis shows the corresponding row percentage; *Chi square test to find out significance.

Table-VI

Multivariate analysis of in hospital outcome of study population

Variables	OR	Sig.	95.0% CI	
			Lower	Upper
Age >50 years	3.222	.005	1.423	7.298
Gender Male	1.457	.273	.743	2.859
NOHCP	2.914	.188	.593	14.328
CVD	.793	.579	.349	1.802
Obese	.743	.403	.371	1.489
LV Dysfunction		.059		
· Good function	1.837	.090	.910	3.709
· Mild LV Dysfunction	.624	.315	.248	1.567
· Moderate LV Dysfunction	2.039	.229	.639	6.507
· Severe LV Dysfunction		.000		
Disease Severity				
· Asymptomatic		.000		
· Mild	3.083	.136	.703	13.520
· Moderate	2.746	.418	.238	31.653
· Severe	50.707	.000	11.732	219.156
· Critically ill	288.919	.000	48.680	1714.738
DM 1.213	.534	.660	2.227	
HTN	.943	.868	.473	1.879

Variable(s) entered on step 1: Age>50Y, Gender, NOHCP, CVD, Obesity, EF category, Disease Severity, DM, HTN. Abbreviation: non-HCP: non-healthcare personnel; CVD: cardiovascular disease; EF: ejection fraction; LV: left ventricular; DM: diabetes mellitus; HTN: hypertension.

Discussion

Important findings of this study are: 1) Healthcare personnel (HCP) were less affected during second wave (11.9% vs 30.7%); 2) Patients aged 21-40 years were more prevalent in first wave (32.2% vs 23%); 3) Patients aged more than 60 years (28.8% vs 22.6%) were more prevalent in second wave; 4) Vaccinated people were less affected by COVID-19; 5) COVID-19 related symptoms were less prominent during second wave; 6) Asymptomatic & severe disease forms were prevalent in second wave; 7) Mortality rate was higher during second wave (5.1% vs 3.4%; p=0.1) and, 8) Age > 50 years, severe left ventricular dysfunction, severe and critically ill patients were the independent predictor of mortality.

Understanding how SARS-CoV-2 infection varies across the age spectrum is the key for developing responses to the COVID-19 epidemic[9]. There is a trend toward decreasing age among persons with laboratory-confirmed SARS-CoV-2 infection, but that these trends seem to be specific to the outpatient population[9].

In our study, the mean age of the patients was higher in second wave than first wave (50.65± 16.63 vs 48.11± 15.75 years). In contrast to our study, the mean age of patients in the second waves was significantly lower in other two studies (53.60±23.05 and 56.84±18.29 years)[10] and (58 ± 26 *vs*. 67 ± 18 years)[11].

Also in Japan, patients in the second wave tended to be younger (median age, 37/ vs 56 years)[4]. Patients aged up to 40 years were more prevalent in first wave (33.9%) than second wave (26%). Although the exact cause for the difference of the patient's age between the two waves is unknown, it has been hypothesized that lower infection rate among HCP may be a reason as they were relatively young. Surprisingly, about 56.2% of individuals were positive below the age of 40 in the first wave, while during the second wave, this number was increased and reached 65.6% in another study[6].

Hypertension, chronic kidney disease and cardiovascular disease were more prevalent in second wave. It may be due to older age of the patients. Similar to our study, Jalali et al.[10] showed cardiovascular disorders, hypertension, chronic renal disorders, malignancies, and opium use were more prevalent among the study population during second wave as compared to the first

wave. A recent meta-analysis of 16 published studies with 3994 patients, found out that comorbidities had a major effect on patients with COVID 19 and leads to higher chances of serious events[12]. The presence of chronic respiratory disorders, chronic kidney diseases, cardiovascular diseases, and diabetes mellitus associated with a 6.6, 5.3, 4.5, and 3.07 times higher risk of developing serious events in COVID 19 patients, respectively[12]. Another meta-analysis by Li et al.[13] found that hypertension and cardio-cerebrovascular diseases had a statistically significant impact on ICU admission.

One of the important findings of this study was that HCP were less affected during second wave (11.9% vs 30.7%). During first wave vaccine was not available in Bangladesh. Lower infection rate among HCP may be due to proper utilization of personal protective measures and vaccination of HCP in priority basis as front line fighter against COVID-19.

COVID-19 related symptoms (fever, body ache, headache, anosmia, sore throat, shortness of breath and diarrhea) were significantly less prominent during second wave in our study. It may be due to mutation of virus. In contrast to our study, gastro-intestinal manifestations were more common in the second wave in another studies[3,10.11].

Vaccination is the simplest method to regulate rapidly spreading infectious diseases[14]. Vaccines were unavailable during first wave in our country. Later on, several vaccines were invented to provide acquired immunity against the coronavirus[15]. Unvaccinated people (80.5%) were more commonly affected by COVID-19 in our study in the second wave. COVID-19 vaccination has substantially altered the course of the pandemic, saving tens of millions of lives globally[16].

Oxygen requirement was more during second wave (45.9% vs 37.4%). High flow nasal cannula was unavailable during first wave but in second wave it was used in 1.6% patients. About 40% patients received IV antibiotics during second wave, although 38.2% patients did not receive antibiotics. Regarding other treatments, patients in the first wave received ivermectin, favipiravir, hydroxychloroquine, and remdesivir, while those in the second wave received remdesivir.

Mortality rate was higher during second wave (5.1% vs 3.4%) in our study. Older age, presence of cardiovascular disease and other co-morbidities may be the reasons for increased mortality in second wave. Age > 50 years, severe left ventricular dysfunction, severe and critically ill patients were the independent predictor of mortality in our study. In contrast to our study, mortality rate was lower in other studies (1.2% vs 7.3%)[4], (8.0% vs 23.4%)[10] and (13.2% vs 24.0%)[11]. Lower mortality rate in Japan in the second wave may be because of the shorter time between disease onset and admission, differences in patient background, co-morbidities, and advances in treatment methods[4]. In the first wave older age and the presence of fever, shortness of breath, acute respiratory distress syndrome, diabetes, and cancer were independently associated with higher mortality[11]. On the other hand, in the second wave age, gender, and the presence of acute respiratory distress syndrome and chronic neurological diseases were associated with mortality[11].

There have several limitations to our study. Firstly, study conducted in non-COVID-dedicated hospital. Secondly, the genomic variants were not considered. Thirdly, COVID variants were not determined and fourthly, the brand name of vaccine was not included.

Conclusion:

In comparison to the first wave, during the second wave healthcare personnel were less affected, symptoms were less prominent, asymptomatic and severe disease form were more prevalent & mortality rate was high. Unvaccinated persons were more prone to SARS-CoV-2 infection. Age > 50 years, severe left ventricular dysfunction, severe and critically ill patients were the independent predictor of mortality.

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Original Article

Characteristics and Clinical Outcomes of Patients on Mechanical Ventilation in the Coronary Care Unit of a Tertiary Care Hospital in Bangladesh

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Abstract:

Background: This study was undertaken to evaluate clinical characteristics, indications, outcomes, and factors affecting outcomes in adult patients on mechanical ventilation admitted to CCU that will help planning of proper MV management programs. There are few studies in the coronary care unit (CCU) population and even fewer from developing countries.

Methods: All adult patients received MV at Ibrahim cardiac hospital CCU between June 2019 and July 2020 were prospectively recruited. Different demographic, indications, type and characteristics of ventilation, concomitant complications and treatment, outcomes, clinical and laboratory variables were recorded at the initiation of mechanical ventilation and daily, all throughout the course of MV & thereafter.

Results: Out of 1563 patients admitted to the CCU, 138 patients received IMV. Mean age was 64.2±12.1. Male were predominant (71.7% vs. 28.3%). DM was the most common (81.9%) risk factor. Reasons for intubation were as follows: type 1 respiratory failure (40%), type II respiratory failure (35%), and post cardiac arrest (25%). Mostly used mode of ventilation was A/C VCV (96.4%). Invasive MV was associated with high APACHE II score, low admission PH, Po2, and high Pco2. A higher in-coronary care unit death was observed in MV patients (65.2%) while that for MI (70.3%) than survivors (34.8%). CAG±PCI was (5.8%) keeping on MV or after extubation. The mean duration of MV, stay in CCU and hospital were (53.5±5.8, 80.5±7.6 and 128.8±12.0) hours respectively. The main factors independently associated with increased mortality were (i) pre-MV factors: age, APACHE II scores, acute left ventricular failure, and cardiogenic shock, sepsis (64.2±12.1, 39.1±19.2, 65.9%, 81.2%, and 70%). (ii) Patient management factors during ventilation: without positive end-expiratory pressure (65.6%) (iii) Factors occurring over the course of MV: PaO_2/FiO_2<100 (61.2±18.75) and development of renal failure (47.8%), VAP (40.6%), MODS (21.0%) & ARDS (8.7%) after initiation of MV.

Conclusion: Outcome among mechanically ventilated patients depended on the factors (including patient's demographics, nature of associated morbidity, characteristics of MV received, and conditions developing over the course of MV). These factors may be present before or develop after initiation of MV as well as on the development of complications and the management protocols in the CCU.

Key words: *Invasive Mechanical ventilation, coronary care unit, outcome.*

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Introduction:

The number of patients requiring mechanical ventilator has increased worldwide. Information about the outcome of patients requiring mechanical ventilation is important because it allows for better counseling of patients and their families. It is used quite frequently in the intensive care unit (ICU). It is an essential life support, given to

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many patients in the coronary care unit (CCU) also, where the majority of admitted patients suffering from acute coronary syndrome (ACS) and may develop serious multi-organ complications, requiring invasive mechanical ventilation. The principal indications for invasive mechanical ventilation in CCU are acute respiratory failure due to pulmonary edema as a complications of STEMI or NSTEMI and resuscitated cardio-respiratory arrest.[1,2]

Patients receiving mechanical ventilators require a complex, well-organized, and technically sophisticated level of care, depending on the severity of the respiratory impairment.[3,4,5] Newer modes of the mechanical ventilator are available now a day, each claiming to be better and have found some clinical acceptability. However, data regarding their utilization in real-life ICU situations are not available.[6,7]

In critically ill patients invasive MV and its impact on outcome have been extensively studied.[8-10] While so far few data have been available on the incidence and outcome of patients requiring MV in CCU.[11-14] Data regarding patients admitted to ICU for specific diseases and then needing the mechanical ventilator is available.[15,16] However, data regarding mechanical ventilators in CCU patients, in Bangladesh is still sparse. Previous studies from Bangladesh have investigated the incidence and characteristics of VAP among cardiac patients requiring invasive mechanical ventilation in CCU,[17] but there is no data on the characteristics and clinical outcomes among patients who underwent mechanical ventilation in CCU. We undertook a study to understand the clinical characteristics of patients, indications, mode of mechanical ventilator used and outcomes of patients admitted to the CCU at Ibrahim cardiac hospital & research institute in Bangladesh.

The understanding of the clinical reason for MV, the techniques used to identify the patient, capable of ventilator discontinuation, managing the interaction between weaning and sedation may help minimize both complications and resource consumption during discontinuation of MV.[18-20]

Method:

This prospective observational study was conducted at Ibrahim cardiac hospital & Research Institute, a tertiary care cardiac hospital located in Dhaka, Bangladesh from June 2019 and July 2020. The study included all consecutive patients admitted to the CCU and requiring assisted mechanical ventilation irrespective of indication for intubation. A total of 1563 patients got admitted to CCU during 1 year. Among them, 138 patients were enrolled in the study who received invasive ventilator. This study was approved by the hospital's Ethical Review Committee, and informed consent was obtained from each patient next to the kin. The following information was collected from each patient who received mechanical ventilator: demographic variables, comorbidities, diagnosis on admission, indication for intubation, clinical and biochemical variables were recorded in a predefined case report form. The clinical parameters were recorded from their medical records and bedside charts. ECG, chest-x-ray, arterial blood gas, acute physiology and chronic health evaluation II (APACHE II), mode of ventilation (assist-control ventilation, pressure-controlled ventilation, pressure support ventilation) and ventilator setting at the time of initiation of MV along with hemodynamic parameters, were monitored at that time. Follow-up of CCU course (including change in ventilator setting-VT, respiratory rate, positive end-expiratory pressure PEEP, peak pressure,

Plateau pressure), use of vasoactive, sedative & neuromuscular blockers, and complications arising during the MV (ARDS, barotrauma, ventilator-associated pneumonia, sepsis and multi-organ failure like (cardiovascular, respiratory, renal, hepatic and hematologic) were recorded.

All Patients included in the study were prospectively followed for the duration of MV, length of stay in CCU, and-hospital in hours, and outcomes until hospital discharge.

There were institutional protocols for the initiation of MV or for weaning from MV and they were done as per the clinical judgment of the treating physician in charge and intensivist who considered the patients likely to resume and sustain spontaneous breathing after a patient met standard criteria for weaning readiness [21] & improvement of the cause of respiratory failure, Pao2 to Fio2 ratio above 200 and stable cardiovascular function. We noted the data of weaning method started from spontaneous breathing trial (T-tube circuit, pressure support and ventilation of 7 cm H20, continuous airway pressure of 5 cm H20, other modes).

Statistical analysis:

Data entry and analysis were done using the statistical package for social science (SPSS window version 16, Chicago, USA). Quantitative data (continuous variables) were expressed as mean ± standard deviation or median (interquartile range: IQR). The categorical variable has been depicted as frequency & percentage.

Results

In the 18 bedded CCU, 1563 patients were admitted during the study period from June 2019 to July 2020 and a total of 138 patients received invasive mechanical ventilation for indifferent times. All these patients were studied during their entire period of mechanical ventilation.

Table 1 shows Baseline characteristics among 138 patients where 99(71.7%) were male and 39(28.3%) were female. Mean age was 64.2 ± 12.1 and BMI was 26.6±3.7, also shows the frequently encountered underlying comorbidities of the patients. DM was the most common (81.9%) risk factors followed by HTN, DL & CKD (68.8%, 50.7% & 30.4%). History of MI was 44.2%, PCI and CABG was (24.6% and 18.1%). 5.1% of patients had hypothyroidism. The blood gas analysis at the time of intubation where most of the patients had Type 1 respiratory failure along with metabolic acidosis. Mortality was male: female population (M:F= 66.7%:33.3%), APACHE II score 39.1±19.2 and CAG±PCI 5.8% on MV or after extubation.

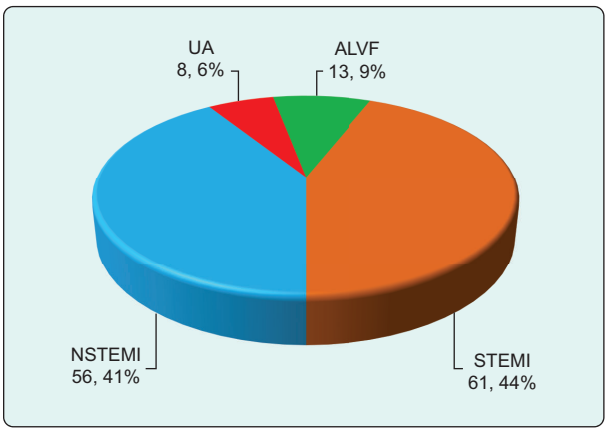
Table-I

Distribution of patients by their demographic characteristics and comorbidities (n=138)

Variables	Survivors	Non-survivors	Total
Age 65.77±10.97	63.49±12.68	64.27±12.12	
Sex			
Male	39(81.2%)	60(66.7%)	99(71.7%)
Female	9 (18.8%)	30(33.3%)	39(28.3%
Total	48(34.8%)	90(65.2%)	13(100%)
BMI	26.55±3.98	26.58±3.6	26.57±3.72
Systolic BP	117.71±34.16	97.33±32.35	104.42±34.2
Diastolic BP	73.54±19.07	60.56±19.10	65.07±20.00
Resp. rate	21.46±7.15	21.64±8.04	21.58±7.71
O_2 Saturation	77.31±14.73	80.90±12.47	79.65±13.36
Hypertension	35(72.9%)	60(66.7%)	95(68.8%)
Diabetes mellitus	41(85.4%)	72(80.0%)	113(81.9%)
Dyslipidemia	32(66.7%)	38(42.81%	70(50.7%)
CKD	18(37.5%)	24(26.7%)	42(30.4%)
Prior MI	23(47.9%)	38(42.2%)	61(44.23%)
Prior CABG	11(22.9%)	14(15.6%)	25(18.1%)
Hypothyroidism	2(4.2%)	5(5.6%)	7(5.7%)
Asthma	6(12.5%)	8(8.9%)	14(10.1%)
COPD	5(10.4%)	2(2.2%)	7(5.1%)
ABG			
pH	7.30±0.13	7.27±0.16	7.28±0.15
PaO_2	76.42±17.01	70.48±17.1	73.20±17.18
$PaCO_2$	44.84±18.36	40.8±19.0	42.22±18.85
HCO_3	20.68±7.27	18.26±6.45	19.10±6.82
Lactate	3.62±2.87	4,94±3.5	4.48±3.36
AG	17.24±10.24	19.2±8.5	18.52±9.18
A-a difference	21.39±19.26	30.8±18.8	27.54±19.42
APACHE Score II	40.23±19.73	38.51±19.0	39.11±19.23
CAG (±PCI) on MV or after extubation			8 (5.8%)

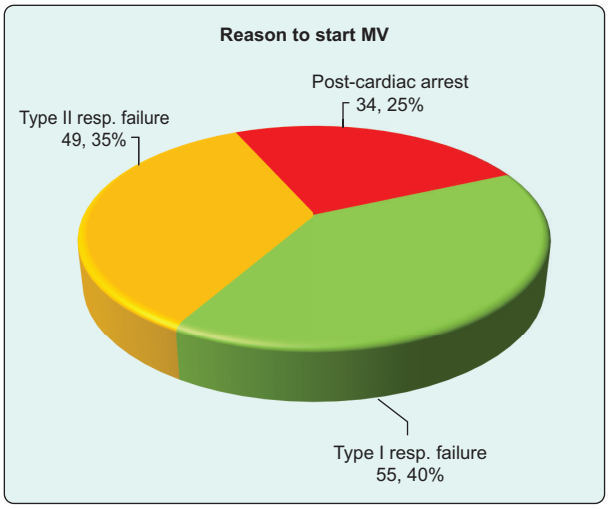
Data were expressed as mean±SD, number (percent) as appropriate.

Pie chart of figure 1 showed the most common reason for CCU admission was STEMI (44%) followed by NSTEMI (41%), ALVF (9%), UA (6%).



Data were expressed as number, percent.

Fig.-1: *Distribution of patients by their Diagnosis on admission (n=138).*



Data were expressed as number (percent)

Fig.-2: *Distribution of patients by their reasons to start MV (n=138)*

Pie chart of Figure 2 shows the most common reason to initiate mechanical ventilation is Type 1 respiratory failure (40%) followed by Type II respiratory failure (35%) and cardiac arrest was (25%).

Table II listed the modes and setting of ventilation according to the indications, investigations and obtaining blood gas analysis for initiation of mechanical ventilation. Most of the patient's mode was A/C VCV (96.4%) without PEEP (50.7%) and with PEEP (49.3%). Peak pressure (28.6±6.1cmH2O) was normal in range and Plateau pressure was (25.6±7.7 cmH2O) which was reduced. PO2/FIO2 ratio was 60.5±18.5 which was also reduced

Table-II

Distribution of patients by their ventilation Mode & Management during Mechanical ventilator (n=138)

Ventilation Mode & management during Mechanical Ventilation	Survivors	Non-survivors	Total
Ventilation mood			
A/C PCV	2(4.2%)	3(3.3%)	5(3.6%)
A/C VCV	46(95.8%)	87(96.7%)	133(96.4%)
Tidal volume (6-mL/kg body weight)	503.33±61.93	506.44±45.43	505.36±51.56
PEEP (cm H_2O)	11(22.9%)37(77.1%)59(65.6%)31(34.4%)		
70(50.7%)68(49.3%)			
Without PEEP			
With PEEP			
Peak pressure (cm H_2O)	27.46±6.29	29.23±5.89	28.62±6.07
Plateau pressure (cm H_2O)	25.25±7.59	25.74±7.81	25.57±7.71
Respiratory rate (Breath/min)	17.0±3.83	17.99±3.21	17.64±3.46
FiO_2	64.54±26.54	88.03±19.16	79.86±24.62
Inspiratory : Expiratory(I:E) ratio	2.0±0.0	2.0±0.0	2.0±0.0
PO_2/FiO_2	59.22±18.34	61.20±18.75	60.53±18.56

Data were expressed as mean±SD, number (percent) as appropriate

The duration of MV until weaning, length of stay in CCU and length of stay in hospital in studied patients are listed in table III. It was mentioned in hours according to the reasons for initiating MV. The times of MV and weaning are exclusive of each other

The patients experienced the following complications over the course of MV showed in figure- 3: cardiogenic shock (81.2%), sepsis (71.0%), ALVF (65.9%), renal failure (47.8%), VAP (40.6%), respiratory failure (25.4%). The development of hepatic failure, nasal bridge ulceration, ARDS, barotrauma & pulmonary embolism were (18.8%, 9.4%, 8.7%, 4.3%, 2.9%)

Factors associated with CCU mortality is shown in figure 4. The presence of MI, renal failure, VAP, MODS, and ARDS (70.3%, 47.8%, 40.6%, 21.0%, and 8.7%) were more common cause of mortality.

Table-III

Distribution of patients by their duration of ventilation (n=138)

Duration of ventilation	Mean ± SEM (Standard error of the mean)	Range
Duration of MV(hours)	53.5 ± 5.8	1-528
Length of stay in CCU (hours)	80.5 ± 7.6	1-528
Length of stay in hospital (hours)	128.8 ± 12.0	1-600

Data were expressed as mean±SD, number (percent) as appropriate

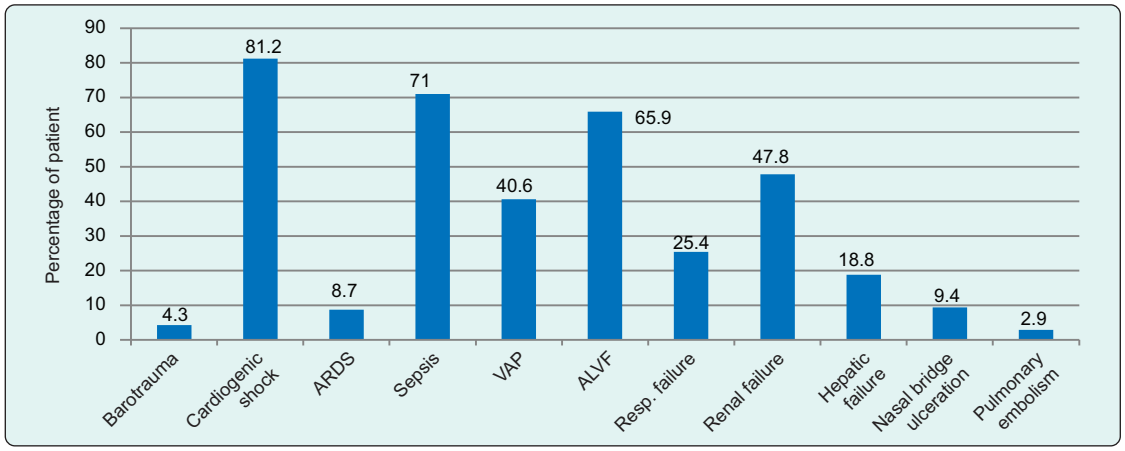


Fig.-3: *Distribution of patients by their complications over the course of MV (n=138)*

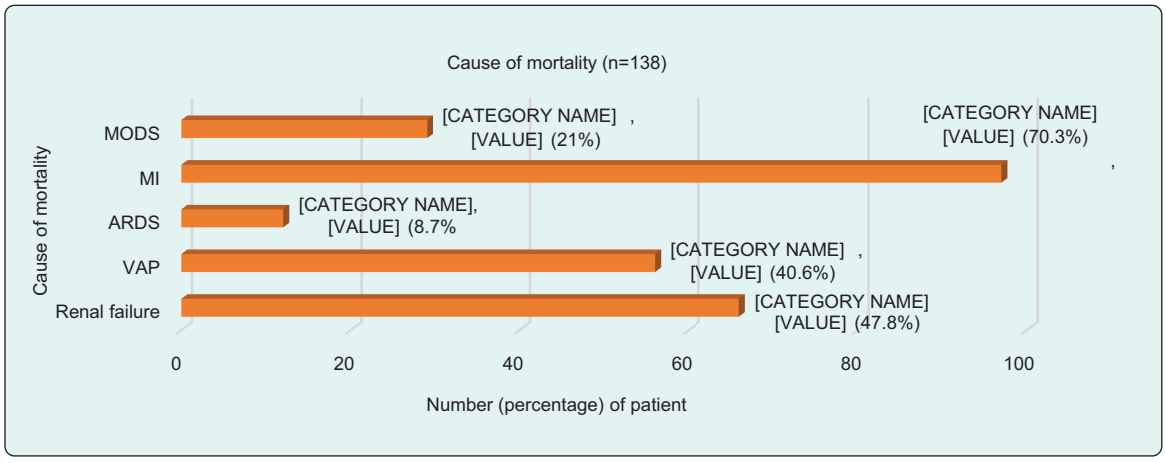


Fig.-4: *Distribution of patients by their causes of mortality (n=138)*

Discussion:

A total of 138 patients were on mechanical ventilation, of which 71.7% were male and 28.3% were female. The need for mechanical ventilation was higher in male than female patients in our study. Mean age was 64.2±12.1 years & majority were overweight as BMI was (26.6±3.7). Behrendt CE, and Esteban A, et al., found in their study that men account for 61% of patients receiving mechanical ventilators in ICU. They explained this by the commonest etiology of respiratory failure due to COPD which is more prevalent in males than females. (6,22,23) In our study COPD was pre-existing among the studied population which was 15.2%.DM is a major risk factor for many diseases in our country, which was 81.9% in our studied population. The probable cause was as the study was done in a diabetes predominant hospitals CCU. We also looked for HTN, DL, prior MI, prior CABG, and CKD, asthma which were (68.8%, 50.7%, 44.2%, 18.1%, 30.4%, and 10.1%) & hypothyroidism was 5.7% in our study. ABG analysis revealed hypoxemia (type-1 respiratory failure) along with metabolic acidosis (78.3%) at the time of intubation. ABG results in PH (7.3) PO2 (73.2) PCO2(42.2), and HCO3 (19.1) matches with the studies done by Confalonieri, Massimo, and Shirakabe, et al., where they showed that lower PH increases the risk of IMV and failure of NIMV by >90%.[24-26] Confalonieri, and Plant, et al., showed high PaCO2 level is predictive of NIMV failure and the need for IMV.[24,27] On the other hand ventatram, et al., showed that there are no differences in the mean PaCO2 value.[28] As regards HCO3, it was lower in IMV patients in our study whereas Madkour, et al., showed a lower level of HCO3 in IMV than in NIMV in their study.[29] Table-I

The APACHE II score was high (39.1) in our study who needed IMV and has higher mortality in patients requiring MV due to acute cardiogenic pulmonary edema. Among ICU patients requiring MV, studies showed that the APACHE II score is independently associated with mortality. [30-32] It is a predictive scoring system based on the factors in the alive group (mean BP, sodium, potassium, creatinine, age, and GCS; giving points based on the APACHE II system, defined as the APACHE II score.[33-37] This score has been reported to be predictive of adverse outcomes in patients requiring intensive care. Outcomes of patients undergoing MV are multifactorial and dependent on those factors that may be present before MV, as well as developed after the onset of MV. Celli, et al., demonstrated that a high APACHE II score proved to predict NIMV failure and the need for IMV.[38] This is because high APACHE II score means more severe acute illness and bad chronic health status of the patients that need IMV rather than NIMV. [39] Table-I

During admission in CCU 44% of the patients had STEMI, 41% NSTEMI, 6% UA and 9% ALVF which causes respiratory failure, cardiogenic shock and cardiac arrest at the time of MV in our study. Figure-1

The most frequent reason to start invasive mechanical ventilation in our study was type 1 respiratory failure (40%) followed by progression to type II respiratory failure (35%) and post cardiac arrest (25%) who was not suitable for non-invasive mechanical ventilation. Figure-2. The common cause of respiratory failure in our study was cardiovascular causes followed by respiratory causes. Pneumonia was the most common respiratory cause and MI-related complications were the common cardiovascular cause of ventilation in our study. Demoule, and Kubler, et al., showed in their study that 60% of mechanically ventilated patients were due to acute on top of chronic respiratory failure and 40% due to post-arrest and coma.[40,41]

In our study the most common initial mode of MV management was A/C VCV (96.4%) to give the traditional tidal volume of 6-8 ml/kg body weight with PEEP (49.3%). 50.7% were without PEEP due to hypotension despite using inotropes (dopamine, dobutamine, or noradrenalin). Kubler, & Ebstain, et al., showed different modes of MV and the relation of PEEP with mortality in patients on MV, especially with ARDS.[41,42] A meta-analysis has shown that high PEEP has a small but significant mortality benefit in ARDS patients and also in unselected groups of MV patients. [43,44] We found the Po2/Fio2 ratio of 60.5 which has a strong correlation with mortality. (When Pao2/Fio2 ratio <100 has increased risk of mortality). Normal Po2/Fio2 is >300. In a study by Esteban, et al (general ICU patients) [45] Sloane, et al [46] Navasrete-Navarro, et al [47] & Kanaus, et al (ARDS patients)[48] showed reduced Pao2/Fio2 ratio has increased risk of mortality. Some studies failed to show any association between Pao2/Fio2 ratio and mortality.[49-51] Table-II

28.6 ± 6.1 patients had peak pressure of less than 35 cmH2O & 25.6 ± 7.7 patients had plateau pressure of more than 35 cmH2o in our study. Vasilyev, et al., reported that a peak inspiratory pressure of more than 35cmH2o was associated with a survival rate of less than 20% while peak inspiratory pressure of less than 30 cmH2o was associated with a survival rate of 60%.[52] Our study revealed an independent association between plateau pressure of more than 35cmH2o and decreased survival

but did not prove that plateau pressure is causally related to the outcome of patients receiving MV. Table-II

The mean duration of MV, length of stay in CCU, and length of stay in hospital were significantly highest in patients who had failed extubation (53.5 hours, 80.5 hours & 128.8 hours) in our study. Table-III

Most common complications throughout IMV in our study were cardiogenic shock 81.2%, sepsis 71% and acute left ventricular failure, renal failure, and VAP, (65.9%,47.8%,40.6%). Figure-3. Mohammad A, et al., showed in their study the highest recorded complication was renal impairment or failure 11.5% followed by ventilation-associated pneumonia 5.77%, and cardiogenic shock 5.77%.[53] In a study done by Esteban, et al. (n= 5183) [42] showed barotrauma 3%, ARDS 22.1%, pneumonia 9.8%, shock 10.6%. The incidence of those complications in our study was (4.3%, 8.7%, 40.6%, 81.2%). Nasal bridge ulceration was (9.4%) which was 3.08% by Mohammad A, et al., [53] Hill, and Holanda, et al., demonstrated in their study that nasal bridge injury is a common problem with NIMV.[54,55] The use of an appropriately sized mask, adjusting head glass and using foam pads and chin shapes can minimize air leaks. [56]

Kubler, et al., showed the commonest etiology of respiratory failure leading to IMV was COPD (14% respectively) followed by ARDS, pneumonia, cardiogenic pulmonary edema.[41] In our study heart failure, pneumonia, post-cardiac arrest, renal failure, ARDS and MODs, arrhythmias were the reason of initiation of MV and increased mortality.

We had 65.2% mortality with weaning failure & which was associated with a high APACHE score. The most common cause was MI (70.3%) followed by renal failure (47.8%). The highest mortality was associated with cardiogenic shock (81.2%) due to MI causing respiratory failure and cardiac arrest in our study. Most of the cases of weaning failure were due to MI-related complications. Figure-4. Therefore, further studies are required to determine whether only cardiovascular disease reduces weaning success in patients requiring MV. Mohammad A, et al., showed in their study that highest mortality was associated with acute hypoxemic respiratory 53.3% followed by post arrest 46.2% and the lowest was associated with acute on top of chronic respiratory failure 6.9%.[53] A study by Esteban, et al., which showed that the only factors independently associated with decreased survival were post arrest, ARDS and sepsis.[42] He also showed mortality rate in ARDS was 60% in their study.[42] In our study mortality due to ARDS was 8.7%, the possible underlying cause was MI. This disparity possibly related to the underlying pathology, as we mostly deal with ACS patients.

Kollef, et al., found mortality to be greater among female patients compared to male patients despite being similarly ill and having similar organ system dysfunction.[56] In our study we found female mortality was 30(33.3%) by the number of patients than the male 60(66.7%) with mean age of 63.49±12.68 years. Luhr OR, and Epstein SK, et al., in their study could not find such a relationship of gender with mortality in patients with respiratory failure undergoing MV.[49,57] Ely, et al., studied 300 mechanical ventilated patients admitted to medical and coronary ICU and found that hospital mortality was 38% among patients older than 75 years [58]

Limitation:

The number of patients included in this study was small and it was a single center study. According to the hospital registry files, the unit serves all patients coming from different regions from Bangladesh but the majority was from Dhaka city. Bangladesh is a vast and diverse country, the slandered of care across the country may vary, and patterns of cases might be different in other parts of the country. We mostly studied the people related to ACS & cardiovascular issues. We could not completely follow up the patients who have transfer to other hospital. Future prospective multi-center study of patients requiring mechanical ventilation are necessary.

Conclusion:

In conclusion, STEMI, NSTEMI & ALVF patients require MV due to either cardiogenic shock or cardiac arrest during admission in CCU. PCI underwent 5.8% of patients followed by CAG while patient was on MV and after extubation. The presence of renal failure, VAP, ARDS, MI, & MODS were the most common cause of weaning failure in our study which causes more death than survivors. Longer weaning duration was associated with increased risk for death. Best weaning protocol was SIMV-PS followed by T-Piece trials. Outcomes of Patients undergoing MV in our CCU are determined by age, APACHE II score on admission, presence of Heart failure, cardiogenic shock, arrhythmias and obesity before initiates Pao2/Fio2 ratio and subsequent additional organ failure. The prospective randomized multicenter trials in Bangladesh, CCU setting will lead additional clarity to these findings. PH <7.35 population that has increase mortality both where more was female than male. Our success rate was half (35%).

Conflict of interest: None

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Original Article

Prediction of Short-Term Outcome after Primary Percutaneous Coronary Intervention by CADILLAC Risk Score

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Abstract:

Introduction: The CADILLAC risk score (CRS) has been developed and validated in the context of primary PCI as a reperfusion strategy for accurate risk stratification. Patients with low CRS have better outcome than those with intermediate to high CRS. However, further studies are needed to validate this score in our population.

Aim of the study: The present study was conducted to predict the short-term outcome after primary percutaneous coronary intervention (pPCI) by CRS.

Method: This prospective observational study was conducted at the National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh from March, 2019 to August, 2020, on 62 patients with two equally divided groups based on CRS: Group I with score 0-2 and Group II with e" 3. The score was calculated by summation of points gathered from each component of the score. Bleeding events, vascular access site complication, heart failure, cardiogenic shock, significant arrhythmia, major adverse cardiovascular and cerebrovascular events (MACCE), were observed during hospital stay and at 30-day follow up.

Result: Mean CRS of the groups were 0.45±0.85 and 4.71±1.74 respectively. Overall adverse outcome, both in-hospital and 30-day, were significantly higher in group II (12.9%vs.35%, p=0.003 and 0vs.22.6%, p=0.001 respectively). Heart failure (22.6%vs.6.5%, p=0.04; 19.4%vs.0, p=0.01) and MACCE (19.3%vs.3.2%, P=0.04; 16.1vs.0%, p=0.02) were significant during hospital stay and at 30-day follow up. Bleeding events (12.9%vs.0, p=0.03) and significant arrhythmia (6.5%vs.0, p=0.04) were significant during hospital stay. Length of hospital stay was also significantly shorter in group I (d"3days: 74.2%vs.35.5%; p= 0.01). The components of CRS except post-PCI TIMI (Thrombolysis in myocardial infarction) flow, intermediate to high CRS, male gender, diabetes mellitus, hypertension, were significant in univariate regression analysis. Moderate to high CRS (in-hospital and 30-day), left ventricular ejection fraction< 40% (in-hospital), triple vessel disease (30-day) were significant in multivariate analysis. ROC curve analysis showed, area under the curve for CRS was 0.745 (95% CI: 0.616-0.874; p=0.001). CRSe"3 predicted in-hospital outcome after pPCI with sensitivity and specificity of 35.5% and 87%, respectively.

Conclusion: In the setting of pPCI, low CRS is associated with better in-hospital outcome in comparison to intermediate to high CRS. Also, in comparison to intermediate to high CRS, low CRS is associated with better 30-day outcome after pPCI, However, for prediction of adverse short-term outcome after pPCI, CRS has got relatively low sensitivity and high specificity.

Key words: *P rimary PCI, CADILLAC risk score, Short term outcome, TIMI risk score, PAMI risk score.*

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Introduction:

The CADILLAC risk score was derived from the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC) trial database which comprises the largest and most comprehensive primary PCI database to date[1]. It consists of seven components: age >65years, Killip class II/III, anemia

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(Hemoglobin [gm/dl]: <13 for male, <12 for female) and renal insufficiency (creatinine clearance rate <60ml/min), triple vessel disease (TVD), ejection fraction (EF) <40%, post-PCI TIMI flow grade II/III. For each component point is 2 except Killip class II/III (3), renal insufficiency (3) and EF (4). Total point is 18. There is 3 strata of this risk score: low (0-2), medium (3-4), high (5-6). These seven clinical and angiographic parameters routinely collected and readily available at baseline or procedural

completion accurately predict 30-day and 1-year mortality rates after primary PCI and these all are integrated in this CADILLAC risk scoring system.[1]It is a simple risk score for predicting mortality and accurate risk stratification after primary PCI. Previous risk scores after reperfusion therapy have incorporated clinical with or without angiographic variables but have not considered baseline left ventricular function. Moreover, prior studies eg. Primary Angioplasty in MI (PAMI) score, Zwolle score have not been validated against independent databases or studies [1]It is found that, beside TIMI (Thrombolysis in Myocardial Infarction) risk score and PAMI (Primary Angioplasty in Myocardial Infarction) risk scores, CADILLAC risk score had high predictive accuracy for 30-day and 1-year mortality as well as for prediction of reinfarction at 30 days with slight superiority of the CADILLAC risk score.[2] It was shown that the CADILLAC risk score is an effective method of patient stratification for early discharge following primary PCI.[3] Predictive value of CADILLAC score was also shown for both primary PCI and rescue PCI.[4] The TIMI, PAMI, CADILLAC and GRACE showed an excellent predictive value for 30-day and 1-year mortality. In AMI patients treated with primary PCI, CADILLAC risk score accurately predict short- and long-term mortality. Of note, measurement of baseline left ventricular function is the single most powerful predictor of survival and should be incorporated into risk score models.

Objectives

a) General objective: To find out the predictive value of CADILLAC risk score for short-term of outcome after primary percutaneous coronary intervention.

b) Specific Objectives:

- To determine the CADILLAC risk score for risk stratification after primary percutaneous coronary intervention.
- To evaluate in–hospital outcome and to observe, estimate the length of stay in hospital after primary percutaneous coronary intervention.
- To observe the 30-day outcome of primary percutaneous coronary intervention PCI, after discharge.
- To find out the relationship between the CADILLAC risk score and the short-term outcome after primary percutaneous coronary intervention.

Methodology & Materials:

This was a Cross sectional, observational study and was carried out in the Department of Cardiology, National Institute of Cardiovascular Disease (NICVD), Dhaka, Bangladesh, over a period of one and half year from march 2017 to august 2018. The sample size was 62 and the study subjects were divided into 2 groups on the basis of CADILLAC risk score: Group I: Low risk group (score: 0 to 2)

Group II: Intermediate to high risk group (score e”3). Statistical analyses were performed using Statistical Package for the Social Sciences software by SPSS Inc., Chicago, IL, USA, version 16.0.

Inclusion criteria:

- STEMI patients undergoing primary PCI, admitted in the Department of Cardiology of NICVD during the study period.
- Age: e”18 years.
- Gender: Both male and female.

Exclusion criteria:

- Cardiogenic shock.
- Failed thrombolysis.
- Requirement of multi-vessel PCI or non-infarct related artery PCI during the index procedure.
- Bleeding diatheses.
- Known hepatic or renal dysfunction.
- Serious co-morbidities with a life expectancy of less than one year

Result:

This cross-sectional study was done in the department of cardiology, National Institute of Cardiovascular Diseases, Sher-E- Bangla Nagar, Dhaka, Bangladesh over a period of time from April, 2019 to August 2020. Sixty-two patients of acute STEMI were selected according to the selection criteria who were undergoing primary PCI. Study subjects were categorized into two groups on the basis of CADILLAC risk score. Group I (Low risk 0-2)

and Group II (Intermediate to high risk e" 3). The main objective of the study was to find out the predictive value of CADILLAC risk score with short- term outcome of acute STEMI patients undergoing Primary PCI. Predefined variables were studied and compared between the groups. Categorical variables were presented as percentages and analyzed using chi square test or Fisher's exact test as appropriate. Continuous variables were expressed as mean and standard deviation (SD) and analyzed using t-test. Variables that were found to be significant in the group analysis and a few variables that were confirmed to be significant in clinical practice were included in the multiple logistic regression analysis. All the statistical tests were 2-tailed, and a p<

0.05 was considered significant. All the analyses were carried out using SPSS statistical software version 16.

Table-I

Distribution of the study groups according to length of hospital stay (N= 62)

Hospital stay (days)	Group I (n = 31)		Group II (n=31)		Total (N=62)		p value
	Number	%	Number	%	Number	%	
≥4 days	8	25.8	20	64.5	28	45.2	
≤3 days	23	74.2	11	35.5	34	54.8	
Mean ± SD	3.1 ± 0.7		3.9 ± 1.0		3.5 ± 1.0		0.01[s]

Table-II

Univariate binary logistic regression analysis of determinants of adverse in-hospital outcome (N= 62)

Variables of interest	Regression coefficient (β)	p value	OR	95% CI of OR
Age>65 years	0.429	0.03[s]	1.53	1.019 – 4.827
Male gender	0.351	0.04[s]	1.37	1.089 – 3.910
Diabetes mellitus	0.612	0.04[s]	2.12	1.117 – 5.248
Hypertension	0.478	0.04[s]	1.48	1.007 – 4.212
Killip class II	1.217	0.03[s]	2.44	1.057 – 3.111
LVEF<40%	1.280	0.02[s]	3.01	1.247 – 5.210
CCR< 60 ml/min	0.701	0.03[s]	1.89	1.091 – 4.201
Hemoglobin	0.357	0.04[s]	1.43	1.044 – 5.394
TVD	1.969	0.03[s]	2.30	1.673 – 4.703
TIMI II flow	0.112	0.07[ns]	1.10	0.084 – 2.412
Intermediate to high CADILLAC risk score	1.312	0.01[s]	3.71	1.030 – 3.381

Table-III

Multivariate binary logistic regression analysis of determinants of adverse in-hospital outcome (N= 62)

Variables of interest	Regression coefficient (β)	p value	OR	95% CI of OR
Age>65 years	0.301	0.49[ns]	1.41	0.261 – 4.211
Male gender	0.041	0.66[ns]	1.01	0.147 – 5.100
Diabetes mellitus	0.410	0.09[ns]	1.99	0.247 – 4.347
Hypertension	0.801	0.29[ns]	2.41	0.520 – 3.312
Killip II	0.010	0.07[ns]	2.22	0.157 – 3.101
LVEF<40%	1.980	0.04[s]	3.55	1.047 – 4.200
CCR<60 ml/min	0.711	0.11[ns]	0.55	0.014 – 4.798
Hemoglobin	0.245	0.69[ns]	1.30	0.301 – 4.201
TVD	0.541	0.07[ns]	2.21	0.243 – 5.103
TIMI II	0.901	0.09[ns]	2.49	0.179 – 3.612
Intermediate to high risk score by CADILLAC	1.201	0.02[s]	3.30	1.020 – 5.281

Table-IV

Univariate binary logistic regression analysis for determinants of adverse 30-day outcome (N= 61)

Variables of interest	Regression coefficient (β)	p value	OR	95% CI of OR
Age>65 years	0.393	0.03[s]	1.51	1.028 – 3.217
Male gender	0.347	0.04[s]	1.35	1.079 – 3.280
Diabetes mellitus	0.600	0.04[s]	2.00	1.110 – 3.212
Hypertension	0.469	0.04[s]	1.42	1.017 – 3.581
Killip II	1.202	0.03[s]	2.29	1.049 – 4.222
LVEF< 40%	1.269	0.02[s]	2.91	1.159 – 5.247
CCR< 60 ml/min	0.692	0.03[s]	1.79	1.081 – 4.274
Anemia	0.344	0.04[s]	1.37	1.039 – 5.384
TVD	1.864	0.03[s]	2.24	1.247 – 5.001
TIMI II	0.109	0.12[ns]	1.07	0.064 – 4.241
Intermediate to high risk score by CADILLAC	1.312	0.01[s]	3.71	1.030 – 5.381

Table-V

Multivariate binary logistic regression analysis for determinants of adverse 30-day outcome (N= 61)

Variables of interest	Regression coefficient (β)	p value	OR	95% CI of OR
Age>65 years	0.379	0.14[ns]	1.51	0.264 – 5.201
Male gender	0.057	0.31[ns]	1.10	0.126 – 3.301
Diabetes mellitus	0.299	0.19[ns]	1.31	0.010 – 3.908
Hypertension	0.280	0.39[ns]	1.21	0.017 – 4.101
Killip II	0.251	0.41[ns]	1.08	0.049 – 4.169
LVEF< 40%	0.407	0.19[ns]	1.61	0.047 – 5.412
CCR< 60 ml/min	0.270	0.41[ns]	1.02	0.110 – 3.345
Anemia	0.152	0.48[ns]	1.12	0.121 – 4.201
TVD	1.110	0.04[s]	3.29	1.034 – 5.044
TIMI II	0.911	0.08[ns]	2.47	0.112 – 5.410
Intermediate to high CRS	1.120	0.02[s]	3.21	1.061 – 6.210

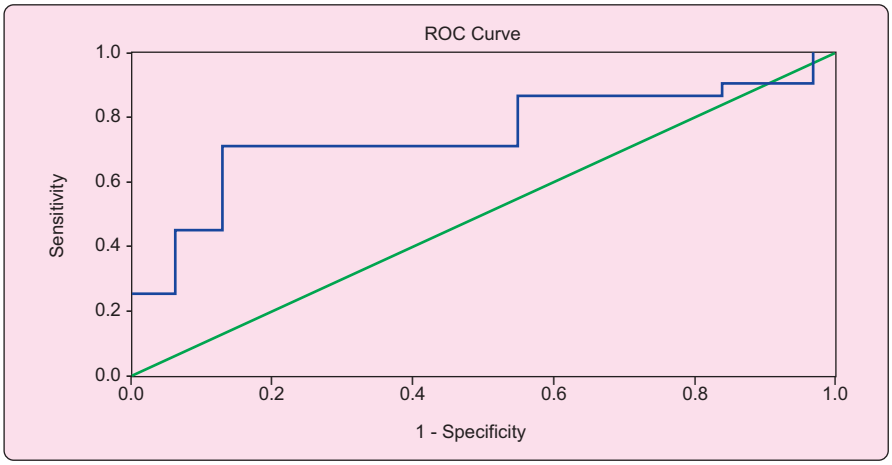

Fig.-1: *Receiver operating characteristic (ROC) curves for intermediate to high CADILLAC risk score (e" 3) for prediction of in-hospital outcome.*

Area Under the Curve

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.745	.066	.001	.616	.874

Discussion:

This prospective observational study was conducted in the Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka on selected acute STEMI patients who underwent primary PCI during their index hospitalization within the time period from march, 2019 to august ,2020. The primary objective of this study was to find out the predictive value of CADILLAC risk score (CRS) for short-term outcome after primary PCI. A total of 62 patients were included in the study. Patients were divided into two groups on the basis of CRS; patients with CRS 0-2 were categorized as group I and those with CRS e"3 were categorized as group II. Among the baseline characteristics of the study groups, age, gender, hypertension, diabetes mellitus, dyslipidemia, family history of premature coronary artery disease, Killip class, baseline left ventricular ejection fraction, hemoglobin level, creatinine clearance rate, triple vessel disease, differed between two groups. The components of the CRS except the post-PCI TIMI flow, male gender, hypertension, diabetes mellitus were found statistically significant. Sharkawi[2] in 2017 found similar result. Another study[1] found all of the components of CRS statistially significant. During hospital stay the observed adverse outcomes, were cardiogenic shock (9.7% vs. 16.1%), heart failure (6.5% vs. 22.6%), bleeding (0 vs. 12.9%), significant arrhythmia (0 vs. 12.9%), and MACCE (Major adverse cardiovascular and cerebrovascular accident) [3.2% vs. 19.3%] Comparison of the study groups revealed heart failure (p=0.04), bleeding events (p= 0.03) significant arrhythmia (p= 0.04) and MACCE (p= 0.04) were statistically significant. 1 patient died in group II (p= 0.31). Comparison of the study groups by overall adverse outcome (OAO) [12.9% vs. 35.5%, p= 0.03] was also statistically significant. Some study[2] found in hospital OAO significant (4 vs. 14%, p= 0.01). Mean length of hospital stay (LOS) in days was 3.5 ± 0 (3.1 ± 0.7 vs. 3.9 ± 1.0). Comparison of the study groups by mean LOS was statistically significant. 74% patients group I could be discharged within 3 days (74.2% vs. 35.5%). In One study[2] found mean LOS (2.99 ±1.0 vs. 3.77 ± 2.245) shorter and statistically significant. In another study[3] did not find mean LOS significant. At 30-day follow up, the observed adverse outcomes were cardiogenic shock (6.5%), heart failure (19.4%), significant arrythmia (6.5%), Bleeding (3.2%), MACCE (16.1%). All of these were observed in group II. Heart failure (p= 0.01) and MACCE (p= 0.02) were statistically significant. Two patients died in group II. Comparison of the study groups by overall adverse outcome (OAO) [0 vs. 12.6%, p= 0.01] was statistically significant. Some study[2] found OAO significant at day-3 or later (0 vs. 12%,

p= .002). On univariate logistic regression analysis, the components of CRS (except final TIMI flow), intermediate to high risk group of CRS, male gender, diabetes and hypertension were found statistically significant. Multivariate analysis showed intermediate to high CRS (both in- hospital and at 30-day follow up), LVEF < 40% (in-hospital) and TVD (at 30-day follow up) were the independent predictors of adverse outcome after primary PCI. The receiver operating characteristic (ROC) curve analysis of CADILLAC risk score, in predicting in-hospital outcome after primary PCI, was done in the present study. The area under the curve (AUC) for intermediate to high CADILLAC risk score (≥3) was 0.745 (95% CI: 0.616 – 0.874, p=0.001) with sensitivity and specificity of 35.5% and 87.1%, respectively. So, the main findings of this study describe that, the group with intermediate to high CADILLAC risk score is associated with adverse short-term outcomes especially bleeding events, significant arrhythmia, heart failure and MACCE in comparison to the group with low CADILLAC risk score.

Limitations of the study

Despite utmost efforts were made during carrying out this study, there were some limitations. The study sample size was relatively small. As purposive sampling was done so there could be a chance of selection bias. Study population was heterogeneous in terms of age, gender, time interval from symptom onset to hospital admission, blood sample collection, vascular access site, number of vessel involvement, vessel stented, and stent size. PCI was performed by multiple operators. So inter-operator variability might have affected the outcome. For identification of significant arrhythmia, patients were observed in CCU for 24 hours only, later it was diagnosed only by ECG on the basis of patient's complains, so some arrhythmias might be missed.

Conclusion and recommendations

In the setting of primary percutaneous coronary intervention, low CADILLAC risk score is associated with better in-hospital outcome in comparison to intermediate to high CADILLAC risk score. Also, In comparison to intermediate to high CADILLAC score, low CADILLAC risk score is associated with better 30-day outcome after primary percutaneous coronary intervention. However, for prediction of adverse short-term outcome after primary percutaneous coronary intervention, CADILLAC risk score has got relatively low sensitivity of 35.5% and high specificity of 87.1%.. Despite lower sensitivity, CADILLAC risk score may be used to predict short-term outcome after primary percutaneous coronary intervention. Larger scale, multi-centric studies should be carried out to validate the findings of the present study and if the result is concordant with future studies, it can be added to the existing armamentarium for prognostication in primary percutaneous coronary intervention.

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Original Article

Relationship between Left Atrial Volume Index and Atrial Fibrillation after Mitral Valve Replacement in Patients with Mitral Valve Disease

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Abstract:

Background: Mitral valve replacement (MVR) is a surgical procedure that is often performed in patients with severe mitral valve disease, to replace the damaged valve with a new artificial valve. Left Atrial Volume Index (LAVI) is a measurement of the size of the left atrium of the heart, normalized to the body surface area which is often used as an indicator of left atrial enlargement, which is a common finding in patients with mitral valve disease. There is a significant relationship between LAVI and Atrial Fibrillation (AF) after MVR in patients with mitral valve disease.

Aim of the study: The aim of this study was to evaluate the relationship between LAVI and the occurrence of atrial fibrillation after mitral valve replacement in patients with mitral valve diseases.

Methods: This prospective observational study was conducted in the department of Cardiac Surgery, National Institute of Cardiovascular Diseases (NICVD), Dhaka from March, 2018 to February, 2019. Total 60 patients were divided into two groups, out of them 30 patients had LAVI e"39 ml/m² (Group-A) and 30 patients had LAVI d" 39ml/m² (Group-B).

Result: In patients with post-operative AF after MVR, was evaluated by ECG in the presence or absence of p-wave and irregular R-R interval and measurement of LAVI more or less than cutoff value 39 ml/m². On postoperative day 3, 7 (23.33%) patients in Group A and 02(6.66%) patients in Group B developed post-operative AF. In Group A there

was reduction in the LAVI but not below the cutoff value whereas in Group B, the LAVI was reduced below the cutoff value (< 39 ml/m2). On overall evaluation, after mitral valve replacement increased LAVI is significantly associated with post-operative AF occurrence and is a better predictor than LA diameters. From univariate analysis in our cohort, high inotropes support, MVT, ACT, CPB time and postoperative LAVI were significantly associated with occurrence of AF. But multiple logistic regression analysis revealed postoperative LAVI to be only significant predictor of occurrence of AF after Mitral valve replacement.

Conclusion: Our study shows that postoperative LAVI measured by 2-D echocardiography is positively and independently associated with the occurrence of post-operative AF following MVR. Moreover, clinical risk factors are fairly good predictors of the occurrence of AF after MVR, but postoperative LAVI was the most significant independent predictor of postoperative AF in our study.

Key words: *Relationship, Left Atrial Volume Index, Atrial Fibrillation, Mitral Valve Replacement and Mitral Valve Disease.*

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Introduction

Incidence of rheumatic heart disease had been reported as 26% of total cardiac disease in Bangladesh.[1] Mitral valve is most commonly affected in rheumatic heart diseases followed by aortic valve in a percentage of 56.7% and 6% respectively in Bangladesh.[2] Isolated mitral stenosis accounts for about 25% of all case and an additional 40% have mixed mitral stenosis and regurgitation.[3] Acquired mitral stenosis usually results from rheumatic heart disease, as does mixed stenosis and regurgitation. It occurs as an isolated valvular condition in 40% of the patient with rheumatic heart disease.[4] Atrial fibrillation (AF) develops sooner or later in most cases of the mitral stenosis. At its onset, the ventricular rate is often more than 140 beats/min and the patient may be rapidly precipitated into acute pulmonary edema. It is an important complication, both because it contributes to the development of cardiac failure and because it is responsible for atrial stasis and the consequent risk of thrombosis and embolism.[5] Atrial fibrillation (AF) is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. AF is thought to be caused by atrial fibrosis and loss of atrial muscle mass. Fibrosis is not limited to the muscle mass of the atria and may occur in the sinoatrial node (SA) node and atrioventricular node (AV node), often leading to sick sinus syndrome (SSS). Prolonged episodes of atrial fibrillation have been shown to correlate with prolongation of the sinus node recovery time, suggesting that dysfunction of the SA node is progressive with prolonged episodes of atrial fibrillation.[6] It can be classified as- (i) Paroxysmal- Continuous AF that stop on its own and lasts <48 hours. (ii) Persistent- Continuous AF that last more than 7 days and requires cardioversion. (iii) Longstanding persistent- Episodic Persistent AF known for >1 year. (iv) Permanent AF episode more than 1-year duration (accepted or therapy failure).[7] The development of paroxysmal and persistent AF is associated with the presence of several factors including age, hypertension, heart failure, mitral valve disease and increased left atrial (LA) dimension as well as LAVI (Left atrial volume index.). Increased LAVI is a major predictor of the both early and late AF. Therefore, in addition to the important prognostic information provided by LAVI and AF occurrence under conservative management in patient with MR, LA size should be integrated into the decision-making process of patients with MR and that surgery may be considered earlier in the course of the disease.[8] After valve replacement surgery, cardiac function gradually improves and the enlarged heart reduces in size, with the cardiothoracic ratio becoming closer to normal. Atrial pressure and left atrial diameter gradually reduce and it may then become possible to convert AF into sinus rhythm.[9] The supero-inferior dimension is measured from the apical 4-chamber view. LAVI e" 39 ml/m² is considered abnormally increased and has a relation for the occurrence of postoperative AF after mitral valve replacement in patient with Mitral valve diseases. The incidence of development of AF is about 18% of patient after successful MVR surgery.[10] The current study was conducted to evaluate the relation of LAVI with occurrence of atrial fibrillation after mitral valve replacement in patient with mitral valve diseases.

Objective

To evaluate the relationship between LAVI and the occurrence of atrial fibrillation after mitral valve replacement in patients with mitral valve diseases.

Methods

This prospective observational study was conducted at the Department of Cardiac Surgery, National institute of cardiovascular diseases (NICVD), Sher-e-Bangla Nagar, Dhaka, Bangladesh. The study duration was 1 year, from Mar, 2018 to Feb, 2019. During this period, a total of 60 patients who underwent Mitral Valve Replacement (MVR) for mitral valve diseases at the study hospital were included in the study following the inclusion and exclusion criteria through a purposive sampling method. The 60 patients were divided in two equal groups, Group A, consisting of 30 Patients who had Left Atrial Volume Index of e" 39 ml/m^2 after operation, while group B had been comprised of 30 patients who had Left Atrial Volume Index of < 39 ml/m^2 after operation. Any patients who had undergone MVR for their mitral valve disease without Atrial Fibrillation (AF) had been included in the study. However, patients with pre-existing AF, patients undergoing concomitant cardiovascular procedures comprising valve surgery (AVR, TVR, PVR) and procedure for congenital heart diseases, patients with electrolyte imbalance and patients with comorbid conditions were excluded. All relevant data were collected from each respondent by use of interview schedule, measured parameters and investigations in a predesigned format. LA volume was measured by planimetry in the four chamber view and two chamber view with the area length method. Informed consent was obtained from the participants prior to their enrollment in the study, and ethical approval for the study was obtained from the ethical review committee of the study hospital. All data were recorded systematically in preformed data collection form (questionnaire). Statistical analyses were performed

by using windows-based computer software devised with Statistical Packages for Social Sciences (SPSS-25). Quantitative data was expressed as mean and standard deviation and qualitative data as frequency distribution and percentage. Continuous variables were compared by using the two independent samples t-test.

Results:

Table I demonstrates demographic characteristics of the study people. Approximately 23.33% of participants in both groups were aged 21-30, while 40% of Group A and 36.66% of Group B were aged 31-40. The majority of participants in both groups were male, with 60% in Group A and 56.66% in Group B, while 40% and 43.33% were female, respectively. In terms of BMI, 53.33% of Group A and 46.66% of Group B had a normal BMI, while 40% and 43.33% were overweight, and 6.66% and 10% were obese, respectively. The mean BMI was slightly higher in Group B than in Group A. Figure 1 shows the NYHA classification of heart failure severity. In our study, 50% of Group A and 36.66% of Group B were in NYHA Class III, while 40% of Group A and 43.33% of Group B were in Class II. Only one participant in Group A was in Class IV, while two participants in Group B were in this class. Table II shows comparison of patients by ECG finding in presence of p-wave and R-R interval (regular). Regarding presence of p-wave and R-R interval (regular) in preoperative period, it was present in both groups. In post-operative period it was present in 25(83.34%) of patients and absent in 5(16.66%) of patient in group B. In Group A presence of p-wave and R-R interval (regular) in post-operative period it was present in 28(93.34%) and absent in 2(6.66%) of patient. The figure 2 shows that the incidence of post-operative AF was significantly higher in Group A than in Group B. Specifically, 23.33% of participants in Group A developed post-operative AF, while only 6.66% of participants in Group B developed it. The p-value for this comparison was 0.015, indicating that this difference was statistically significant. Table III demonstrates that various measurements of left atrial (LA) diameter, LA volume, LA volume index (LAVI), left ventricular internal dimension in diastole (LVIDD), left ventricular internal dimension in systole (LVIDS), and left ventricular ejection fraction (EF) measured pre-operatively and at the third day and first month post-operation. The results demonstrate that Group B had significantly smaller LA diameter, LA volume, LAVI, LVIDS, and LV ejection fraction compared to Group A at all three measurements post-operation ($p<0.05$). However, there was no significant difference between the groups in LVIDD and LV ejection fraction pre-operation ($p>0.05$). Table IV shows that there was no significant difference between Group A and Group B in terms of the need for prolonged ICU stay (>48 hrs.) and prolonged hospital

Table-I

Demographic characteristics of the study people (N=60)

Variables		Group A (n=30) No. (%)	Group B (n=30) No. (%)	p-value
Age (Years)	10-20	1 (3.33%)	2 (6.66%)	0.699^{ns}
	21-30	7 (23.33%)	8 (26.66%)	
	31-40	12(40%)	11(36.66%)	
	41-50	8(26.66%)	6 (20%)	
	51-60	2 (6.66%)	3 (10%)	
	Mean ± SD	39.88 ± 13.51	39.32 ± 11.16	
Gender	Male	18 (60%)	17 (56.66%)	0.382^{ns}
	Female	12 (40%)	13(43.33%)	
BMI (kg/m^2)	Normal (18.5-24.9)	16(53.33%)	14(46.66%)	0.329^{ns}
	Overweight (25.0-29.9)	12(40.0%)	13(43.33%)	
	Obese (>30.0)	2(6.66%)	3(10.0%)	
	Mean ±SD	24.85±2.61	25.73±4.18	

Statistical analysis was done by Chi-square-test
P value ≤0.05 was accepted as significant,
ns = Not significant.
n= Number of subjects in each group.
N= Total number of patients.

stay (>14 days.) as indicated by the p-value of 0.061 and 0.090 respectively. However, there was a significant difference between the two groups in terms of the need for prolonged mechanical ventilation time (>24 hours) and prolonged inotrope support (>48 hrs.) as indicated by the p-values of 0.016 and 0.0006 respectively. Group B had a higher percentage of participants who needed prolonged mechanical ventilation time and inotrope support compared to Group A. Table V shows Multivariable stepwise logistic regression analysis was done to demonstrate that LAVI is independently associated with the occurrence of postoperative AF.

Table-II

Comparison of patients between two groups by ECG finding: presence of p-wave and R-R interval (N=60)

Variables	Group A (n=30) No. (%)		Group B (n=30) No. (%)		p-value
	Presence of p wave	R-R interval (Regular)	Presence of p wave	R-R interval (Regular)	
Pre-operative	30 (100%)	30 (100%)	30 (100%)	30 (100%)	0.023s
Post-operative	23(76.66%)	23(76.66%)	28(93.33%)	28(93.33%)	
% change from preoperative to post-operative	76.66%	76.66%	93.33%	93.33%	

Statistical analysis was done by Chi-square-test
P value d" 0.05 was accepted as significant,
s = Significant.
n= Number of subjects in each group.
N= Total number of patients.

Table-III

Comparison of patients between two groups by Echocardiographic findings (N=60)

Variables		Group A (n=30)	Group B (n =30)	p-value
LA diameter (mm)	Pre-operative	56.56 ± 4.891	47.04 ± 6.093	<0.001^{s}
	Post-operative (3rd day)	51.84 ± 5.121	43.60 ± 5.439	<0.001^{s}
	Post-operative (1month)	49.00 ± 4.690	39.16 ± 4.913	<0.001^{s}
LA Volume(ml)	Pre-operative	60.88 ±8.090	54.20 ±8.684	0.034^{s}
	Post-operative (3rd day)	58.20 ±8.073	51.76±8.136	0.039^{s}
	Post-operative (1month)	54.20±7.963	46.56 ±7.932	0.034^{s}
LAVI (ml/m²)	Pre-operative	48.88 ±7.190	35.21 ±6.884	0.044^{s}
	Post-operative (3rd day)	46.20 ±6.273	33.76±5.146	0.035^{s}
	Post-operative (1month)	42.40±8.664	29.56 ±8.732	0.041^{s}
LVIDD (mm)	Pre-operative	42.86 ±7.090	40.20 ±5.484	0.054^{ns}
	Post-operative (3rd day)	38.20 ±6.073	36.76±8.166	0.059^{ns}
	Post-operative (1month)	35.20±8.963	32.56 ±7.532	0.044^{s}
LVIDS (mm)	Pre-operative	34.96 ± 6.617	30.12±7.155	0.017^{s}
	Post-operative (3rd day)	33.04±6.541	28.32±6.556	0.014^{s}
	Post-operative (1month)	31.40±6.545	26.92±6.164	0.016^{s}
LV Ejection fraction (EF) (%)	Pre-operative	56.00 ± 8.231	60.32 ± 7.674	0.061^{ns}
	Post-operative ((3rd day)	57.48 ±7.241	61.36 ± 6.849	0.057^{ns}
	Post-operative (1month)	58.76 ± 6.815	62.36 ± 6.396	0.060^{ns}

Statistical analysis was done by unpaired Student t-test.
The test of significance was calculated and p values d" 0.05 was accepted as level of significance.
s = Significant
ns = Not significant
n = Number of subjects in each group
N = Total number of patients

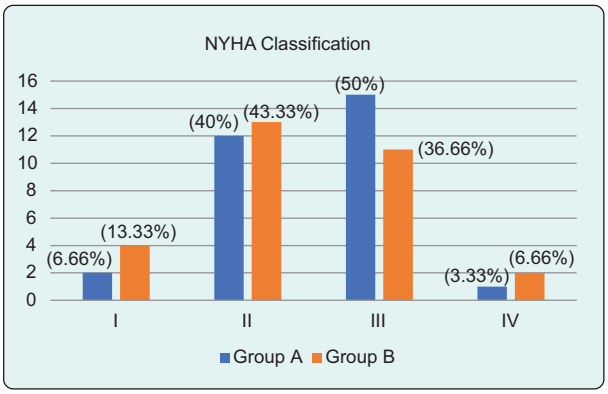

Fig.-1: *Distribution of the patients by NYHA classification between two study groups (N=60)*

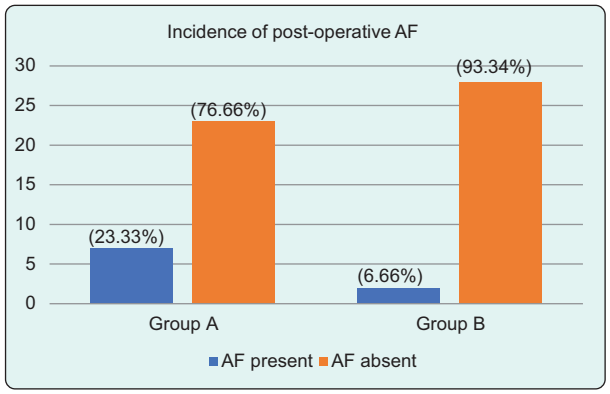

Fig.-2: *Comparison of post-operative AF between two groups (N=60)*

Table-IV

Comparison of Prolonged postoperative care among study groups (N=60)

Post-Operative Variables		Group A (n=30) No. (%)	Group B (n=30) No. (%)	p-value
Prolong ICU stay (>48 hrs.)	Needed	2(6.66.0%)	5(16.66%)	0.061[s]
	Not needed	28(93.34.0%)	25(83.34%)	
Prolong Hospital stay (>14 days)	Needed	6(20.0%)	3(10.0%)	0.090[s]
	Not needed	24(80.0%)	27(90.0%)	
Prolong mechanical ventilation time (>24 hours)	Needed	1(3.33%)	4(13.3%)	0.016[s]
	Not needed	29(96.66%)	26(86.7%)	
Prolong inotrope support (>48 hrs.)	Needed	5(16.66%)	15(50.0%)	0. 0006[s]
	Not needed	25(83.34%)	15(50.0%)	

Fisher Exact was done to analyze the data.
p values d" 0.05 was accepted as level of significance.
s = Significant
ns = Not significant
n = Number of subjects in each group
N = Total number of patients

Table-V

Predictors of post-operative AF according to multiple logistic regression analysis:

Variable(s)	Odds ratio (OR)	95% CI	p value
MVT	1.042	0.967-1.123	0.276[ns]
Inotropes support	1.059	0.920-1.218	0.224[ns]
LAVI	1.098	1.020-1.813	0.001[s]
ACT	0.002	0.0001-12.2	0.166[ns]
CPB	1.92	0.304-11.9	0.148[ns]

Data were expressed as Odds ratio (OR)
S = Significant ($p < 0.05$)
NS = Not significant ($p > 0.05$)

Discussion:

The current study was conducted to evaluate the relation of LAVI with occurrence of atrial fibrillation after mitral valve replacement in patient with mitral valve diseases. In present study, the mean age (years) of the patients in Group A and Group B were 39.88 ±13.51 and 39.32 ± 11.16 years respectively. In Group A, 60% were male and 40 % were female whereas in Group-B, 56.66% were male and 43.33% were female. The mean BMI of group A and group B were 24.85± 2.61 kg/m² and 25.73± 4.18 kg/m² respectively. In our study, the differences in demographic variables (age, sex, BMI) between two groups were not statistically significant (p=0.699, p = 0.382, p = 0.329). These findings are consistent with other similar studies.[11-13] Regarding the NYHA functional class 40% were in NYHA class-II followed by 50% in NYHA class-III in Group-A. In Group-B, 43.33% were in NYHA class-II followed by 36.66% in NYHA class-III. We found no significant association between NYHA and postoperative AF in our study which corresponds to the previous studies.[12] In this study, development of Atrial fibrillation (AF) after MVR was evaluated by the absent of p-wave and irregular R-R interval in ECG. On 3rd POD, postoperative AF was seen in 07 (23.33%) patients in group A and 02 (6.66%) patients in Group B, the difference being significant statistically. Various other prospective studies revealed similar finding claiming postoperative LAVI to be a significant predictor of occurrence of AF.[10] In present study the echocardiographic finding of mean LA diameter was 56.56 ± 4.891 mm in Group A and 47.04 ± 6.093 mm in Group B in preoperative period. After MVR on 3rd post-operative day mean LA diameter was decreased to 51.84 ± 5.121mm in Group A and 43.60 ± 5.439 mm in Group B. After 01 month, mean LA diameter was 49.00 ± 4.690 mm in Group A and 39.16 ± 4.913 mm in Group B. There was significant decrease in LA diameter within 01 month of post-operative period. Petrikovits E et al.[14] performed a prospective study that showed decrease in LA diameter from 56.2 ± 3.42 mm to 50.9 ± 4.40 mm. Another study done by Raine D et al.[13] also showed decrease in LA diameter from 49.2 ± 9.3 mm to 43.2 ± 6.9 mm. In the current study, LA volume was 60.88 ± 8.090 mm in Group A and 54.20 ±8.684 mm in Group B in preoperative period. In 3rd postoperative period it was 58.20 ±8.073 mm in Group A and 51.76 ± 8.136 mm in Group B and during 1st month of post-operative period it was 54.20 ±7.963 mm in Group A and 46.56 ± 7.932 mm in Group B. Raine D et al.[13] also found similar sort of results that revealed reduction from 59.0 ± 6.0 mm to 50.1 ± 8.4 mm. Regarding LAVI, in preoperative period it was 48.88 ± 7.190 ml/m² in Group A and 35.21 ± 6.884 ml/m² in Group B. In 3rd Post-operative period it was 46.20 ± 6.273 ml/m² in Group A and 33.76 ± 5.146 ml/m² in Group B. During 1st month of post-operative period it was 42.40 ±8.664 ml/m² in Group A and 29.56 ±8.732 ml/m² in Group B. The finding was similar to that of Nardi F et al.[15]. In this study, Ejection fraction (EF) in preoperative period was 56.00 ± 8.231%, in 3'rd post-operative day it was 57.48±7.241% and after 01 month 58.76 ±6.815% in Group A. Mean EF in Group B in preoperative period was 60.32 ±7.674%, after 3'rd postoperative day it was 61.36 ± 6.849% and after 01 month it was 62.36 ± 6.396 %. In a study by Osranek M et al.[16], the mean EF was 57.9 ± 12.4% and 59.9 ± 13.4 in two groups, which was close to the findings of our study. Regarding MVT, occurrence of AF was more in patients who required prolonged mechanical ventilation than those who did not. The difference between two groups is statistically significant (p = 0.016). On the other hand, prolonged ICU care needed for more patients in group B than group A, whereas the length of hospital stay was higher in group A than in group B, none of the differences being significant statistically (p =0.061and 0.09 respectively). This finding differs from that of the study carried out by Osranek M et al.[16], they found median length of ICU hospital stay was significantly higher in patients with post-operative AF compared with those without AF (p=<0.0001). The reason may be such that, in our cohort, there were some confounding variables dictating discharge from both ICU and hospital. May be due to these unavoidable factors, length of ICU and hospital stay turned out to be insignificant variable in our study. In this study prolong inotrope support (>48 hours) was needed in 5(16.7%) and 15(50.0%) patients in group A and group B respectively and not needed in 25(83.3%) and 15(50.0%) patients in group A and group B respectively. The difference between the two groups is statistically significant (p = 0.006). The finding was similar to that of Nardi F et al.[15]. Serum electrolytes and serum calcium were done in all patients pre-operatively and postoperatively. Any imbalance or impairment were accordingly managed. But these variables showed no significant effect on postoperative AF. Finally, we performed multivariate logistic regression analysis to find out relation of post-operative AF with others risk factors which revealed that LAVI is an independent risk factor of post-operative AF (OR 1.098; 95% CI; p=<0.001). So, our study showed that left atrial enlargement is associated with post-operative AF and all left atrial echocardiographic parameters were positively associated with postoperative atrial fibrillation. Among the left atrial echocardiographic parameters, left atrial volume index (LAVI) was found to

be more strongly associated with post-operative AF (p=<0.001). Kang MK et al.[10] also found similar result in their study.

Limitations of the study

In our study, there was a small sample size and an absence of a control for comparison. The study population was selected from one center in Dhaka city, so it may not represent the wider population. The study was conducted over a short period of time. There was a lack of reliable methods for recording postoperative arrhythmias. Multiple surgical teams performed the surgery.

Conclusion:

Our study shows that postoperative LAVI measured by 2-D echocardiography is positively and independently associated with the occurrence of post-operative AF following MVR. Moreover, clinical risk factors are fairly good predictors of the occurrence of AF after MVR, but postoperative LAVI was the most significant independent predictor of postoperative AF in our study.

Recommendation:

To maintain sinus rhythm following mitral valve replacement in patients with mitral valve disease, it is wise to add the other surgical procedure to prevent atrial fibrillation along with mitral valve replacement. Measurement of LAVI is a simple and important tool for risk stratification and as a guide for surveillance and therapy in patients with mitral valve disease. A longer period of follow-up would be beneficial to see the exact picture of the association between postoperative LAVI and the occurrence of AF following mitral valve replacement. A multi-center, large-scale study should be carried out.

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Original Article

Association of Left Atrial Volume Index and In-hospital Outcome in Patients with Acute ST Segment Elevation Myocardial Infarction and It's Correlation with the Level of NT-proBNP

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Abstract:

Background: ST segment elevation myocardial infarction (STEMI) is associated with ventricular dysfunction due to ischemic myocardial damage, decrease ventricular compliance and increase filling pressure resulting in left atrial stretching, dilatation, increase left atrial volume and subsequently increase secretion of atrial natriuretic peptides. This study is aimed to determine the association between increase left atrial volume index (LAVI) and in-hospital outcome and to explore the correlation between LAVI and NT-proBNP in patients suffered from acute ST segment elevation myocardial infarction (STEMI).

Methods: This cross sectional analytic study include 92 patients with acute STEMI admitted for reperfusion therapy. 2D Echocardiography was done and based on LAVI, study population were grouped as Group A:LAVI >34 ml/m^2 (n=48) & Group B:LAVI d"34 ml/m2(n=44).

Results: In-hospital outcome, plasma level of NT-proBNP and echcardiographic evaluation was done successfully. Mean NT-proBNP was significantly high in Group A than Group B (1234.6±738.77 vs 689.52±721.04). Statistically significant association was present between LAVI and adverse in-hospital outcome. Persistent chest pain, hypotension, acute LVF, arrhythmia, acute kidney injury were higher in Group A than Group B and acute LVF occurred significantly (p<0.05) more in Group A than Group B (38.3% vs. 9.1%). Statistically significant correlation was present between LAVI and NT-proBNP (r=0.453; p=0.001). According to receiver-operating characteristic curve (ROC) analysis, LAVI with a cut off value of 33.75 ml/m^2 can predict adverse in-hospital outcome in patients of acute STEMI underwent reperfusion therapy with sensitivity 66.2% and specificity 75% and better than NT-proBNP with more sensitivity (66.2% vs 50.0%).

Conclusion: Significant association present between increase LAVI and adverse in-hospital outcome and it can predict adverse in-hospital outcome better than NT-proBNP. There is also positive correlation between LAVI and NT-proBNP in acute STEMI.

Key Words: *STEMI, LAVI, NT-proBNP, In-hospital outcome, 2D Echocardiography, Acute LVF.*

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Introduction:

Many factors have been shown to have prognostic value in STEMI. Demographic variables, symptoms, severity, physical signs, echocardioagraphic and radiological measurements, hemodynamic and neuro-hormonal parameters, high TIMI and Mayo risk score, reduced exercise capacity have been shown to be associated with poor outcome[1]. Various degrees of left ventricular systolic and diastolic dysfunction occur during STEMI[2]. It was demonstrated that left atrium (LA) size has a greater predictive value compared with left ventricular (LV) diastolic function measurements and filling pressures, which are substantially influenced by hemodynamics[3,4]. They concluded that LA enlargement implies a poor prognosis in patients with acute MI. The prognostic usefulness of LA volume persisted after adjustment for clinical predictors of outcome and conventional echocardiographic indices of LV systolic and diastolic function. If confirmed in prospective studies, measurement of LA volume could emerge as a simple and important tool for risk stratification and as a guide for future surveillance and therapy in patients with acute MI. The LA volume has been compared to the "glycated hemoglobin of diabetes mellitus", as it is a reflection of long-standing hemodynamic condition. Estimation of LA volume by Simpson's method of disc is well validated and recommended by the American Society of Echocardiography (ASE) guidelines. LA volume is then indexed to body surface area and called LAVI. The upper normal limit for 2D echocardiographic LA volume is 34 mL/m2 for both sexes[5]. Plasma concentration of NT-proBNP increases after acute myocardial infarction and this increase correlates to the severity of the infarction. Patients with smaller infarcts show an increase NT-proBNP 20 hours after the initiation of symptoms. Patients with larger infracts, lower ejection fraction and more frequent signs of heart failure, reach maximum BNP levels 5 days after admission. In the acute phase, NT-proBNP values do not reflect patient's hemodynamic profile, but four days later BNP levels correlate well with the ejection fraction of the left ventricle and with pulmonary wedge pressure[6,7] . Patients with STEMI and short time of presentation may present with completely normal NT-proBNP, but level increases following reperfusion. NT-proBNP reflects ischemic burden, reperfusion success and prognosis, and the current data support repetitive sampling in patients with ACS[8] .NT-proBNP is an established risk scoring biomarker and it has independent prognostic value in determining adverse cardiovascular outcome like hypotension, left ventricular dysfunction, atrial fibrillation, conduction disturbances, re-infarction and death in patients who have an acute STEMI. By identifying association of increase LAVI with adverse in-hospital outcome and by exploring correlation between LAVI an NT-proBNP in the patients suffering from acute STEMI we can be able to predict averse in – hospital outcome by only doing the LAVI in any low resource setting hospital.

The clinical importance of the proposed study is that measuring left atrial volume index (LAVI) by 2D echocardiography and NT-proBNP in patients who have suffered an acute STEMI could provide valuable correlation among them and able to predict the adverse cardiovascular outcome by only doing the LAVI in any low resource setting hospital. Although the prognostic significance of elevated NT-proBNP is well known, the correlation with LAVI could also guide us to predict adverse cardiovascular outcome and may help explain the adverse outcome associated with LA dilatation in patients at the early stage of acute STEMI.

Methods:

This study was conducted in a single tertiary coronary care center National Heart Foundation Hospital and Research Institute(NHFH&RI), Mirpur, Dhaka from from May, 2017 to April, 2018. Patients with acute STEMI underwent reperfusion therapy were included in the study. 2D Echocardiography was performed, NT-proBNP and adverse in-hospital outcome was recorded very meticulously.

Study population

92 patients of acute STEMI admitted for reperfusion therapy were included in the study. Exclusion criteria were as follows: (a)Chronic atrial fibrillation; (b)Poor Echo window; (c)Patients with atrial infarction (elevated PR segment at leads I, II, III, V5, and V6; 0.15-mV depression at precordial leads or 0.12-mV depression at leads I, II, and III); (d)Patients with Killip class III heart failure or above; (e)Patients with any debilitating illness; (f)Patients with a history of prior MI, percutaneous coronary intervention (PCI), or coronary artery by-pass graft operation; (g)Patients with cardiomyopathy or moderate to severe valvular heart disease. All patients were evaluated and treated according to current guidelines. The regional committee for medical research and ethics approved the research protocol. All participants gave written informed consent.

Investigations:

Patient's baseline 12 lead ECG, daily ECG with time and date including any new event like arrhythmia took at a paper speed of 25mm/sec and 10 mm standardization. Baseline investigations like blood sugar, HbA_1C, serum creatinine, fasting lipid profile, troponin-I, CK-MB, serum electrolytes done accordingly. Heparinized plasma was sent before the echocardiographic evaluation of the patient to measure the plasma level of NT-proBNP by radioimmunoassay. NT-proBNP value 100-900 pg/ml was considered normal and > 900 pg/ml was considered as higher value according to the reference range of the test kit and Getein1100 Immunofluorescence Quantitative Analyzer.

Echocardiography:

ECG guided transthoracic echocardiography was performed by using Vivid E9 made by GE Healthcare with a 2.5 MHZ phased array transducer by an experienced echocardiologist after reperfusion therapy. LA volume obtained from apical four chamber and apical two chamber view in end-systole before mitral valve opening by using the modified Simpson method. The American Society of Echocardiography(ASE) Guidelines recommended the biplane disk summation technique, which incorporates fewer geometric assumptions and the preferred method for measurement of LA volume. The upper normal limit for 2D echocardiographic LA volume is 34 mL/m2 for both genders. It was calculated using the disk summation technique by adding the volume of a stack of cylinders of height (h) and area calculated by orthogonal minor and major transverse axes (D1 and D2) assuming an oval shape[5].

$$\text{LA Volume} = \pi/4(h)\ \Sigma\ (D1)\ (D2)$$

LA volume is then indexed to body surface area as recommended by the American Society of Echocardiography. The upper normal limit for 2D echocardiographic LA volume is 34 mL/m2 for both genders[5]. Left ventricular ejection fraction (EF) was measured by using Simpson's biplane method.

Statistical Analysis:

Structured case record from was prepared for data collection which include particulars of the patients, baseline clinical variables, baseline heart failure status, distribution of risk factors, biochemical variables, echocardiographic variables, treatment strategies, in-hospital outcome. Comparison between groups were done by Student's T-test for continuous variables and categorical data were analyzed by chi-square test. The level of significance was set <0.05 (p value). Association of LAVI and adverse in-hospital outcome association of NT-proBNP level with adverse in-hospital outcome and correlation analysis between LAVI and NT-proBNP was performed and shown in different tables and figures. Correlation analysis was performed to assess the relationships between the LAVI and the NT-proBNP level. ROC curve of LAVI and NT-proBNP was drawn for prediction of adverse in-hospital outcome in the study population. Statistical analysis was performed using SPSS Version 22.0 software (SPSS Inc., Chicago, Illinois, USA).

Result:

A total of 92 consecutive patients were enrolled and divided into two groups on the basis of LAVI. In group A: LAVI >34 ml/m^2, 48 patients and in group B: LAVI d"34ml/m2, 44 patients were assigned. Baseline data of all patients (n = 92) given in Table 1. It was observed that mean age was 55.15±11.93 years in Group A and 52.18±11.26 years in Group B. Higher study populations (38.8%) in Group B was in the 4^{th} decade and in group A was (35.4%) in the 5^{th} decade and age difference was statistically not significant between two groups. Male patients were 41(85.4%) and 38(86.4%) in Group A and in Group B respectively. Female patients were 7(14.6%) and 6(13.6%) in Group-A and in Group B and the differences were not statistically significant. According to Killip classification of heart failure, statistically no significant difference was present between two groups. All the risk factors except family history of IHD were more in Group B (41.7%, 41.7%, 43.7%, 52.1% and 41.7% vs 43.2%, 54.5%, 46.5% and 54.5%). Family history of IHD were more in Group A than Group B (41.7% vs 27.3%). The differences between two groups were not statistically significant. Table 2 demonstrates baseline clinical variables in the study population. It was observed that acute left ventricular failure occurred more in Group A (22.9%) than Group B (18.2%). Atrial fibrillation occurred more in Group B but PVC occurred more in Group A. VT and VF occurred equally in both the groups . STEMI (extensive anterior) occurred more in group A than Group B (33.2% vs 22.7%). All the above clinical variables were not statistically significant between two groups.

We analyzed 2D echocardiographic variables in the study populations shown in Table 4, and we observed that LA diameter and LVEF were not statistically significant between the two groups but the mean LAVI in Group A is significantly higher than Group B (43.59±7.62 vs 25.18±5.74).

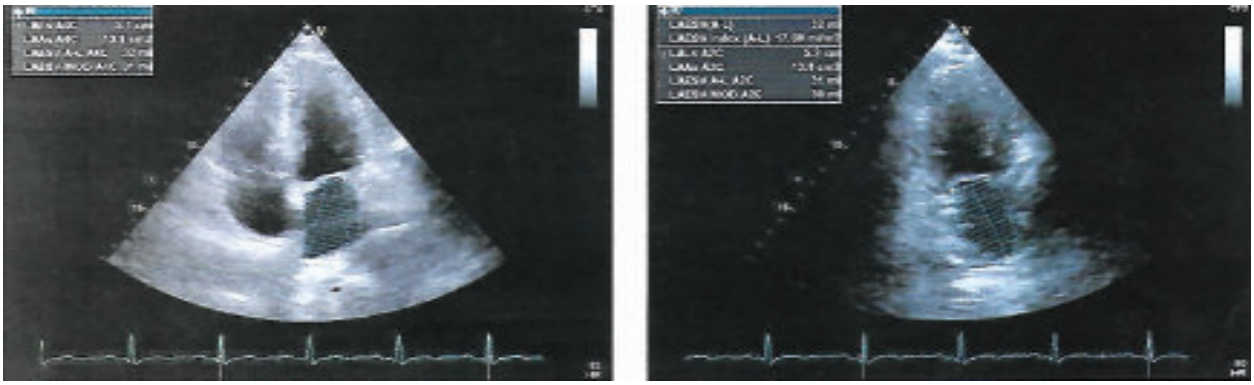

Fig.-1: *Simpson's biplane method of disks (Echolab, NHFH&RI, 2017).*

Table-I

Clinical characteristics in the pooled cohort consisting of all 92 patients

Particulars of the patients	Group-A (LAVI >34 ml/m^2) (n=48)	Group-B (LAVI ≤34 ml/m^2) (n=44)	*p* value
Mean Age ±SD(p value reached from Student's t Test)	55.15±11.93	52.18±11.26	0.224^{ns}
Sex(%)(p value reached from Chi-square(x2) test)			
Male	85.4	86.4	0.896^{ns}
Female	14.6	13.6	0.896^{ns}
Heart Failure Status(%)(p value reached from Chi-square(x2) test)			
Killip Class I	81.3	86.4	0.569^{ns}
Killip Class II	18.7	13.6	0.569^{ns}
Risk factors(%)(p value reached from Chi-square(x2) test)			
Diabetes	41.7	43.2	0.883^{ns}
Hypertension	41.7	54.5	0.217^{ns}
Dyslipidemia	43.7	46.5	0.792^{ns}
Smoking	31.3	34.1	1.000^{ns}
Family history of IHD	41.7	27.3	0.148^{ns}

ns=non significant, s=significant at p<0.05.

Table-II

Baseline clinical variables in the study population (n=92)

Clinical Information On Admission	Group-A (LAVI >34 ml/m^2) (n=48)	Group-B (LAVI ≤34 ml/m^2) (n=44)	*p* value
Acute LVF on Presentation(%)	22.9	18.2	^{b}0.575^{ns}
Arrhythmia(%)			
Atrial Fibrillation	12.5	40	0.157^{ns}
PVC	50	20	0.093^{ns}
VT	12.5	20	0.793^{ns}
VF	12.5	20	0.793^{ns}
Location of STEMI(%)			
Anteroseptal	4.2	15.9	0.058^{ns}
Anterior	18.8	27.3	0.259^{ns}
Extensive anterior	33.2	22.7	0.259^{ns}
Lateral	0.0	2.3	0.293^{ns}
Inferior	20.7	15.8	0.445^{ns}
STEMI Equivalent	2.1	4.5	0.507^{ns}
Inferior + Posterior	4.2	2.3	0.697^{ns}
Inferior + Posterior +RVI	6.3	2.3	0.374^{ns}
Inferior+ RVI	6.3	2.3	0.374^{ns}
Anterior + Inferior	2.1	2.3	1.000^{ns}
Inferior +Posterior + Lateral	2.1	2.3	1.000^{ns}

s= significant, ns= not significant , ^{a}p value reached from unpaired t test, ^{b}p value reached from Chi square test.

Table-III

Biochemical variable in the study population(n=92)

Biochemical variable	Group-A (LAVI >34 ml/m^2) (n=48) Mean±SD	Group-B (LAVI ≤34 ml/m^2) (n=44) Mean±SD	*p* value
NT- ProBNP	1234.6±738.77	689.52±721.04	0.001^s

s= significant, ns= not significant , p value reached from Student's t Test.

Table III shows mean NT-proBNP value was more in Group A than Group B(1234.6±738.77 vs 689.52±721.04) and the differences between the two groups were statistically significant(*p* value=0.001).

Table-IV

Echocardiographic variables in the study population (n=92)

Echocardiography 2D	Group-A (LAVI >34 ml/m^2) (n=48)	Group-B (LAVI ≤34 ml/m^2) (n=44)	*p* value
LA Diameter (%)			
Normal (≤40mm)	91.7	97.7	b0.201ns
Increased (>40mm)			
LAVI (Mean±SD)	43.59 ±7.62	25.18 ±5.74	a0.001s
LVEF (Mean±SD)	41.73 ±6.7	43.3 ±6.16	0.093^{ns}

Regarding different treatment strategies(Thrombolysis by streptokinase=STK, Primary PCI= PPCI or Pharmacoinvasive) as shown in Table V, it was observed that the difference in number of patients between two groups were not statistically significant.

Table-V

Treatment strategies in the study population(n=92)

Treatment	Group-A (LAVI >34 ml/m^2) (n=48)	Group-B (LAVI ≤34 ml/m^2) (n=44)	*p* value
Streptokinase(%)	52.1	47.7	0.979^{ns}
Primary PCI(%)	45.8	50.0	0.950^{ns}
Pharmacoinvasive(%)	2.1	2.3	0.950^{ns}

Table-VI

In-hospital outcome in the study population (n=92).

In Hospital outcome	Group-A (LAVI >34 ml/m^2) (n=48)	Group-B (LAVI ≤34 ml/m^2) (n=44)	*p* value
Death	0.0	0.0	
Reinfarction	0.0	0.0	
Persistent Chest Pain			
Yes	16.7	6.8	0.146^{ns}
No	83.3	93.2	0.146^{ns}
Hypotension			
Yes	50.0	31.8	0.077^{ns}
No	50.0	68.2	0.146^{ns}
Cardiogenic Shock	0.0	0.0	
Acute LVF			
Yes	38.31	9.1	0.001^{s}
No	61.7	90.9	0.001^{s}
Arrhythmia			
AF	2.1	2.3	0.985^{ns}
VT	4.2	2.3	0.609^{ns}
Others	6.2	13.6	0.234^{ns}
No	87.5	81.8	0.448^{ns}
Acute Kidney injury			
Yes	33.3	15.9	0.054^{ns}
No	66.7	84.1	0.054^{ns}
Hospital Stay Period (Days)			
≤5	66.7	86.4	0.469^{ns}
6-10	27.1	9.1	0.240^{ns}
>10	6.3	4.5	0.540^{ns}
Mean±SD	5.0±2.2	4.4±2.4	^{a}0.214^{ns}

s= significant, ns= not significant, *p* value reached from Chi-square(x^2) test.

As shown in Table VI in-hospital outcome in the study population and there was no death, reinfarction and cardiogenic shock in the study population. The mean stay period was 5 day in Group-A and 4.4 day in Group-B. Persistent chest pain, hypotension, acute LVF, arrhythmia and acute kidney injury occurred more in Group A than Group B. Total 72 adverse outcome occurred in Group A and 36 in Group B. Acute LVF was significantly higher in Group A than Group B (38.3% vs 9.1%).

Table-VII

In-hospital outcome of different treatment strategies in the study population(n=92).

Outcome	Treatment			*p* value
	STK %	PPCI %	STREPTOKINASE	
Adverse Outcome	78.3	70.5	100.0	0.416^{ns}
Uneventful	21.7	31.8	0.0	0.416^{ns}

According to Table 7 in hospital outcome of different treatment stratigies, 78.3% patients in the STK Group,70.5% patients in PPCI group and 100% patients in the Pharmacoinvasive Group had adverse outcome.

Table-VIII

In-hospital outcome according to LAVI in the study population(n=92).

Outcome	LAVI				*p* value
	High (LAVI >34 ml/m^2) Group A		Normal (LAVI ≤34 ml/m^2) Group B		
	n	%	n	%	
Adverse Outcome	42	87.5	26	59.1	0.002^{ns}
Uneventful	6	12.5	18	40.9	0.002^{ns}

As shown in Table 8 According to LAVI, in-hospital outcome in the study population showed that more patients had adverse outcome in high LAVI Groups than normal LAVI (87.5% vs 59.1%, p=0.002). The differences was statistically significant between two groups.

Table-IX

In-hospital outcome according to NT-proBNP level in the study population (n=92).

Outcome	NT-proBNP				*p* value
	High (>900 pg/ml)		Normal(100-900 pg/ml)		
	n	%	n	%	
Adverse Outcome	35	89.7	33	62.3	0.003^{ns}
Uneventful	4	10.3	20	37.7	0.003^{ns}

Table IX demonstrates that adverse outcome occurred significantly more in high NT-proBNP groups than normal NT-proBNP (89.7% vs 62.3%).

Table-X

Association between LAVI and NT-ProBNP in the study population (n=92)

LAVI	NT-proBNP				*p* value
	High (>900 pg/ml)		Normal (100-900 pg/ml)		
	n	%	n	%	
High (Group A)	30	76.9	18	34.0	
Normal (Group B)	9	23.1	35	66.0	

s=significant

Measures of agreement, Kappa Value 0.422, p value 0.001[s]
Kappa value indicates moderate agreement

Agreement = 70.7% Kappa	Interpretation
< 0	Poor agreement
0.0 – 0.20	Slight agreement
0.21 – 0.40	Fair agreement
0.41 – 0.60	Moderate agreement
0.61 – 0.80	Substantial agreement
0.81 – 1.00	Almost perfect agreement

The results of the two modalities, LAVI and NT-proBNP, found Kappa value=0.422 with p value <0.05. This measure of agreement is statistically significant with moderate agreement between LAVI and NT-proBNP. Among 39 high value of NT-proBNP patients, 30(76.92%) patients have high LAVI and 9(23.07%) patients have Normal LAVI. Among total 53 normal NT-proBNP value patients 18(33.96%) patients have high LAVI & 35 (60.03%) have normal value.

In Figure 2, LAVI value was expressed in the study population as ml/m2 and NT-proBNP value as picogram/ml. The value of Pearson's rank correlation coefficient was 0.453 and p value was 0.001. So it can be concluded that, there is a positive significant (p<0.05) correlation between NT-proBNP and LAVI in the study population.

Receiver- operating characteristic curve (ROC) of LAVI and NT-ProBNP for prediction of adverse in hospital outcome in the study population, as shown in figure 3, LAVI had the best area under the curve. ROC curve was constructed as LAVI had area under the curve 0.729, showing cut off value of 33.75 with 66.2% sensitivity and 75.5% specificity and NT-ProBNP had area under the curve 0.631 showing cut off value 900 with 50.0% sensitivity and 83.3% specificity for prediction of adverse in-hospital outcome. ROC analysis showed that LAVI can predict adverse in-hospital outcome better than NT-proBNP (66.2% vs 50.0%).

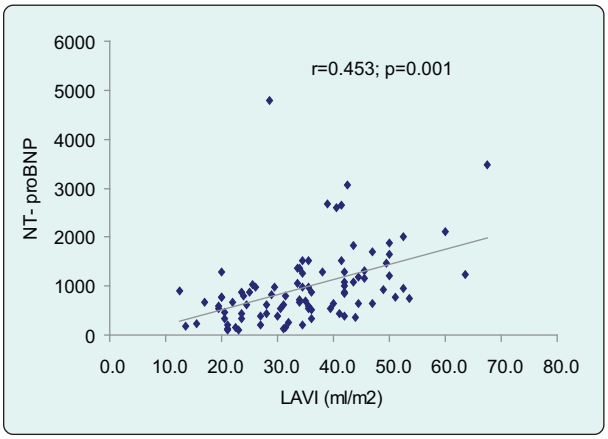

Fig.-2: *Scatter diagram showing positive significant correlation (r=0.453; p=0.001) between LAVI (ml/m2) and NT-proBNP.*

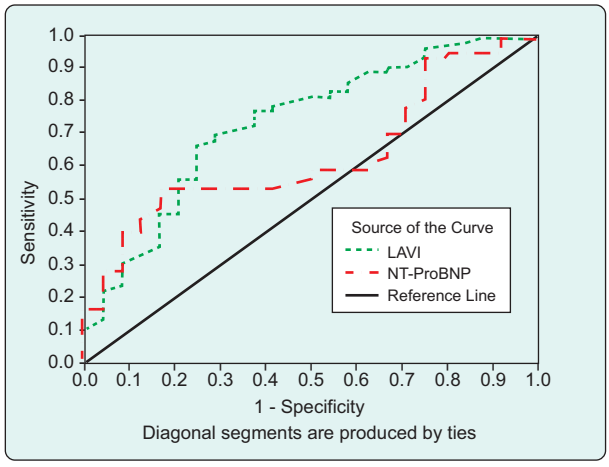

Fig.-3: *Receiver- operating characteristic (ROC) curve of LAVI and NT-ProBNP for prediction of adverse in hospital outcome in the study population.*

Table-XI

Receiver operating characteristic (ROC) curve of LAVI and NT-ProBNP to predict adverse in-hospital outcome in the study population

	Cut of value	Sensitivity	Specificity	Area under the ROC curve	P value	95% Confidence interval (CI)	
						Lower bound	Upper bound
LAVI	33.75	66.2	75.0	0.729	0.001[s]	0.611	0.848
NT- ProBNP	900.0	50.0	83.3	0.631	0.057[ns]	0.511	0.751

Discussion:

To the best of our knowledge, this is the first study in Bangladesh showing association between LAVI and adverse in-hospital outcome and also positive correlation between LAVI and the level of NT-proBNP in patients of acute STEMI. This study showed that increased LAVI can predict adverse in hospital outcome with good sensitivity compare to NT-proBNP level (6.2% vs 50.0%).

It was observed that 35.4% patients belonged to age 51-60 years in Group A and 27.3% in Group B. The mean age was 55.15±11.93 years in group A and 52.18±11.26 years in Group B. The difference was statistically not significant between two groups. It was observed that the mean age of patients was 66±11 years in LAVI <40 ml/m2 group and was 69±11 years in LAVI> 40m1/m2 group[9] and the mean age was 61±10 years in higher LAVI group[10]. Similarly, it was observed that mean age was 67.9± 10.4 years in LAVI < 32ml/m2 Group and mean age 70.2±9.7 years in LAVI e" 32ml/m2 patients group[11]. It was also observed that the mean age of LAVI < 32ml/m2 patients group was 65 years with range from 53-75 years and mean age in LAVIe"32m1/m2 patients group was 76 years with range from 67-82 years[12]. The higher mean age and age range obtained by the above authors may be due to geographical variations, racial, ethnic differences and genetic causes that have significant influence on coronary artery disease in their study subjects.

In this study, it was observed that 85.4% patients were male in Group A and 86.4% in group B. The difference was statistically not significant between two groups. Gender differences in LA Volume index does not occur as per reviewed literatures[10,13] .

In this series, it was observed that 22.9% patients had acute LVF on presentation in Group A and 18.2% in Group B, 16.7% arrhythmia in Group A and 9.1% in Group B, 50.0% PVC in Group A and 20.0% in Group B, 33.2% patients suffered extensive anterior MI in Group A and 22.7% in Group B. All the baseline clinical variables were statistically not significant between two groups and there is no differences in location of MI in the study population[14]. Similarly in this study, though extensive anterior MI was more common, but no statistically significant difference were present between the two groups.

In this study, it was observed that 81.3% patients was in Killip Class 1 status in group A and 86.4% in group B. The difference was statistically not significant between two groups. It was also showed no baseline differences in Killip and NYHA class of heart failure among the study groups[14].

In this series, it was observed that 41.7% patients had diabetes in group A and 43.2% in group B, 41.7% hypertensive patients in Group A and 54.5% in Group B, 43.7% dyslipidemia in Group A and 46.5% in Group B, 31.3% were smoker in Group A and 34.1% in Group B, 41.7% had family history of IHD in Group A and 27.3% in Group B. The difference was statistically not significant between two groups. It was showed that LAVI correlated positively with hypertension, diabetes mellitus, dyslipidemia and smoking[15]. These risk factors in the study population was consistent with those found in other studies[16,17]. However different studies carried out in different countries demonstrated different patterns and this may be due to ethnic and cultural differences in the study population.

In this study, it was found that LA diameter was Increased (>40mm) in 8.3% patients in Group A and 2.3% in Group B. The mean LAVI was 43.59±7.62 ml/m2 in group A and 25.18±5.74 ml/m2 in Group B. Mean LVEF was 41.73±6.7 % in Group A and 43.68±6.16 % in Group B. The difference of LAVI was statistically significant between two groups. In one study it was showed significant differences between the two groups[14]. Increased LAVI is consistent with chronic elevation of LV filling pressure and may be an indicator of increased cardiovascular risk [15]. LAVI has been showed to be highly predictive of cardiovascular risks including arrhythmias, atrial fibrillation, left ventricular failure, stroke and death after acute myocardial infarction[4,12,15].The most widely used method for LA diameter measurement is to acquire its anteroposterior (AP) diameter in the parasternal long-axis view but the fact is that linear dimension doesn't correlate with the actual LA size and this should not be used as the sole measurement for LA[18,19].

In this series, it was observed that 52.1% patients got STK in Group A and 47.7% in Group B, 45.8% underwent Primary PCI in group A and 50.0% in Group B, 2.1% underwent pharmacoinvasive therapy in Group A and 2.3% in Group B. The differences were statistically not significant between two groups. It was found that, 78.3% patient had adverse outcome in STK group and 70.5 patients in PPCI group.

In this study, it was observed that there was no in-hospital death, reinfarction and cardiogenic shock occurred in the study population. Mean hospital stay period was 5 days in higher LAVI Group and 4.4 days in normal LAVI Group. 16.7% patients had persistent chest pain in Group

A and 6.8% in Group B, 50.0% hypotension in Group A and 31.8% in Group B, 38.3% acute LVF in Group A and 9.1% in Group B, 4.2% VT in group A and 2.3% in group B, 33.3% acute kidney injury was in Group A and 15.9% in Group B. 72 adverse outcome occurred in Group A and 36 in Group B, ie, more adverse outcome occurred in higher LAVI group. In the present study acute LVF was significantly more in Group A with increased LAVI. Other study also showed that LAVI was associated with systolic and diastolic dysfunction which may result let ventricular failure. One study found that LA volume correlate positively with the grade of diastolic dysfunction, and negatively with LV systolic dysfunction[20]. One study proved that there is a positive correlation with the wall motion score index (WMSI) and LA volume index >32 ml/m2[12] . Another study suggested that when LV dysfunction is present with increased stiffness or non-compliance, LA pressure rises to maintain adequate LV filling and the increased atrial wall tension leads to chamber dilatation and stretch of the atrial myocardium[21]. In this study we also found LV dysfunction in higher LAVI group. One study suggested that LA size is increased and LA emptying decreased in patients with either systolic or diastolic heart failure and it is associated with the new development of LV failure[22].

It was observed that, more patients (87.5%) had adverse in-hospital outcome in Group A with higher LAVI than Group B (59.1%) with normal LAVI. Similarly, more patients (89.7%) had adverse outcome in higher NT-proBNP group compare to normal NT-proBNP group (62.3%). It was demonstrated that, LAVI was a predictor of mortality after AMI, even after adjustment for conventional indices of systolic and diastolic function and concluded that LA volume could emerge as a simple and important tool for risk stratification and as a guide for future surveillance and therapy in patients with AMI[12]. One research showed that atrial natriuretic peptides are useful for guidance of therapy and risk stratification of patients in heart failure and AMI[23]. It was found that plasma NT-proBNP level is a sensitive indicator of cardiac dysfunction, both in the presence and absence of systolic dysfunction, and an useful tool for identiûcation and management[24.] showed that in comparison to other biochemical markers, NT-proBNP level was the most powerful indicator of major adverse cardiovascular events (MACE) and a single measurement of the NT-proBNP level appears to be useful as a prognostic factor in the prediction of MACES in patients after ACS[25 .]

In this present study, it was observed that, 39 patients had higher NT-proBNP level and among them 30(76.9%) patients had higher LAVI and 9(23.1%) belonged to normal LAVI. Normal NT-proBNP level was found in 53 cases among them 18(34.0%) had higher LAVI and 35(66.0%) belonged to normal LAVI. Measures of agreement was 70.7% with Kappa Value 0.422, thus indicates moderate agreement between LAVI and NT-proBNP for prediction of adverse outcome in acute STEMI patients. . In a recent study, it was found that in the setting of acute coronary syndrome, NT-proBNP is an extremely powerful prognostic indicator including patients with STEMI at higher risk[26]. It was showed that NT-proBNP concentration are higher in patients with more severe symptoms and in those with more severe cardiac damage[27]. It was found that there is a strong association between high plasma NT-proBNP and incomplete ST-resolution in patients after reperfusion, and it highlights the potential relevance of NT-proBNP for early risk stratification in the setting of reperfusion therapy after acute myocardial infarction[28]. A study in NICVD showed that raised plasma BNP concentration strongly predict short-term mortality and morbidity in subjects with acute heart failure in STEMI[29].

LAVI value was expressed in the study population as ml/m2 and NT-proBNP value as picogram/ml. The value of Pearson's rank correlation coefficient was 0.453 and p value was 0.001. Thus there was a positive significant correlation between NT-proBNP and LAVI in the study population.

According to receiver-operating characteristic curve it was observed in the present study that LAVI had best area under the curve 0.729 with a cut off value of 33.75 having 66.2% sensitivity and 75.0% specificity for predicting in-hospital adverse outcome. However NT-ProBNP had area under the curve 0.631 with cut off value 900 having 50.0% sensitivity and 83.3% specificity, which indicates that LAVI is better predictor for prediction of adverse in hospital outcome in the study population compared to NT-ProBNP. A study showed that indexed LA volume e"28 ml/m2 was 82.0% sensitive and 93.0% specific and indexed LA volume e"27 ml/m2 was 89.0% sensitive and 86.0% specific for the detection of abnormal diastolic function[15]. Indexed LA volume e"32 ml/m2 was 100.0% specific for the detection of abnormal diastolic function, although the sensitivity decreased to 67.0%, which are comparable with the current study.

Finally, in the present study by ROC analysis it was found that LAVI with a cut off value of 33.75 ml/m2 can predict adverse in-hospital outcome in patients of acute STEMI underwent reperfusion therapy with sensitivity 66.2% and specificity 75%. Moreover, it was found by ROC analysis

that LAVI can predict better adverse in-hospital outcome in comparison to NT-proBNP (66.2% vs 50.0%).

Study Limitations

Although the result of this study supports the hypothesis, there were some limiting factors which might affect the results:(a) this study was conducted at a single tertiary care hospital which may not represent the general population, (b)purposive sampling was done instead of random sampling.

Conclusion:

The findings of the present study showed a significant association between increased LAVI and adverse in-hospital outcomes. Thus, it may be concluded that increased LAVI can predict adverse in-hospital outcomes in acute STEMI patients. There is a significant correlation between LAVI and NT-proBNP in patients suffering from acute STEMI. Although the prognostic significance of elevated NT-proBNP is well established, the correlation with LAVI could also guide us in predicting adverse cardiovascular outcomes associated with LA dilatation in patients at an early stage of acute STEMI. LAVI can be measured by a 2D echocardiography machine in any low-resource hospital in our country, and it can be used as a cost-effective tool for predicting adverse cardiovascular outcomes in acute STEMI.

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Disclosure

The author have no conflict of interests to disclosure.

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Original Article

Magnitude of ST-Segment Elevation in Acute Inferior Myocardial Infarction and the Proximity of Right Coronary Artery Lesion

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Abstract:

Background & objective: Involvement of the right coronary artery frequently occurs in acute inferior myocardial infarction. Typical ECG changes in this condition involve ST-segment elevation in inferior leads. The present study was intended to predict the site of the lesion in the right coronary artery (RCA) in patients with acute inferior wall myocardial infarction using the height of ST-segment elevation as the predictor variable.

Methods: The present cross-sectional study was carried out in the Department of Cardiology, National Institute Cardiovascular Diseases (NICVD), Dhaka, Bangladesh over a period of one year between July 2010 to June 2011. Patients with acute inferior myocardial infarction admitted to CCU of NICVD within 12 hours of the onset of chest pain and underwent coronary angiography within 4 weeks of acute myocardial infarction (AMI) were the study population. With the help of a 12-lead ECG, magnitudes of ST-segment elevation in leads II, III, and aVF were measured. The highest degree of stenosis along the RCA revealed by an angiogram was accepted as the culprit lesion. The right coronary artery was divided into proximal (from its ostium to the origin of the RV branch), mid (from the RV branch to the acute marginal branch), and distal (from the acute marginal branch onward) parts. The sum of ST-segment elevation was then computed and compared among the three groups of patients divided on the basis of the site of lesion in RCA.

Result: The findings of the study showed that nearly half (48%) of the patients had lesions in the proximal, 38% in the mid, and the rest (14%) in the distal part of the right coronary artery (RCA). While patients with proximal lesions had the highest mean sum of the ST-segment elevation (12.1 ± 0.6 mm), those with distal lesions had the lowest mean sum of the ST-segment elevation (6.1 ± 0.2 mm). The three groups were significantly heterogeneous ($p < 0.001$). The magnitude of ST-segment elevation in Lead II, III, and aVF and the sum of ST-segment elevation all were significantly higher in patients with proximal lesions than those in patients with mid and distal lesions ($p < 0.001$).

Conclusion: The magnitude of ST-segment elevation can predict the site of lesion in RCA in inferior wall myocardial infarction. The greater the height of ST-segment elevation, the higher the probability of lying the lesion in the proximal part of the RCA.

Key-words: *Acute inferior myocardial infarction, right coronary artery (RCA), ST-Segment elevation, etc.*

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Introduction:

Acute myocardial infarction (AMI) is a major component of acute coronary syndrome which usually results from anterior and inferior myocardial wall involvement.[1] Unlike anterior wall acute myocardial infarction, which is a fairly homogenous entity, the extent of acute inferior wall MI depends on the infarct-related artery and its site.[2] Nearly half of the patients with acute inferior wall MI have specific haemodynamic and brady-arrhythmic complications, usually due to the total occlusion of the proximal right coronary artery (RCA), which significantly alters an otherwise favourable prognosis.[1,3] Such patients, including those in whom electrocardiogram (ECG) evidence of right ventricular infarction (RVI) increase the risk for death, shock, and arrhythmia.[4,5]

The incidence of mortality and complications is high in patients with acute inferior wall MI with right ventricular involvement.[6] The incidence of right ventricular infarction in an acute inferior MI setting is about 30%.[5] The mortality of patients with only inferior wall acute myocardial infarction is 5-6%, which increases to 25-30% if the right ventricle is also involved. The patients with RVI had a higher hospitalization rate and a higher incidence of major hospital complications than those without RVI (31 vs. 6%, $p < 0.001$; 64 vs. 28%, $p < 0.001$).[7] Acute inferior myocardial infarction (AIMI) is sometimes complicated by hypotension and arrhythmia. In the majority (80%) of patients with acute inferior wall myocardial infarction, the infarct-related artery is the RCA, while it is the left circumflex artery (LCx) in the rest.[1] In the presence of complications, RCA is generally the infarct-related artery. Therefore, immediate diagnosis of the infarct-related artery and its site of the lesion has implications in predicting prognosis and deciding management. Predicting the probable site of occlusion within RCA is of utmost importance, for proximal occlusions are likely to cause greater myocardial damage and an early invasive strategy may be planned in such cases.

Early and almost accurate identification of infarct-related coronary arteries in acute MI can guide the decision-making process not only regarding the urgency of revascularization but also to avoid therapy that may adversely affect the outcome. But the conclusive diagnosis of the culprit artery with lesion site and size is feasible only with an angiogram, which is a time-consuming and invasive procedure. It has been observed in several studies that the height of ST-segment elevation from bedside ECG can predict the site of lesion in major coronary arteries with a fair degree of accuracy. The present study was intended to predict the site of lesion in RCA in acute inferior myocardial infarction using the height of ST-segment elevation with the hypothesis in mind that the magnitude of ST-segment elevation in acute inferior myocardial infarction correlates with the proximity of the right coronary artery lesion.

Materials and methods:

This cross-sectional study was conducted in the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh over a period of 12 months from July 2010 to June 2011. Patients with acute inferior myocardial infarction who got admitted to CCU within 12 hours of chest pain and underwent coronary angiogram (CAG) within 4 weeks of AMI were the study population. Of them, whose coronary angiogram showed culprit lesions in RCA were included in the study. However, patients with previous MI or a coronary angiogram (CAG) showing a non-dominant RCA or culprit lesion at the LCX, intraventricular conduction defects, ventricular ectopics, and paced rhythm, left ventricular hypertrophy, cardiomyopathy, valvular or congenital heart disease, acute pericarditis, and acute myocarditis were excluded from the study.

Ethical clearance was obtained from the Ethical Review Committee of NICVD. A total of 100 patients were included in the study. Initial evaluation of the patients was done by history, clinical examination, and relevant investigations. Demographic data (age, sex) were recorded. Risk factor profiles, such as hypertension, diabetes, dyslipidemia, family history of coronary artery disease, and smoking were noted. In all patients, a standard 12-lead ECG, right-sided precordial ECG (V_3R through V_6R) was recorded at a paper speed of 25 mm/sec and an amplification of 10 mm/mV immediately after admission to the hospital. ST-segment elevation > 0.1 mV in two or more leads of II, III, and aVF was considered to be an indicator of acute inferior myocardial infarction. The isoelectric line was defined as the level of the preceding TP segment. The magnitude of ST-segment elevation in leads (II, III, and aVF) was measured 80 ms after the J point relative to the TP segment. The sum of the ST segment was then calculated. The right ventricular injury (RVI) was diagnosed by the presence of an ST-segment elevation e" 0.1 mV in lead V_4R. Coronary angiography was done within 28 days of MI. All standard views were taken. In selected cases, additional views were taken.

The CAG was analyzed by visual estimation. Seventy percent or more luminal stenosis was considered significant. The reporters of CAG had no prior knowledge of the ECG status of the patients. The lesion with the

highest degree of stenosis along RCA was accepted as the culprit lesion. The right coronary artery was divided into proximal, mid, and distal segments. The segment of RCA from its ostium to the origin of the RV branch was considered as proximal, from the RV branch to the acute marginal branch as mid, and from this point onward as the distal segment. Based on the site of lesions, patients were divided into three groups – Proximal (n = 48), Mid (n = 38), and Distal (n = 14) groups. The sum of ST-segment elevation was then computed and compared among the three groups of patients divided on the basis of the site of the lesion in RCA. Data were processed and analyzed using computer software SPSS (Statistical Package for Social Sciences). ECG findings were then correlated with angiographic findings. The test statistics used to analyze the data were ANOVA statistics and Chi-square ($\div^2$) Test. The level of significance is 0.05 and a p-value < 0.05 was considered significant.

Results:

The demographic characteristics were almost homogeneously distributed among the three groups. The mean duration of chest pain was almost similar among the three groups. The proportion of patients with shortness of breath, sweating, and nausea was significantly higher in the proximal group than those in the other two groups. The distributions of the risk factors like smoking habit, diabetes, and dyslipidemia were significantly higher in the proximal group (p = 0.017, p = 0.001, and p < 0.001 respectively). Hypertension and family history of IHD were also higher in the proximal group than those in the Mid and Distal groups, although the difference did not turn significant (p = 0.199 and p = 0.302 respectively). All the in-hospital complications were also observed to be significantly higher in the proximal group (p < 0.001, p = 0.012, p < 0.001, p = 0.005) (Table I).

Echocardiographic findings demonstrate that over 70% of the patients in the Proximal group had regional wall motion abnormality (RWMA) compared to 52.6% in the Mid group, and 7.1% in the Distal group (p < 0.001). The mean left ventricular ejection fraction (LVEF) of the Proximal group was significantly reduced compared to the other two groups (p = 0.060). More than half (52.1%) of the proximal lesions and 7.9% of the mid lesions in RCA had RVI. None of the distal lesions had RVI. RVI demonstrated a significant presence in the proximal group (p < 0.001) (Table II). The mean heights of ST-segment elevation in Lead II, Lead III, and aVF and the mean sum of ST-segment elevation showed a decreasing trend with the progress of lesion from the proximal to the distal site of RCA (p < 0.001, p < 0.001, p < 0.001 and p < 0.001 respectively) (Table III & Fig. 1).

Table-I

Distribution of baseline characteristics among the three groups

Baseline characteristics	Site of RCA lesion			p-value
	Proximal(n = 48)	Mild(n = 38)	Distal(n = 14)	
Age (year)#	52.5 ± 9.3	49.2 ± 9.2	50.2 ± 9.6	0.241
Sex*				
Male	43(89.6)	35(92.1)	14(100.0)	0.450
Female	5(10.4)	3(7.9)	00	
Mode of presentation				
Duration of chest pain*(hrs)	6.9 ± 2.8	7.5 ± 2.6	7.1 ± 2.5	0.582
Shortness of breath#	33(68.5)	16(42.1)	5(35.7)	0.016
Sweating#	34(70.8)	13(34.2)	4(28.6)	0.001
Nausea#	28(58.3)	7(18.4)	4(28.6)	0.001
Vomiting#	14(29.2)	5(13.2)	4(28.6)	0.187
Risk factors				
Smoking habit	34(70.8)	23(60.5)	4(28.6)	0.017
DM	31(64.6)	10(26.3)	4(28.6)	0.001
HTN	27(56.3)	15(39.5)	5(35.7)	0.199
Dyslipidaemia	28(58.3)	5(13.2)	2(14.3)	<0.001
Family H/O IHD	11 (22.9)	4(10.5)	2(14.3)	0.302
In-hospital complications				
Hypotension	36(75.0)	6(15.8)	3(21.4)	< 0.001
Cardiogenic shock	13(27.1)	3(7.9)	0(0.0)	0.012
Acute LVF	22(45.8)	4(10.5)	0(0.0)	< 0.001
Arrhythmia	19(39.6)	4(10.5)	2(14.3)	0.005

#Data were analyzed using ANOVA statistics and are presented as mean± SD.

*Data were analyzed using $\div^2$ Test; Figures in the parentheses denote corresponding percentage.

Table-II
Echocardiographic findings among the three groups

Echocardiogram	RCA Lesion			p-value
	Proximal(n = 48)	Mild(n = 38)	Distal(n = 14)	
RWMA*				
Yes	34(70.8)	20(52.6)	1(7.1)	< 0.001
No	14(29.2)	18(47.4)	13(92.9)	
LVEF (%)#	48.5 ± 12.7	51.5 ± 10.7	56.7 ± 7.3	0.060
RVI*				
Present	25(52.1)	3(7.9)	0(0.0)	<0.001
Absent	23(47.9)	35(92.1)	14(100.0)	

#Data were analyzed using ANOVA statistics and are presented as mean± SD;
*Data were analyzed using the $\div^2$ Test; figures in the parentheses denote corresponding percentage.

Table-III
Association of ST-segment elevation with the site of lesion in RCA

ST-segment elevation (mm)#	RCA Lesion			p-value
	Proximal(n = 48)	Mild(n = 38)	Distal(n = 14)	
Lead II	3.4 ± 0.5	2.0 ± 0.5	1.5 ± 0.5	<0.001
Lead III	4.7 ± 0.4	3.4 ± 0.5	2.6 ± 0.5	<0.001
aVF	4.2 ± 0.8	2.6 ± 0.5	2.0 ± 0.3	<0.001
Sum of ST-segment elevation	12.1 ± 0.6	8.0 ± 0.1	6.1 ± 0.2	<0.001

#Data were analyzed using ANOVA statistics and are presented as mean ± SD;

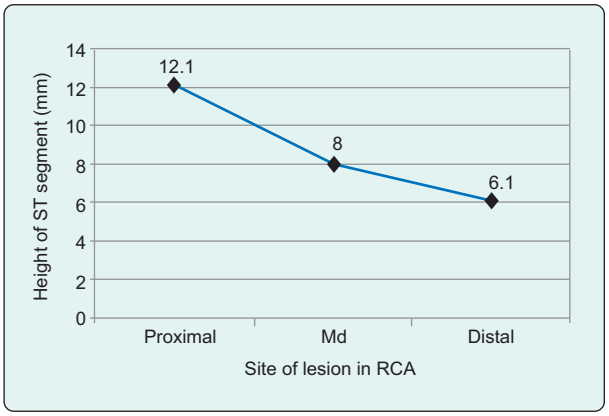

Fig.-1: *Relationship between the height of ST-elevation and the site of lesion in RC*

Discussion:

In the present study, the age and sex distribution were almost identical among the three groups. The mean duration of chest pain was also similar among the groups. The presence of smoking habit, diabetes, and dyslipidemia was higher in the proximal group than those in the mid and distal groups. Naqvi and associates[8] in a similar study reported that nearly half (48.4%) of the patients had the culprit lesion in the proximal, 38.5% in the mid, and 13.4% in the distal part of RCA bearing consistency with the findings of the present study. In our study, the magnitude of ST-segment elevation in leads II, III, and aVF and the sum of ST-segment elevation all were significantly greater in patients with proximal RCA lesions than that in the mid-lesion group, which, in turn, were again greater than that in the distal group ($p < 0.001$). These findings indicate that the greater the magnitude of ST-segment elevation, the higher the likelihood of proximity of lesion in RCA. Consistent with these findings, Naqvi et al[8] reported that total ST-elevation showed a decreasing trend as the lesion progressed from the proximal to the distal location in RCA.

Alim and colleagues[9] reported that patients with the culprit lesions in the proximal portion of the RCA had a mean ST-segment elevation of 12.6 ± 3.8 mm, while the patients having culprit lesions in the mid and distal portions had mean ST-segment elevations of 6.9 ± 1.2 mm and 5.1 ± 0.9 mm respectively which favour the findings of the present study. They also demonstrated a significant positive correlation between the magnitude of ST-segment elevation and the culprit lesion proximity ($r = 0.82$, $p < 0.01$ for the proximal and $r = 0.7$, $p < 0.05$ for the mid portions of RCA). In addition to the occlusion of RCA, the proximity of the culprit lesion along the course

of the artery is also important with regard to the potential complications (SA node dysfunction, RV infarction, etc.) in the setting of acute inferior-wall MI. Right ventricular myocardial infarction is associated with an increased risk of death, shock, ventricular tachycardia or fibrillation, and atrioventricular blocks.[10] The majority of the patients with proximal lesions in RCA exhibited higher rates of in-hospital complications like hypotension, cardiogenic shock, acute LVF, and arrhythmias compared to their mid and distal counterparts which are consistent with the findings of Manka.[10] The frequency of RVI associated with inferior wall MI was demonstrated to be 33% in Naquvi's study[8], which is quite similar to the findings of the present study (28%). Several investigators and [11] also reported similar incidences of RVI (28% and 27% respectively).[11,12]

Conclusion:

The findings of the study suggest that the magnitude of ST-segment elevation is indicative of the proximity of the lesion in RCA. The greater the height of ST elevation, the higher the probability that the lesion will lie in the proximal part of the artery.

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Original Article

Assessment of Heart Failure Patients in a Tertiary Care Hospital: A Retrospective Study

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Abstract:

Background: Heart failure is a complex syndrome that arises from abnormalities in the structure and/or function of the heart, whether inherited or acquired. It impairs the ability of the left ventricle to fill or eject blood, leading to a growing number of hospitalizations, deaths, and healthcare costs worldwide. According to the Global Burden of Disease Study 2019, heart failure affected approximately 64 million people worldwide in 2019, and caused over 3 million deaths. The prevalence of heart failure has been increasing worldwide, with an estimated 26% increase in age-standardized prevalence and a 14% increase in age-standardized incidence between 1990 and 2019. This increase in prevalence and incidence is due to a variety of factors, including the aging of the population, improved survival rates from other cardiovascular diseases, and changes in lifestyle and risk factors such as obesity and diabetes.

Heart failure patients have various presentations and different etiologies. This study aimed to describe the baseline characteristics, associated co-morbid conditions, presenting features, and causes of heart failure in a study population of 3650 patients admitted to Colonel Maleque Medical College, Manikganj.

Methods: This study was done to see Clinical Presentation of Heart Failure Patients admitted in Colonel Maleque Medical College, Manikganj and Maikganj Sadar Hospital. A total of 3650 patients were enrolled in this study during the period of April 2018 to March 2023.

Results: Most of the patients (60%) were of 51-70 years age group. 70 % (2555) patients were male. 99% patients presented with SOB, 95% patients had basal creps, 70% had orthopnoea, 49% had Paroxysmal Nocturnal Dyspoea (PND), 40% had leg edema and 25% had raised JVP. Average heart rate was 84 beats/min, average systolic B.P. was 128 mm Hg and average diastolic B.P. was 76 mm Hg. 49% population had hypertension, 39% patients had diabetes and 28% had concomitant respiratory illness. Average EF was 37 %. Ischemic Cardiomyopathy was the commonest (40%) cause of heart failure, acute coronary syndrome was the second leading (30%) cause, valvular heart disease and hypertension are the third common causes.

Conclusion: This study highlights the significant burden of heart failure in a population of patients admitted to a tertiary care hospital in Bangladesh. The findings underscore the importance of early detection and management of risk factors for heart failure, such as hypertension and diabetes, to prevent the development and progression of this condition. The identification of the most common causes of heart failure may guide targeted prevention and management strategies in this population. Most common causes are ischemic cardiomyopathy, a sequel of ischemic insult of the heart. So, patients of acute or chronic ischemic heart diseases should be treated and followed up with care, considering their socioeconomic conditions also.

Key words: *Clinical presentation, Heart failure, Hospitalized patients.*

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Introduction:

Bangladesh is passing through an epidemiological transition. The burden of infectious diseases is coming down while with increased life expectancy and wide spread change of lifestyle, non-communicable diseases are on the rise [1] . Cardiovascular diseases are one of the main causes of morbidity and mortality in this country now. Heart failure (HF) is a significant and growing health problem as the population ages. Despite improvements in therapy, mortality and morbidity remain high [1,2]. Introduction:

Heart failure is a complex clinical syndrome that arises secondary to abnormalities of cardiac structure and/or function (inherited or acquired) that impair the ability of the left ventricle to fill or eject blood.[1]

The worldwide prevalence and incidence rates of heart failure are approaching epidemic proportions, as evidenced by the relentless increase in the number of heart failure attributable death, and the spiraling costs associated with the care of heart failure patients.[1] According to the Global Burden of Disease Study 2019, heart failure affected approximately 64 million people worldwide in 2019, and caused over 3 million deaths. The prevalence of heart failure has been increasing worldwide, with an estimated 26% increase in age-standardized prevalence and a 14% increase in age-standardized incidence between 1990 and 2019. This increase in prevalence and incidence is due to a variety of factors, including the aging of the population, improved survival rates from other cardiovascular diseases, and changes in lifestyle and risk factors such as obesity and diabetes. Estimates of the prevalence of symptomatic heart failure in the general European population are similar to those in the United States and range from 0.4 to 2 percent. The prevalence of heart failure follows an exponential pattern, rising with age, and affects 6 to 10 percent of people older than 65 years. The overall prevalence of heart failure is thought to be increasing, in part because our current therapies for cardiac disorders, such as myocardial infarction, valvular heart disease, and arrhythmias, are allowing patients to survive longer. The signs and symptoms typically associated with heart failure do not always arise directly from the cardiac abnormalities in the failing heart, but can be a result of abnormalities that occur in distant organs such as the kidneys or skeletal muscle. The dysfunction in these organs cannot be fully explained by reduced perfusion pressure alone, and suggests that other systemic processes such as neurohormonal activation may also contribute to the development of heart failure. Although it was previously believed that heart failure occurred mainly in patients with reduced left ventricular ejection fraction (EF), epidemiological studies have shown that approximately half of heart failure patients have a normal or preserved EF. Furthermore, studies have demonstrated that patients can have significant abnormalities in left ventricular contraction and relaxation, yet remain asymptomatic, in which case they are considered to have asymptomatic heart failure. In cases where a patient with chronic heart failure experiences a decline in their condition, they are referred to as having decompensated heart failure. If the onset of symptoms is sudden, it is referred to as acute decompensated heart failure. The term “congestive heart failure” is considered outdated, as it originally referred to patients with heart failure who often had edema or were overloaded with fluids. However, with modern medical and device therapies, most heart failure patients are able to maintain normal fluid levels, and are simply referred to as having heart failure.[2,3,4,5,6]

In developed countries, coronary artery disease, often in conjunction with hypertension, appears to be the predominant cause of heart failure. However, it can be challenging to identify the primary cause of heart failure in patients with multiple possible contributing factors, such as diabetes mellitus, atrial fibrillation, and hypertension. Even in patients without obvious hypertension at presentation, it is possible that hypertension played a significant role in the past and contributed to the development of heart failure[7].

The initial cohort of the Framingham Heart Study, which was monitored until 1965, found that hypertension was the primary cause of heart failure in 30% of men and 20% of women, with an additional 33% and 25%, respectively, citing hypertension as a contributing factor. The presence of left ventricular hypertrophy on electrocardiogram in individuals with hypertension was associated with an approximately 15-fold increased risk of developing heart failure.

However, over the years of follow-up, coronary heart disease became increasingly prevalent as the cause of new cases of heart failure. In the 1950s, it accounted for 22% of cases, but by the 1970s, it was responsible for almost 70% of cases. In contrast, the relative contribution of hypertension and valvular heart disease declined significantly during this period.[5,6]

During the period under study, the prevalence of hypertension decreased by around 5% and 30% per decade among men and women, respectively. This decline can be attributed to the widespread use of antihypertensive medications. The decrease in the prevalence of left ventricular hypertrophy, a common complication of hypertension, further supports this conclusion. In addition, improvements in the accuracy of

diagnosing coronary heart disease likely contributed to its increasing recognition as a significant factor in heart failure during this same period..[7-9]

The Framingham data on heart failure should be interpreted with caution, as it relied solely on clinical criteria to identify cases and may have included individuals who did not have associated left ventricular systolic dysfunction. In contrast, many large-scale clinical trials have primarily enrolled patients with reduced left ventricular ejection fractions and excluded patients based on a wide range of criteria.[10-11]

Methods: Methods:

The study population consisted of patients admitted to the hospital with features of heart failure. Diagnostic criteria for the diagnosis of congestive heart failure are paroxysmal nocturnal dysnoea, ortopnea, exertional dyspnoea, elevated JVP, pulmonary basal creps, third heart sound, peripheral edema, night cough, hepatomegaly, pleural effusion, cardiomegaly on CXR, pulmonary edema on CXR, and echocardiographic findings. A total of 3650 patients were enrolled in this study during the period of April 2018 to March 2023.

Ethical approval for this study was granted by the ethics committee. A cardiologist took a standardized medical history and examined all the patients after hospital admission, and the clinical findings of the admitting doctors were noted. Whenever possible, an electrocardiogram, chest radiograph, transthoracic echocardiogram, serum biochemistry, hematology, and thyroid function tests were performed. The echocardiogram was done according to a standard protocol and according to accepted guidelines by a cardiologist, and the two-dimensional, M-mode, Doppler, and color-flow images were recorded.

Results:

Table-I

Baseline Characteristics of study population N=3650

Characteristics	
Age range	18-87 years
Average age	47 ± 07 years
Most of patients (75%)	51-70 years
Male patients	2555(70%)
Female patients	1095 (30%)
Ejection fraction	37%
Average LV dimension in Diastole	58 mm
Average LV dimension in Systole	44 mm
Average pulse/min	84 beats/min
Average Systolic BP	128 mm Hg
Average Diastolic BP	76 mm Hg

Table-II

Associated co-morbid conditions of study population N=3650

Characteristics	Percentage (%)
Hypertension	49%
Diabetes	39%
Dyslipidemia	20%
Atrial fibrillation	09%
Respiratory Diseases	28%

Table-III

Presenting Features of study population N=3650

Characteristics	Percentage (%)
Shortness of Breath	99%
Bilateral Basal Creps	95%
Orthopnoea	70 %
Paroxysmal Nocturnal Dysnoea	49%
Leg edema	40%
Raised JVP	25%

Table-IV

Causes of Heart Failure of study population N=3650

Characteristics	No. (%)
Acute coronary syndrome	1095 (30%)
Chronic ischemic heart disease	73 (02%)
Ischemic cardiomyopathy	1460 (40%)
Dilated Cardiomyopathy	219 (06%)
Postpartum Cardiomyopathy	146 (04%)
Hypertension	292 (08%)
Valvular heart disease	292 (08%)
Cor –pulmonalae	21 (0.58%)
Congenital heart disease adult	15 (0.42%)
Hypothyroidism	23 (0.63%)
Hyperthyroidism	14(0.37%)

Discussion:

Total 3650 patients of heart failure were enrolled. Patients are of 14 to 87 years age range. Average age was 47 ± 07 years. Most of the patients (75 %) in 51-70 years age groups. In SOLVD clinical trial,[12] mean age was 61 years. In DIG study (1997),[13] RALES study,[14] MERIT-HF study,[15] ATLAS Study[16] mean age was 64 years. M Kabiruzzaman et al [22] showed mean age was 54 years.

The Hillingdon heart failure study evaluated the incidence and aetiology of heart failure in one district of west London, England using clinical and echocardiographic data and a case definition based on three cardiologists

applying the ESC definition of heart failure .The median age at the time of diagnosis of heart failure was 76 years. The incidence of heart failure was significantly higher in men than women at all ages with an age-standardised ratio of 1.75. The primary aetiologies were coronary heart disease (36%), unknown (34%), hypertension (14%), valve disease (7%), atrial fibrillation alone (5%), and other (5%) [8].

Mcmurray et al[9] studied trends in hospitalization for heart failure in Scotland 1980-1990. They found seventy-eight percent of discharges were in persons aged e" 65years and 48% of discharges were male.

In our study, Male was 70% and Female was 30%. In SOLVED clinical trial, male was 80 % and female was 20 %. In DIG study[13] and MERIT-HF [15] male was 78 %.

In this study 99 % patients presented with shortness of breath , 70 % patients presented with orthopnea, 49 % presented with paroxysmal nocturnal dyspnoea (PND), 40 % presented with ankle edema, 25 % presented with raised JVP and 95% had bilateral basal crepitations .

In the present study, as a co- morbid condition, 49 % had history of Hypertension, 39 % had Diabetes, 28 % had Respiratory disease, 09% had Atrial Fibrillation. In SOLVD (1991) clinical trial,[12] 42 % had Hypertension, 26 % had Diabetes, 10 % had Atrial Fibrillation. In MERIT-HF clinical Trial,[15] 44 % had Hypertension, 25 % had Diabetes and 17 % had Atrial Fibrillation.

In our study, 72 % diagnosed as Ischemic Heart Disease (Acute Coronary Syndrome 30 %; Ischemic Cardiomyopathy 40 % and Chronic Ischemic Heart Disease 2%. In SOLVD[12] clinical trial 71% had ischemic cause of heart failure, in DIG[13] study 70 % had ischemic cause of heart failure, in MERIT- HF [15] 66 % had ischemic cause of heart failure. In ATLAS [16] study, 64 % had ischemic cause of heart failure. In RALES study,[14] 54 % had ischemic cause of heart failure. 08.39% patients had hypertensive heart failure, In DIG [13] (1997) study, 09% had hypertensive heart failure and in ATLAS [16] study , 20 % had hypertensive heart failure.

In the present study, 08 % had valvular cause of heart failure. In ATLAS study, 06% had valvular cause of heart failure. In SPICE registry, [17] 05 % had valvular cause of heart failure.

In our study, 06 % was diagnosed as DCM (Dilated Cardiomyopathy) as a cause of heart failure. In SOLVD (1991) clinical trial 18% had DCM, in DIG study 15 % had DCM as a cause of heart failure, in SOLVD [18] registry 13 % had DCM. In ATLAS study, 28 % had DCM as a cause of heart failure. In SPICE [17] registry, 17 % had DCM.

In Pakistan, Jafary et al studied 196 patients with mean age 61.2 ± 12.8 years with a high preponderance of males. All of them were suffering from systolic heart failure with LVEF d" 40%, requiring hospital admission with more than 60% suffering from hypertension (67.3%) and diabetes mellitus (60.7%) and more than three-fourths havinga history of coronary artery disease in the past [23].

In the United Kingdom, most patients admitted to hospital with heart failure are more than 65 years old. The prevalence of heart failure rises from around 1% in the age group 50-59 years to between 5 and 10% of

those aged 80-89 years. Heart failure is frequently due to coronary artery disease [24].

Seow et al 25 studied 225 patients in Singapore with LVEF d" 40%, their mean age was 68.5± 2.3 years and more than 51.1% of the subjects were aged 70 years and more. The most common cause of HF was coronary heart disease (85.5%). Co morbid medical conditions were prevalent in this cohort, with 83.5% having at least one co-morbid condition. Hypertension was the most prevalent co-morbid condition; affecting 60% of the patients; followed by diabetes mellitus (56.9%).

Remes et al27 studied incidence of heart failure in 45-74 year old inhabitant in four rural communities in Eastern Finland. The incidence rates of heart failure increased with age in both sexes. Coronary heart disease or hypertension was evident in 80% cases.

Another study published in the Journal of the Bangladesh College of Physicians and Surgeons in 2020 assessed the clinical presentation, risk factors, and comorbidities of heart failure in 150 patients admitted to a tertiary care hospital in Dhaka, Bangladesh. The study found that the most common presenting symptoms were dyspnea (96%), fatigue (90%), and edema (71%). The most common comorbidities were hypertension (69%), diabetes mellitus (44%), and ischemic heart disease (24%). The study also found that the majority of patients had reduced ejection fraction (73%). This study highlights the similarities in the clinical presentation and comorbidities of heart failure in Bangladesh, as well as the importance of identifying and managing risk factors for heart failure.[28]

A study published in the Bangladesh Journal of Medicine in 2016 analyzed the clinical characteristics of 200 patients with heart failure admitted to a tertiary care hospital in Dhaka, Bangladesh. The study found that dyspnea was the most common presenting symptom

(94%), followed by fatigue (70%), orthopnea (52%), and edema (36%). The study also found that the most common comorbidities were hypertension (69%), diabetes mellitus (28%), and ischemic heart disease (23%).[29]

A study published in the European Journal of Heart Failure in 2014 analyzed the clinical presentation of 3,791 patients with acute HF across 19 European countries. The study found that the most common symptoms at presentation were dyspnea (89%), fatigue (70%), and edema (43%). The most common signs at presentation were elevated jugular venous pressure (74%), crackles on lung examination (72%), and peripheral edema (65%).6

Another study published in the Journal of the American College of Cardiology in 2017 analyzed the clinical characteristics of 5,887 patients hospitalized for HF across 162 hospitals in the United States. The study found that dyspnea was the most common presenting symptom (82%), followed by edema (47%), fatigue (41%), and orthopnea (38%). The study also found that patients with HF with reduced ejection fraction (HFrEF) were more likely to present with dyspnea and orthopnea, while patients with HF with preserved ejection fraction (HFpEF) were more likely to present with edema and fatigue.[30]

A study published in the Journal of Emergency Medicine in 2018 analyzed the clinical characteristics of 106 patients with acute HF who presented to the emergency department. The study found that the most common presenting symptoms were dyspnea (86%), cough (46%), and orthopnea (29%). The most common physical exam findings were elevated jugular venous pressure (79%), rales on lung exam (78%), and lower extremity edema (76%).[31]

A study published in the Journal of the American Medical Association in 2013 analyzed the clinical presentation of 48,612 patients hospitalized for HF across 254 hospitals in the United States. The study found that dyspnea was the most common presenting symptom (71%), followed by fatigue (48%), orthopnea (41%), and edema (38%). The study also found that patients with HFrEF were more likely to present with dyspnea and orthopnea, while patients with HFpEF were more likely to present with fatigue and edema.[32]

Another study published in the European Journal of Heart Failure in 2020 analyzed the clinical characteristics of 10,906 patients hospitalized for HF in 20 countries across Asia, Europe, and South America. The study found that dyspnea was the most common presenting symptom (80%), followed by fatigue (62%), orthopnea (48%), and edema (47%). The study also found that patients with HFrEF were more likely to present with dyspnea and orthopnea, while patients with HFpEF were more likely to present with fatigue and edema.[33]

A study published in the Journal of Cardiovascular Medicine in 2020 analyzed the clinical characteristics of 574 patients hospitalized for acute HF in Italy. The study found that dyspnea was the most common presenting symptom (88%), followed by fatigue (50%), orthopnea (49%), and edema (43%). The study also found that patients with HFrEF were more likely to present with dyspnea and orthopnea, while patients with HFpEF were more likely to present with fatigue and edema.[34]

A study published in the European Journal of Heart Failure in 2019 analyzed the clinical characteristics of 1,008 patients hospitalized for HF in Spain. The study found that dyspnea was the most common presenting symptom (91%), followed by fatigue (49%), orthopnea (45%), and edema (37%). The study also found that patients with HFrEF were more likely to present with dyspnea and orthopnea, while patients with HFpEF were more likely to present with fatigue and edema.[35]

Another study published in the Journal of Cardiac Failure in 2020 analyzed the clinical characteristics of 1,267 patients hospitalized for HF in the United States. The study found that dyspnea was the most common presenting symptom (78%), followed by edema (56%), orthopnea (47%), and fatigue (40%). The study also found that patients with HFrEF were more likely to present with dyspnea and orthopnea, while patients with HFpEF were more likely to present with fatigue and edema.[36]

A study published in the Korean Circulation Journal in 2019 analyzed the clinical characteristics of 1,259 patients hospitalized for acute HF in Korea. The study found that dyspnea was the most common presenting symptom (83%), followed by edema (52%), orthopnea (43%), and fatigue (41%). The study also found that patients with HFrEF were more likely to present with dyspnea and orthopnea, while patients with HFpEF were more likely to present with fatigue and edema.[37]

Conclusion:

Despite a decline in age-adjusted mortality from coronary heart disease (CHD) in developed countries overall, the number of patients with chronic CHD is increasing. This is principally the result of two separate trends. Firstly, the proportion of elderly in the population is increasing rapidly, and these subjects have the highest incidence of CHD and hypertension. Secondly, survival for those with coronary artery disease is improving. In particular, it has been shown that survival after acute myocardial infarction has increased notably over the past decade, at least in part because of better medical treatment. As coronary artery disease is the most powerful risk factor for heart

failure, it is likely that the aforementioned trends will lead to an increase in its future prevalence. Chronic heart failure may, therefore, become a more common manifestation of chronic heart disease and contribute to many more deaths. 19-21,26

In our study, most of the heart failure patients were in the elderly age group. The most common cause is ischemic cardiomyopathy, a sequel of ischemic insult of the heart. So, patients with acute and chronic ischemic heart disease should be treated and followed up with care, considering their socioeconomic condition as well. NSAIDs, steroids, and other fluid-retaining drugs should be used cautiously in cardiac patients prone to developing heart failure. The number of patients with heart failure is bound to rise at a premature age if appropriate measures are not taken to manage risk factors and increase public awareness. A clinical and epidemiological study is needed to explore further details.

The study provides a comprehensive overview of the characteristics and comorbidities of a large population of patients with heart failure. These findings may help clinicians better understand the clinical profile of patients with heart failure and improve their management and treatment.

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Original Article

Any Target Value of LDL-cholesterol before Elective PCI? A study at NICVD on Association of LDL-Cholesterol levels with Myocardial Injury during Elective PCI

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Abstract:

Background: Raised LDL cholesterol has already been established as a strong risk factor the for pathogenesis of atherosclerotic plaque leading to coronary heart disease. During PCI procedure, many patients develop peri-procedural myocardial injury due to mainly atherosclerotic plaque disruption, side branch occlusion and distal small vessel embolization which subsequently affects the mortality and morbidity of patients. Objectives: To find out the association of pre-procedural LDL cholesterol levels with myocardial injury in elective PCI patients. Methods: This Cross-sectional observational study was conducted at National Institute of Cardiovascular Diseases (NICVD), Dhaka from July 2020 to June 2021. The sample size was 170. LDL cholesterol and troponin-I were measured before the procedure and troponin I was measured 6 hours after PCI procedure. On the basis of pre-procedural cholesterol level, the study population were categorized into two groups: Group I: patients with normal LDL-C level (≤70mg/dl) and Group II :patients with raised LDL-C (>70mg/dl). Results: Total 54(31.8%) patients developed peri-procedural myocardial injury, among them 15(19.7%) were in normal LDL-C group and 39(41.5%) were in raised LDL-C group. Elevation of troponin I after PCI was higher in group II than group I patients with statistically significant difference (p<0.001). Multivariate logistic regression analyses showed that raised LDL-C was an independent predictor of PMI (OR 4.71; 95% CI, 2.072-10.658; *p*<0.001). There was positive correlation found between pre-procedural LDL-C and myocardial injury (*r*=0.44, *p*<0.001) by Pearson's correlation coefficient test. Conclusion: Pre-procedural raised LDL-C level was positively and independently associated with myocardial injury after elective percutaneous coronary intervention (PCI).

Key words: *Percutaneous Coronary Intervention (PCI), Low density lipoprotein (LDL) cholesterol, Peri-procedural myocardial injury (PMI).*

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Introduction:

Intervention (PCI) remains an important approach for the treatment of occlusive coronary artery disease (CAD)[1]. Since the first description of coronary angioplasty in man by Andreas Gruentzig in 1977, the procedure has been extensively modified. The technical advances coupled with the use of coronary stents and adjuvant drug therapy have resulted in high procedural success rates and low restenosis rates. Older patients are now being treated, more complex multiple coronary lesions deemed appropriate for PCI

About one-third of all elective PCI procedures are associated with significant myocardial injury (termed peri-procedural myocardial injury, PMI) which has been associated with increased subsequent mortality[2]. In 2012, the third universal definition of MI[3] was the first to provide a clinically oriented definition of both type 4a MI and myocardial injury. According to 4th universal definition, myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL).

The elevation of cardiac marker after PCI mainly results from incidental minor side branch occlusion around target lesions and distal embolization of plaque debris or thrombus from target lesion.

Troponin (Troponin-I and Troponin-T) are more sensitive and specific markers of cardiac injury than CK-MB[4]. Troponin increases following PCI had originally thought to carry less prognostic importance than CK-MB elevations, although recent studies and meta-analysis have shown that troponin elevations post PCI are prognostically significant. Nienhuis et al. (2008) in their meta-analysis of 15581 patients from 20 studies over a 19 year period reported the incidence of troponin release post PCI in elective PCI to be 33% and increased mortality was significantly associated with troponin elevation after PCI (4.4 vs 3.3%, p=0.001; OR=1.35)[5] . Peri-procedural MI can be difficult to rule out as the symptoms, electrocardiographic changes, angiography and others imaging modalities can be uncertain due to older ischemic injuries and discomfort associated with the procedure itself. A study showed that on angiography only approximately 60% of peri-procedural MI could be explained[6]. Clinicians must therefore rely considerably on cardiac biomarkers.

In Bangladesh about one-third of all elective PCI procedures are associated with significant myocardial injury[7]. Traditional risk factors for PMI are advanced age (>50years), female gender, DM, BMI, hypertension, smoking, dyslipidaemia ,type C lesion, multiple stents, post dilatation and hs-CRP. Ahmed et al. (2015) reported that those patient had hs-CRP > 3mg/dl has 20% more chance (33.06% vs 11.52%) of myocardial injury than normal hs-CRP (d"3 mg/dl) leveled patients[8]. This is probably due to sustained inflammatory response of coronary artery which subsequently leads to distal micro embolization. Stent length has also impact on myocardial injury. In 2014 a study was done in NICVD and that study showed that those patient had been implemented 20 mm or more stent length has more chance of myocardial injury[7]. This is due to side branch occlusion. It has been reported in 12.5%-19% of cases in which a stent was placed across a major side branch[9]. Herrmann (2005) in his review article, classified it as type-1 or proximal PMI. Another type, Type-2 PMI or distal PMI is due to atherosclerotic plaque disruption , local vessel trauma and distal embolization. Type 2 PMI constitutes 50%-75% of PMI cases which is directly related to the process of atherosclerosis[10].

Low density lipoprotein cholesterol (LDL) is most atherogenic cholesterol. It was found that LDL cholesterol is directly related to atherosclerosis. It was also found that 1% rise in serum total cholesterol level leads to 2% increase in ischaemic heart disease (IHD). Regarding Bangladeshi population mean LDL cholesterol level is 119mg/dl[11]. In NICVD a study was done regarding the LDL cholesterol level in ACS patient during 2016 to 2018 period. That study reported that LDL cholesterol level was found high (>130mg/dl) in 58% patients. Similar study was done in some tertiary level hospital in Bangladesh, showed that patient admitted with acute myocardial infarction had dyslipidaemia in the form of raised LDL cholesterol (> 100mg/dl) in 75% cases[12]. More lipo-protein in blood is associated with more bigger SYNTAX score which means the coronary vessel lesion more complex. LDL cholesterol is also a direct predictor for coronary plaque progression. Patients with LDL-C bellow 70 mg/dl displayed a significant attenuation in plaque progression as compared with those follow-up LDL-C levels e" 70 mg/dl (12.7±38.2 mm^3 vs 44.2±73.6 mm^3 , respectively; p=0.014)[13]. Coronary artery plaque is the hallmark for peri-procedural myocardial injury (especially type-2 PMI). Kabir et al. (2019) reported that dyslipidaemia is an important target for prevention of periprocedural myocardial injury[14]. The ARMYDA-RECAPTURE trial showed that reloading patients who are already receiving statin treatment (application of 80mg of atorvastatin > 12h prior to procedure and application of an additional pre-procedural dose of 40mg)

markedly reduces the primary end point of cardiac death, MI or unplanned revascularization at 30 days (3.7% vs 9.4%). Statin pretreatment also reduces the rate of peri-procedural myocardial injury and Major Adverse Cardiac Events (MACE) following stent implantation[15]. Chronic treatment with statin can reduce fibrous-cap thickness of lipid rich plaque and this explain why patients receiving chronic statin treatment experiences less PMI during PCI. This current study was aimed to evaluate the association between pre-procedural LDL cholesterol levels with myocardial injury in elective cases.

Methods:

This Prospective observational study was conducted in the Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka during the period from July 2020 to June 2021. Patients admitted with Coronary Artery Disease (CAD) who were undergoing for elective Percutaneous Coronary Intervention (PCI) included in this study. Initially 190 patients were enrolled. Among them, 12 patients were found CTO lesion during CAG, 3 patients developed VT during the procedure, TIMI grade-3 flow was not produced in 2 patients, 1 patient died just after the procedure. Finally total number (n) of patient was 170. Patients with primary PCI, pharmaco-invasive PCI, primary PCI strategy, angiographic failure or death <24 hours after PCI, raised troponin I before the procedure, cardiac arrest during and after procedure, history of aortic dissection, CABG, valvular heart disease, Chronic Liver Disease (CLD), Chronic Kidney Disease (CKD), malignancy, advanced Chronic Obstructive Pulmonary Disease (COPD), asthma, coronary dissection during procedure, major side branch (≥ 02 mm) occlusion during procedure, no/slow reflow after procedure, acute stent thrombosis, multi-vessels PCI (>2), use of ≥3 stents, Complex PCI (LM/bifurcation lesion/ CTO lesion/Calcified lesion/Thrombus burden) were excluded. Informed written consent was taken from each patient before enrollment. LDL-C level was measured by Serum LDL cholesterol (Beckman Coulter Analyzer (Model- OLYMPUS AU480). In established CAD, target LDL-C level ≤70 mg/dl (ACC dyslipidaemia guideline, 2018). So I divided the patient into two groups according to the target level of LDL cholesterol. In group-1 patients with LDL cholesterol ≤70 mg/dl and group-2 patients with LDL cholesterol > 70 mg/dl. Meticulous history was taken and detailed clinical examination was done and recorded in predesigned structured questionnaire. Demographic data like Age, Gender, height, body weight were recorded. Risk factor profile for CAD, Smoking Hypertension, Diabetes Mellitus, Family history of coronary artery disease (CAD) were noted. Routine laboratory investigations like Hb level, RBS, serum creatinine, serum electrolytes and screening blood tests were measured. Pulse and BP were recorded both before and after intervention. 12 lead resting ECG was done before sending the patient to cath laboratory and one hour after the procedure. Troponin I value was measured by Immulite 1000 troponin I (Siemens medical solutions diagnostics, Los Angeles, USA) in the morning before the procedure. Patient with normal Troponin I (< 0.2 ng/ml) was selected for the study. The troponin kit used had a cut off value of ≥0.2 ng/ml for diagnosis of myocardial injury. Premedication was done by operator choice i.e loading doses of aspirin 300mg, clopidogrel 300mg, atorvastatin 40mg. Procedural variables i.e number of vessels involvement, procedural duration, amount of dye used, any use of eptifibatide, number of used stents, peri-procedural arrhythmia, peri-procedural cardiogenic shock, were noted. PCI was carried out in an artery having ≥70% guided by ischaemia protocol. During PCI procedure ACT was maintained between 250-350s. Low molecular weight heparin was continued for 48 hours after the procedure. Following PCI procedure patient was brought to CCU for 24 hours and monitored for any arrhythmia or cardiogenic shock. Blood sample was collected for troponin-I, 6 hours after but within 24 hours of PCI. Analysis was conducted on SPSS 23.0 for windows software (SPSS Inc., Chicago, IL, USA, 2015).Nature of the data were explored. Quantitative data was expressed as mean and standard deviation and comparison was done by “student t” test. Qualitative data was expressed as frequency and percentage and comparison was done by Chi-square(x2) Test and Fisher’s Exact Test. Logistic regression analysis was done (both univariate and multivariate). To see the correlation between LDL levels and myocardial injury, Pearson’s Correlation Test was done. Value of $p<0.05$ was considered statistically significant. Observations are expressed as tables and bar diagram. The study protocol was approved by ethical review committee of NICVD. Confidentiality regarding all information and records was maintained strictly and the patients had the right to withdraw him/herself from the study at any time during the study period.

Results:

The general objective of the study was to evaluate the association between pre-procedural LDL cholesterol and myocardial injury in patients undergoing elective PCI. The findings were shown below:

Table-I

Comparison of age distribution of the study patients

Age group (years)	Group-1(n=76)		Group-2(n=94)		Total (n=170)		p- value
	Number	%	Number	%	Number	%	
20 – 29	1	1.3	0	0.0	1	0.6	
30 – 39	7	9.2	11	11.7	18	10.6	
40 – 49	24	31.6	21	22.3	45	26.5	
50 – 59	21	27.6	37	39.4	58	34.1	
60 – 69	19	25.0	19	20.2	38	22.4	
70 – 79	3	3.9	5	5.3	8	4.7	
e”80	1	1.3	1	1.1	2	1.2	
Mean±SD	51.7±.10.8		52.4±10.7		52.1±10.7		0.66^{ns}

Unpaired t-test was done.
ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.

The above table illustrates age distribution among the study patients. Most of the patients were in the age range of 50-59 years of 27.6% in group-1 and 39.4% in group-2. The following highest age range was 40-49 years of 31.6% in group-1 and 22.3% in group-2. The mean age of Group-I was 51.7±.10.8 years and Group-II was 52.4±10.7 years. Analysis displays statistically no significant (p =0.66) mean age difference between the study groups. The mean age of the total patients was 52.1±10.7 years.

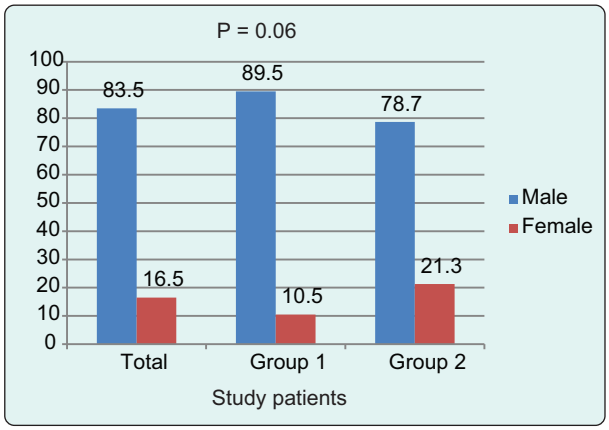

Fig.-1: *Bar diagram showing gender distribution of the study patients.*
Chi-square test was done.
ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.

Table-II

Comparison of risk factors among the study patients

Risk factors	Group-1 (n=76)		Group-2 (n=94)		Total (n=170)		p- value
	Number	%	Number	%	Number	%	
Smoker	27	35.5	38	40.4	65	38.2	0.51^{ns}
Hypertension	24	31.6	26	27.7	50	29.4	0.58^{ns}
Diabetes mellitus	27	35.5	38	40.4	65	38.2	0.51^{ns}
F/H of CAD	8	10.5	7	7.4	15	8.8	0.48^{ns}
BMI kg/m(mean±SD)	26.0±3.7		25.7±2.4		25.9±3.0		*0.51^{ns}

Chi-square test and *unpaired-t test were done.
Ns= not significant (p>0.05)
Group-1: Patients with LDL cholesterol d”70mg/dl
Group –2: Patients with LDL cholesterol >70mg/dl

The above table shows that all characteristics of risk factors had almost identical in group-1 compared to group-2 with no statistical significant difference (p>0.05).

Table-III

Comparison of study patients by diagnosis

Diagnosis	Group-1(n=76)		Group-2(n=94)		Total (n=170)		p- value
	Number	%	Number	%	Number	%	
CSA	38	50.0	42	44.7	80	47.1	0.49^{ns}
OMI	38	50.0	52	55.3	90	52.9	0.51^{ns}

Chi-square test was done.
ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.
Patients having CSA had higher in group-1 compare to group-2 (50% vs. 44.7%). On the contrary, OMI old patients had more in group-2 than group-1 (55.3% vs. 50%). There was no significant difference in terms of diagnosis (p=0.49) between the groups.

Table-IV

Comparison of study patients by Left Ventricular Ejection Fraction

LVEF in %	Group-1(n=76)		Group-2(n=94)		Total (n=170)		p- value
	Number	%	Number	%	Number	%	
Reduced EF(<40%)	12	15.8	20	21.3	32	18.8	0.36^{ns}
Mid Range EF(40-50)	40	52.6	46	48.9	86	50.6	0.63^{ns}
Preserved EF (>50%)	24	31.6	28	29.8	52	30.6	0.81^{ns}

Chi-square test was done.
Ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.
The above table indicates that left ventricular ejection fraction between the two groups was almost similar with no significant difference (p>0.05).

Table-V

Comparison of biochemical status of the study patients

Variables	Group-1 (n=76) mean±SD	Group-2 (n=94) mean±SD	p value
Total cholesterol mg/dl	129.4±20.7	181.9±53.3	<0.001^{s}
Triglyceride mg/dl	142.7±84.7	178.6±77.7	0.005^{s}
HDL mg/dl	41.5±24.1	39.2±10.2	0.42^{ns}
LDL mg/dl	62.1±8.2	122.3±30.2	

s= significant (p<0.05) and ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.

The above biochemical characteristics were higher in group-2 than group-1 with significant difference (p<0.05) except HDL cholesterol. HDL was found higher in group-1 than group-2 with no significant difference (p=0.42).

Table-VI

Comparison of procedural characteristics among the study patients

Variables	Group-1(n=76)		Group-2(n=94)		Total (n=170)		p- value
	Number	%	Number	%	Number	%	
Vessel involvement							
SVD	54	71.1	61	64.9	115	67.6	0.39^{ns}
DVD	22	28.9	33	35.1	55	32.4	
Procedural duration							
d"60 minute	50	65.8	60	63.8	110	64.7	0.79^{ns}
>60 minute	26	34.2	34	36.2	60	35.3	
No. of stent used							
Single stent	50	65.8	61	64.9	111	65.3	0.90^{ns}
Double	26	34.2	33	35.1	59	34.7	
Amount of Dye used							
d"300 ml	52	68.4	69	73.4	121	71.2	0.48^{ns}
>300 ml	24	31.6	25	26.6	49	28.8	
Use of Eptifibatide							
Yes	10	13.2	10	10.6	20	11.8	0.61^{ns}
No	66	86.8	84	89.4	150	88.2	

Chi-square test was done.
ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.
The above table indicates that CAG characteristics had almost identical in group I compared to group II with no statistical significant association (p>0.05).

Table-VII

Comparison of peri-procedural complications among the study patients

Variables	Group-1(n=76)		Group-2(n=94)		Total (n=170)		p- value
	Number	%	Number	%	Number	%	
Arrhythmia	4	5.3	10	10.6	14	8.2	0.20^{ns}
Cardiogenic shock	4	5.3	9	9.6	13	7.6	0.29^{ns}

Fisher's Exact test was done.
ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group –2: Patients with LDL cholesterol >70mg/dl.
Among the post procedural characteristics arrhythmia was insignificantly occurred in group II patients in compared to group I patients with p value 0.20. Cardiogenic shock was occurred in group II in compared to group I with statistically insignificant association (p=0.29).

Table-VIII

Association between pre-procedural LDL-C and myocardial injury among the study population

Study population	Group-1 =(LDL-C ≤70mg/dl)	Group-2 (LDL-C >70mg/dl)	P –value
Troponin I Positive (≥0.2)	15(19.7)	39(41.5)	<0.002^{s}
Troponin I Negative (<0.2)	61(80.3)	55(58.5)	
Troponin I (ng/dl)	0.37±0.16	2.52±1.50	*0.001^{s}

Paired t-test and *Chi-square test were done.
s= significant (p<0.05) and ns= not significant (p>0.05).
Group-1: Patients with LDL cholesterol ≤70mg/dl.
Group-2: Patients with LDL cholesterol >70mg/dl.
Mean value of troponin I was higher in Group-2 than Group-1 with statisttically significant difference (p<0.001). Proportion of PMI is also higher in Group-2 than Group-1 which is also statistically significant (p=0.002).

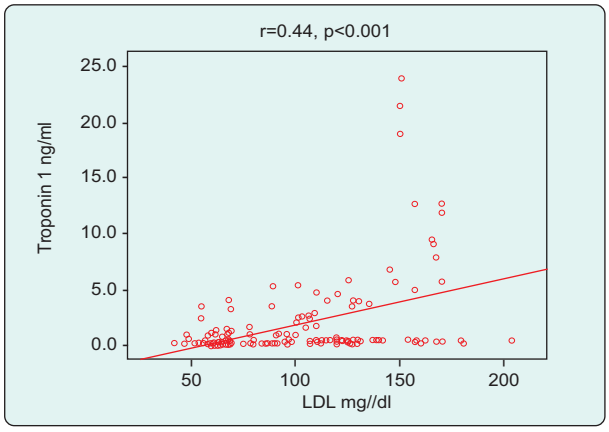

Fig.-2: *Correlation between LDL cholesterol and Troponin I ng/ml among the study patients by scatter plot diagram*

The figure shows that there is a moderate positive correlation between LDL cholesterol and Troponin I by Pearson's Correlation (r=0.44). The figure indicates that LDL level is increasing as well as Troponin I is also increasing. It was observed that the correlation statistically significant (p<0.001) by correlation t-test.

Table-IX

Univariate association of relevant risk factors with myocardial injury events

Variables of interest (Confunding Variables)	Odds Ratio (OR)	95% CI of OR	p value
Age>50 yrs	0.88	0.456 – 1.681	0.70^{ns}
Female gender	0.52	0.197 – 1.361	0.18^{ns}
Smoking	0.51	0.266 – 0.986	0.05^{ns}
Hypertension	1.77	0.836 – 3.739	0.13^{ns}
Diabetes mellitus	2.60	1.810 – 4.168	0.002^{s}
Raised BMI	0.96	0.865 – 1.066	0.44^{ns}
EF<40%	1.56	0.708 – 3.457	0.26^{ns}
OMI	2.05	1.537- 5.089	0.001^{s}
DVD	1.30	0.663 – 2.573	0.44^{ns}
Use of 2 stent	1.11	0.569 – 2.179	0.75^{ns}
Procedural duration>60 min	1.07	0.548 – 2.094	0.84^{ns}
Amount of dye>300 ml	2.62	1.813 – 3.226	0.003^{s}
Use of eptifibatide (Yes)	0.67	0.229 – 1.939	0.45^{ns}
Low HDL	0.97	0.941 – 1.011	0.14^{ns}
Elevated TG	1.23	1.234– 2.003	0.004^{s}
Elevated TC	1.18	1.135 – 1.876	0.002^{s}
Arrhythmia	0.44	0.148 – 1.337	0.14^{ns}
Cardiogenic shock	1.08	0.318 – 3.682	0.89^{ns}
Raised LDL	4.89	1.500 - 13.050	0.002^{s}

ns= Not significant (p>0.05), s= Significant (p<0.05)

The above table describes that Diabetes, previous history of MI , elevated LDL-C, TC TG, use of more dye (>300ml) during PCI procedure univariately associated to develop myocardial injury .

Table-X

Multivariate logistic regression analysis of myocardial injury events with relevant risk factors

Variables of interest	Standardized regression coefficient (β)	Odds Ratio (OR)	95% CI of OR	p value
Increased TC	-0.008	0.992	0.983 – 1.001	0.07^{ns}
Increased TG	0.000	0.999	0.995 – 1.004	0.74^{ns}
Raised LDL-C	1.549	4.71	2.072 - 10.658	<0.001^{s}
Diabetes	-0.004	0.887	0.913-1.002	0.16^{ns}
OMI	0.0224	0.998	0.998-1.009	0.15^{ns}
Use of > 300ml dye	-0.002	0.913	0.967-1.005	0.82^{ns}

ns= Not significant (p>0.05)

The above table describes sex distribution among the study patients. In group-1, 68 (89.5%) were male and 8(10.5%) were female. In group-2, 74(78.7%) were male and 20(21.3%) were female. Statistically no significant difference was found in term of sex between the groups (p=0.06). Male: Female ratio was 5.1:1. Male patients had predominant in the study.

The above table provides the binary logistic regression analysis of Odds Ratio for characteristics of the subjects likely to myocardial injury cardiac events. The above mentioned variables of interest are all entered into the model directly as confounding independent exposures for the developing of myocardial injury (dependent variable). The raised LDL-C was found to be significantly associated with myocardial injury with the ORs being 4.71. Hence it can be concluded that an odds of having myocardial injury for raised LDL-C had 4.71 times that of taget level LDL-C in this study.

Discussion:

All patients with chronic coronary syndrome undergoing PCI were considered for the study after considering inclusion and exclusion criteria during the period of July 2020 to June 2021 admitted in NICVD. Procedural characteristics were recorded during the procedure. A total number of 170 patients were included in the study. On the basis pre-procedural LDL cholesterol levels, study subjects were categorized into two groups. Patients with target LDL cholesterol level (≤70 mg/dl) were considered as Group 1 and patients with raised LDL cholesterol level (> 70mg/dl) were considered as Group 2.

The mean (±SD) age of the study population was 52.1±10.7 years ranging from 26 to 80 years, 51.7±10.8 in group 1 and 52.4 ± 10.7 in group 2. The mean age difference was not statistically significant (P= 0.66) between two groups. This finding was very close to the other relevant studies in our country[16]. Studies done by Chaowalit et al. (2007) and Mustelier et al.(2011) found that mean age was respectively 68 ± 13 years and 61.3 ± 8 years which was higher than present study probably due to longer life expectancy, geographical and racial difference[17]. In BRAVE study among 4500 cases of first MI admitted into NICVD, mean age of the patients was 53 ± 10 years[18].

The gender distribution of this study population in group-1, 68(89.5%) patients were male and 8(10.5%) were female. In group-2, 74(78.7%) were male and 20(21.3%) were female. Male female ratio was 5.1:1. No significant (p = 0.06) difference was observed between two groups. Gender distribution of this study population was not comparable to the overall population of Bangladesh because there were fewer female in this study. Male patients were predominant in both groups which correlates to study done by Herrmann et al. (2012) and de Winter et al. (2003). They also showed male predominance in their study population[19]. In almost all studies related to coronary artery disease (CAD) similar male preponderance was found[20]. As females were given less attention and access for them to the health care facilities particularly in low socioeconomic population like Bangladesh may contribute for this male predominance. Moreover, smoking as a risk factor of IHD is less common in our country among female, which may also explain male predominance of IHD. Among two groups, female was more in group-2 than group-1, though the difference was not statistically significant (p>0.05). Li, et al. (2014) also reported a higher prevalence of female gender in patients with raised LDL cholesterol[21].

Regarding risk factors, patient with raised pre-procedural LDL cholesterol had highest percentage of smoking (38.2%) and diabetes (38.2%) followed by hypertension (29.4%) and family history of premature coronary artery

disease (8.8%). Similarly patients with target level pre-procedural LDL cholesterol group smoking (35.5%) and diabetes (35.5%) were predominant followed by hypertension (31.6%) and family history of premature coronary artery disease (10.5%). Cross lab analysis found no statistically significant differences between two groups (p>0.05). Ahmed et al. had done a study on PMI and found the similar dominance of characteristics of risk factors among the subjects in our country also consistent with those found by Hermann et al. (2005)[8,10]

Proportions of different values of left ventricular ejection fraction in both group-1 and group-2 are almost similar with no significant difference (p >0.05)

Regarding stenting, single vessel stenting was done in 71.1% in group-1 and 64.9% in group-2, double vessel stenting was done in 28.9% in group-1 and 35.1% in group-2. Though proportion is more in group-2 but there was no statistical significance (p.0.05) which is consistent with those found by Li et al. (2014)[21].

In this study, during PCI procedure, number of vessel involvement, total procedural duration, extent of dye used, use of eptifibatide were assessed.. Single vessel involvement in group-1 and group-2 are 71.1% and 64.9% respectively; On the other hand, double vessel involvement are 28.9% and 35.1%.

Duration of procedure > 60 minutes (34.2% vs 36.2%). Use of more dye (>300ml) (31.6% vs 26.6%) , use of eptifibatide (13.2% vs 10.6%) were similar in both groups which were not statistically significant. This findings were consistent with study done by li et al. (2014)[21].

Regarding biochemical analysis of this study, mean value of HDL cholesterol is 41.5±24.1 and 39.2 ±10.2 in group-1 and group-2 respectively and found insignificant (p=0.42), Though mean value of total cholesterol, TG, LDL cholesterol in group-1 & group-2 were (129.4±20.7 vs 181.9±53.3), (142.7±84.7 vs 178.6±77.7), (62.1±8.2 vs 122.3±30.2) respectively and found statistically significant (p<0.001).

Following PCI, total 54(31.7%) population develop PMI . Proportion of PMI in our study is consistent with studies in our country (Ahmed et al., 2015 and Kabir et al., 2012) also some international studies like li et al., 2014 and Neinhuis et al., 2008[5,8,10,21]. In normal LDL-C level, 15(19.7%) patient develop PMI out of 76 . In raised LDL-C group , there was PMI in 39(41.5%) out of 94 cases. The mean rise of troponin I was significantly higher in group-2 than group-1 (2.52±1.50 vs 0.37±0.16, p<0.001). Also there was moderate positive linear correlation between pre-procedural LDL cholesterol level and rise of troponin I value (r=0.44) following procedure and it was statistically significant (p<0.001). Li et al. (2014) studied on 2529 patients with normal pre-procedural cardiac troponin I who was successfully underwent elective PCI, found that 142(25.5%) out of 559 patients are associated with PMI when LDL cholesterol level ≤70 mg/dl and 758 (38.5%) out of 1970 patients with LDL cholesterol level >70 mg/dl which was consistent with our study[21].

In a recent analysis, Patti et al. (2005) showed that , statin therapy reduced peri-procedural MI. This relationship suggests that LDL cholesterol may predict the risk of plaque vulnerability and distal embolization[22].

Nienhuis et al. (2008) in their meta-analysis of 15581 patients from 20 studies over a 19-year period reported the incidence of troponin release post-PCI in elective PCI to be 33.0% and increased mortality was significantly associated with troponin elevation after PCI (4.4% vs 3.3%, p=0.001;OR=1.35)[5].

Tanaka et al. (2009) in their recent meta-analysis of 15 studies incorporating 7578 patients observed that elevation of troponin-I occurred 28.7% of procedures and 14.5% patients met the new criteria for peri-procedural myocardial infarction and these patients are at high risk of further adverse events both during the hospital stay and at 18 months.

In univariate logistic regression analysis, Diabetes, previous history of MI , elevated LDL-C, TC, TG and use of more dye (>300ml) during PCI procedure were found significant for the cause of PMI. But when multivariate logistic regression analysis was done among these predictors of PCI outcome like diabetes mellitus, previous history of MI ,TG, total cholesterol and use of >300 ml during procedure; Only raised LDL-C was found as independent predictors of peri-procedural myocardial injury during PCI with OR 4.71 and p=0.001. This study findings were similar with the study done by Lin li et al. (2014)[21].

Previously very limited study was available in Bangladesh to find out the association of LDL cholesterol and myocardial injury. Results of this study established significant and independent association of pre-procedural LDL cholesterol levels with myocardial injury in patients undergone elective PCI.

Conclusion:

The present study demonstrated that pre-procedural raised LDL cholesterol in patients of Chronic coronary syndrome was associated with more incidence of

significant troponin I elevation after percutaneous coronary intervention (PCI). Pre-procedural raised LDL cholesterol may be considered as a predictor of cardiac injury in patients undergoing with elective PCI.

Limitations of study:

Although the results of this study supports the hypothesis, there are some facts to be considered which might have affected the result of the current study-

- PCI were done by various operators.
- It was a single centered study.
- Sampling was done by non-randomized sampling method.
- Sample size was small.

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Original Article

Association of Cardiac Risk Factors with Socio Demographic Profile in Young Stroke Patients in a Tertiary Care Hospital in Bangladesh. An Observation Study of 100 Patients.

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Abstract:

Background: Young people who avoid strokes live longer and have more productive lives than their senior counterparts. The prevalence of the main vascular risk factors in atherosclerosis patients rises with age, starting in early middle age, and declines beyond 70–80 years. The majority of studies on young people are small and single-center, which makes generalization challenging. Stroke risk factors differ between men and women, and vascular risk factors are more prevalent in older age groups of young adult stroke patients.

Objective: The aim of the study is to find out the association of cardiac risk factors in young patients.

Methods: This hospital-based cross-sectional study was carried out in the indoor patients in the Shaheed Suhrawardy Medical College Hospital, Dhaka, from April 2015 to October 2015. Young stroke patients (15-45 years old) admitted to the hospital were used as the study's sample size.

Results: According to this study, the majority of patients (36.0%) were between the ages of 41 and 45. The percentages of patients in the 26–30–year–old, 31–35–year–old, and 36–40–year–old age groups were fairly close to one another (18.0%, 20.0%, and 22.0%, respectively). Just 4.0% of the population was under 25. The majority of the patients (64.0%) suffered from valvular heart disease. In addition, 10.0% had ischemic heart disorders, 16.0% had myocardial infarction, and 8.0% had atrial fibrillation. More over 75.0% of stroke patients also had some type of heart illness, according to the Framingham Heart study risk calculation.

Conclusion: Young adults from Bangladesh who suffered an ischemic stroke exhibited a high incidence of known cardiac risk factors, significant sex disparities, and alarmingly rising trends with age in both sexes. Regardless of where a person lives in the nation, preventive actions must be more aggressive and customized to each individual's specific risk profile.

Keywords: *Stroke, Young patients, Cardiac diseases, Ischemic stroke, Risk Factors.*

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Introduction:

Strokes in young people (18–50 years old) are on the rise and make up 15–18% of all strokes at this time.[1,2] These young adults are particularly vulnerable to repeated strokes, with many of them considering whether or not to start families.[3] Cerebral infarction, arteriovenous malformation, cardiogenic emboli, and intracranial

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hemorrhage are all symptoms of stroke in people aged 15 to 45. The most frequent causes in young people are atherosclerosis and cardiogenic emboli. Younger people experience ischemic strokes for different reasons than older people. The most frequent causes of ischemic stroke in young people were arterial dissections and cardiac embolism.[4] Rarely do other known causes of stroke exceed atherosclerosis in the large and small arteries. Atherosclerotic cerebral infarction is predisposed by hypertension, previous TIA, and hyperlipidemia. Up to one-third of young stroke sufferers experience cardiogenic cerebral embolism. Emboli are prevented from entering the systemic circulation by the pulmonary capillary bed.[5] Paradoxical embolism occurs when venous blood crosses the pulmonary capillary bed.[7-8]

Arteriovenous malformation and hypertension are the two main causes of intracerebral hemorrhage. Aneurysms, intraventricular hemorrhage, and arteriovenous malformations are the three main causes of SAH. Users of oral contraceptives had a ninefold higher risk of stroke.[9] Cardiac arrhythmias and structural Heart diseases causean increased risk of cerebral embolism.[10-11] Patients with an undiagnosed cerebral infarction can have cardiac emboli origins found via transesophageal echocardiography.[12] Young adult strokes with ambiguous risk factors may be detected via transesophageal echocardiography and angiography. features of the etiology and predictability of stroke in young persons related to age. It is crucial to understand the risk factors for stroke in the general population in order to develop primary and secondary prevention strategies that are affordable. Younger people with ischemic stroke are more likely than older people to have a long-term effect on quality of life.[13,14] Stroke prevention in young individuals is more likely to result in more productive years and a higher level of life than in older people. In patients with ischemic stroke, the prevalence of main vascular risk factors rises with age, beginning in early middle life, and diminishes after the age of 70 to 80.[15] Most studies that focus on young people are small and represent single-center cohorts, which makes them challenging to generalize. Several research have demonstrated that the distribution of stroke risk factors differs between men and women.[16-19], indicating that vascular risk factors are more prevalent in older age groups of young adult stroke patients.[16,19–24] For instance, larger-scale research on gender and age-specific disparities are required to gather more helpful and general information for the prevention of young-onset stroke. So, this cross sectional study at Shaheed Suhrawardy Medical College will look at the association of cardiac risk factors with socio demographic profile in young individuals.

Objectives

- To find out the at the association of cardiac risk factors with socio demographic profile in young individuals.

Methodology

This was carried out in the Department of Medicine of Shaheed Suhrawardy Medical College Hospital, Dhaka, from April 2015 to October 2015 on indoor patients who were admitted to the hospital during this period. The study's sample size consisted of patients with stroke who were between the ages of 15 and 45 when they were admitted to the hospital..

Inclusion criteria-

- Stroke patients of both sex between 15-45 years.
- Patients who have given informed written consent.

Exclusion criteria-

- Patient having associated chronic infections or illness
- Patients of cancer and immunosuppressive illness.

Sample Size

Purposive sampling technique was adopted in this study. All the available subjects during the data collection period who fulfilled the study selection criteria were included in the study. As in this study purposive sampling technique was used and due to the time constraint, 100 samples were taken.

Data Collection and Analysis

A semi-structured survey was created. The questionnaire was created using the desired variables. The questionnaire asked about socio-demographics, disease features, and other information. A check list was also created. A pre-test session preceded the actual data gathering. Modifications were made before to the survey. Interviews and document reviews were used to acquire data. We examined and modified all data collected. Then the data were entered into the computer using SPSS. Chi-square was done after frequency run.

Results:

Most of the patients were between the ages of 41 and 45. (36.0 percent). The percentage of patients aged 26 to 30 years, 31 to 35 years, and 36 to 40 years was relatively similar (18.0 percent , 20.0 percent and 22.0 percent respectively). Only 4.0 percent of participants were under the age of 25.

Table-I

Distribution of the patients according to residence

Residence	No. of the patients	Percent (%)
Urban	45	45.0
Rural	55	55.0
Total	100	100.0

Majority of the patients lived in rural area (55.0%). This may be due to the fact that urban patients are usually referred to Tertiary Government Hospitals for further care.

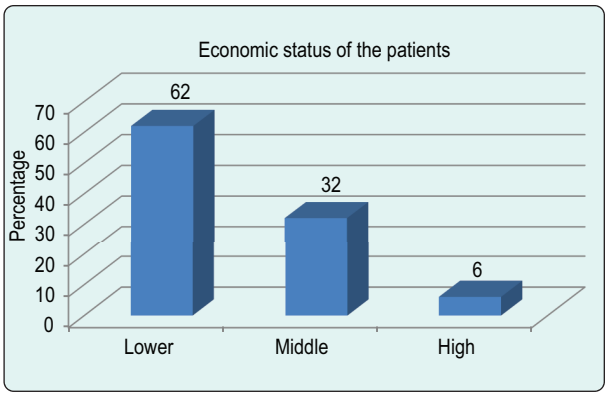

Fig.-1: *Economic status of the patients*

Among the patients majority were from lower economic status(62.0%). Less than one third were from middle class (32.0%). Only 6.0% were from high economic status.

Table-II

Distribution of patients by occupation

Occupation	No. of the patients n=100	Percent (%)
Student	16	16.0
Service	12	12.0
Business	8	8.0
Laborer	8	8.0
Farmer	6	6.0
Housewife	28	28.0
Unemployed	16	16.0
Others	6	6.0
Total	100	100.0

More than one fourth of the patients were housewife (28.0%). The proportion of student and unemployed were equal (16.0%) which were near about the proportion of service holder (12.0%). The result shows that most of the patients were house wives this could be because the stay at home and lead a sedentary life style.

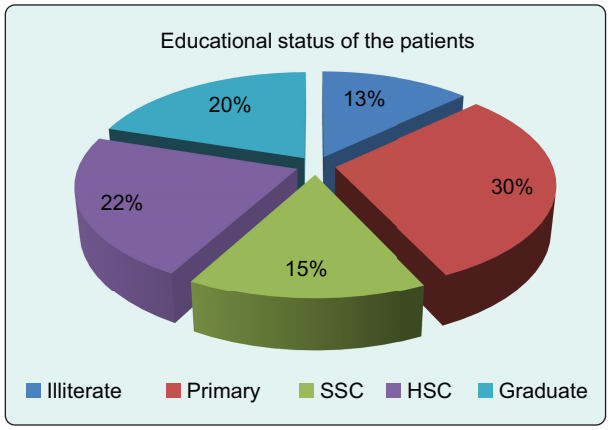

Fig.-2: *Educational status of the patients*

Among the patients the proportion of primary education level was highest (30.0%) followed by H.S.C. (22.0%) and Graduate (20.0%) patients. Fifteen percent patients were in S.S.C. level and 13.0% were illiterate. The result shows that most of the patients were educated up to primary school. It could be that these people were not aware about the causes and risk factors of stroke.

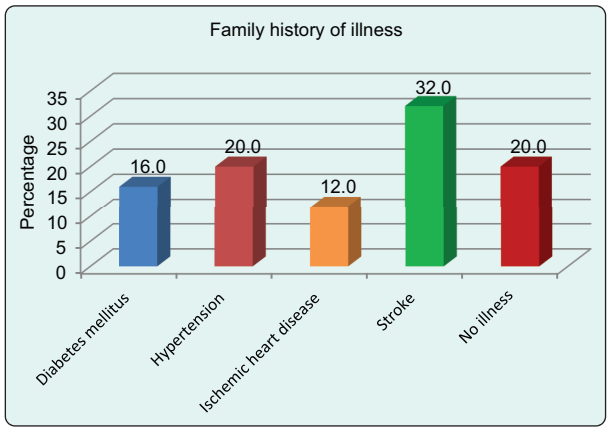

Fig.-3: *Family history of illness*

Near about one third of the patient had family history of stroke (32.0%) followed by hypertension (20.0%), diabetes mellitus (16.0%) and ischemic heart disease (12.0%). The study shows that most of the patients who have family history of stroke suffer from stroke. This could mean that people with family history of stroke should be more careful as the have higher chances of developing stroke.

Table-III

Past history of stroke and TIA (Transient Ischemic Attack)

Past History	No.of patients n=100	Percent (%)
Stroke	8	8.0
TIA	10	10.0
No illness	82	82.0

Past history of Transient ischemic attack was 10.0% and stroke was 8.0%. This table shows that the patients who had no previous history of stroke or TIA suffered stoke. This could be probably due to the fact that those who have previously suffered stroke or TIA lead a healthier life style than those who have not suffered stroke or TIA.

Table-IV

Proportion of the patients with hypertension

HTN		No. of the patients n=100	Percent (%)
Previously Known	Regularly treated	12	25.0
	Irregular/no treatment	20	41.7
Diagnosed on admission		16	33.3
Total		48	100.0

Among the patients with HTN, 66.7% was known case and 33.3% was diagnosed on admission. This shows that patients with no treatment or irregular treatment of hypertension were the highest category to suffer stroke**.**

Table-V

Proportion of different heart diseases

Diseases		No. of the patients n=100	Percent (%)
Myocardial infarction	Anterior	12	12.0
	Inferior	4	4.0
Myocardial Ischemia	Inferior ischemia	2	2.0
	Anterior ischemia	8	8.0
Valvular heart disease	Mitral stenosis	48	48.0
	Mitral stenosis with mitral regurgitation	14	14.0
	Mitral stenosis with aortic stenosis	3	3.0
Atrial fibrillation		9	9.0
Total		100	100.0

Most of the patients had ischemic type of stroke (91.0%). Only 9.0% had stroke due to intracerebral haemorrhage. In the study it was seen that the pateints who suffered stroke a greater number suffered ischaemic stroke.

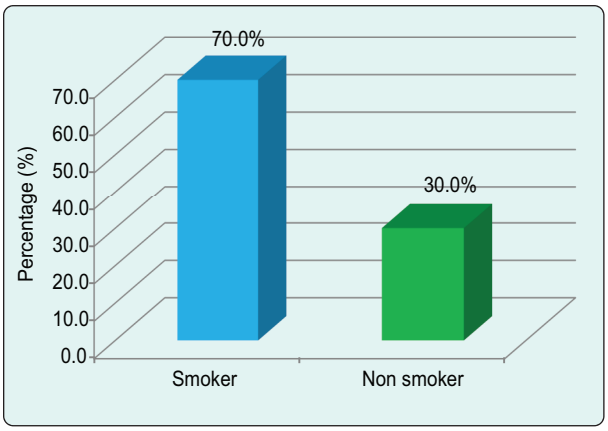

Fig.-4: *Smoking status of the patients*

Smokers comprised 70.0 percent of all patients, while non-smokers comprised the remainder (30.0 percent). A greater incidence of stroke is found among smokers compared to nonsmokers because smoking is a risk factor for atherosclerosis.

Table-VI

Proportion of oral contraceptive user in female patients

Use of contraceptive	No. of patients n=100	Percent (%)
Current contraceptive user	26	56.5
Contraceptive used previously	4	8.7
Never used	16	34.8
Total	46	100.0

Among the female patients more than half were current contraceptive user (56.5%). Only 8.7% were past contraceptive user. Incidence of stroke was higher in patients who were current contraceptive users.

Discussion

A cross sectional study was conducted to find out the relation of stroke with cardiac diseases in young patients, identify the risk factors associated with stoke in the young age group and socio demographic factors with stroke among the young stroke patients.

This study showed that among the patients majority were in 41 to 45 years age group (36.0%). The proportion of 26 to 30 years, 31 to 35 years and 36 to 40 years age group patients were very close (18.0%, 20.0% and 22.0% respectively). Only 4.0% were less than 25 years age. Majority of the patients lived in rural area (55.0%). Stroke incidence rose exponentially with increasing age.[25] in his study of stroke in young adults also found similar picture & showed that only 4% occurred in <20 years & 36% in 41-45 years. Bell D et al[26] (1990) studied 50 patients with stroke and found most of the incidence of stoke was between the ages of 50-69 years. Razzaq A A et al[27] (2002) studied in 118 young stroke patient in South Asia. About three quarters of the patients were in the 35-44 years of age. The highest incidence of stroke was between 5th to 7th decades. A study done by Chowdhury S. Z.M[28] also found peak incidence between 5th to 7th decades.

In this study more than half of the patients were male (54.0%). The present study coincides with study of Kurzke J F et al [18] where it showed that frequency of stroke is 30.0% higher in men than women. This study also differs with a previous study of Mannan & Alamgir[28], male : female ratio 4:1 and showed that stroke incidence in male is 22.0% higher than the female in South Asia.

Near about one third of the patient had family history of stroke 32.0% followed by hypertension in 20.0%, diabetes mellitus in 16.0% and ischemic heart disease in 12.0% of the patients. Past history of Transient ischemic attack was in 10.0% and stroke was in 8.0% of the patients.

Current study showed that more than two thirds of the patients were smokers (70.0%) and rests were non smoker (30.0%). Multiple individual studies have demonstrated that the risk of stroke was increased among the cigarette smokers Donan et al have shown strong association between cigarette smoking and stroke. Similar study in Copenhagen and Finland had shown increase risk of stroke in smoker. An analysis from 32 studies, found that relative risk of ischemic stroke from smoker was 1.9 times more than that of non smoker.

In this study among the female patients more than half were current contraceptive user (56.5%). Only 8.7% used contraceptives previously. There is little doubt that, regular use of estrogen is associated with increase risk of stroke. According to sex variation study with two different variable the research found that out of 24 female patients, about 58.3% patients suffered from stroke who had strong history of regular use of oral contraceptive. The use of oral contraceptive pill is associated with nine fold increase risk of cerebral infarction in women.

Conclusion

Young adults from Bangladesh who suffered an ischemic stroke exhibited a high incidence of known cardiac risk factors, significant sex disparities, and alarmingly rising trends with age in both sexes. Regardless of where a person lives in the nation, preventive actions must be more aggressive and customized to each individual's specific risk profile. It is imperative that our cross-sectional findings are replicated in a prospective study using uniform procedures.

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Case Report

First Ever Hybrid Procedure of Balloon Co-arctoplasty & Patent Ductus Arteriosus (PDA) Ligation in a Neonate in Bangladesh

Nurun Nahar Fatema[1], Md. Shaukat[2], Ashik Md. Raihan Chowdhury[3]

Abstract :

Coarctation of the aorta is a rare form of congenital heart disease, though preductal co-arctation is not very uncommon. This is a report on a 28-day-old girl who was referred to our center for evaluation. Doppler echocardiography showed severe preductal coarctation of aorta and a small-sized Patent Ductus Arteriosus (PDA). Later, it was confirmed by aortogram which showed critical coarctation of aorta (COA) with a very large Patent Ductus Arteosus (PDA). This full-term infant with symptomatic COA and a large PDA was treated successfully with a hybrid procedure of Balloon angioplasty and PDA ligation.

(Bangladesh Heart Journal 2023; 38(1): 88-91)

Introduction:

A combination of Coarctation of aorta (COA) and Patent Ductus Arteriosus (PDA) can lead to heart failure and severe symptoms, which can be manifested by shock, and subsequently other organ failure.

Aortic coarctation indicates a narrowing at some point along the course of the aorta. Preductal coarctation is one of the common type of Coarctation and circulation below the level of coarctation is maintained by patent ductus arteriosus (PDA) . A neonate or infant with severe coarctation of the aorta may present with symptoms of congestive heart failure, having been well days before when the ductus was still open[1,2]**.** Subsequently, they may develop renal failure and the feature of hypoperfusion to other organs once the ductus is closed. So, the infant becomes acutely, seriously, and even critically ill**[3].** Any neonate or infant with such critical symptoms needs urgent intervention which includes medical management like injection prostaglandin to keep the ductus open, surgical intervention, or balloon angioplasty(1). Here we are reporting a case of pre-ductal coarctation of aorta who needed balloon angioplasty and PDA ligation at 28 days of age and was cured completely.

Case Report:

A 28-day-old girl weighing 2.4 kg presented with severe respiratory distress and difficulty feeding and was referred to our center at Bangladesh Specialized Hospital (BSH), Dhaka, for evaluation. Clinical examination showed that the child was ill-looking, pale, dehydrated, dyspneic, and tachypneic, with visible bilateral chest indrawing, a clearly audible systolic murmur, and pulse of the lower limbs was reduced in comparison to the upper extremity.

Chest x-ray showed –cardiomegaly, with the plethoric lung fields and electrocardiography (ECG) showed right axis deviation with Right ventricular hypertrophy (RVH).

Echocardiography showed, severe Coarctation of aorta (COA), associated with a small sized Patent Ductus

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Arteriosus (PDA), Small Atrial Septal Defect (ASD) and severe pulmonary hypertension (PAH). So patient was planned for urgent Balloon coarctoplasty. Considering the small PDA size, PDA device closure was not planned.

After admission, the patient was stabilized over one week, her hydration was maintained by Intravenous fluid and relevant investigations were sent. Injectable antibiotics were added to cover infection. After proper premedication and aseptic precautions, patient was taken into the cath lab on 21st march 2023 for balloon coarctoplasty .

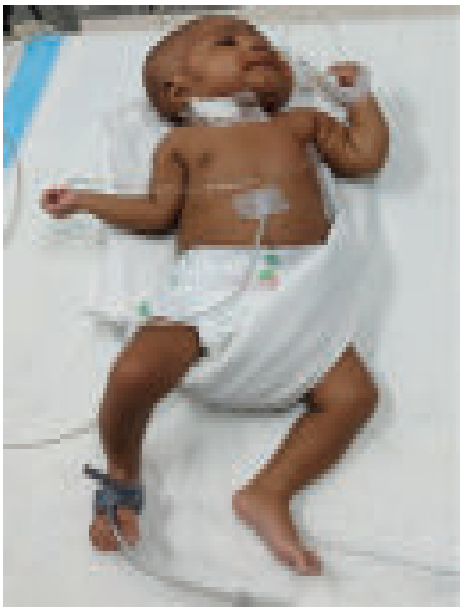

Fig.--1: *Patient A after PDA ligation*

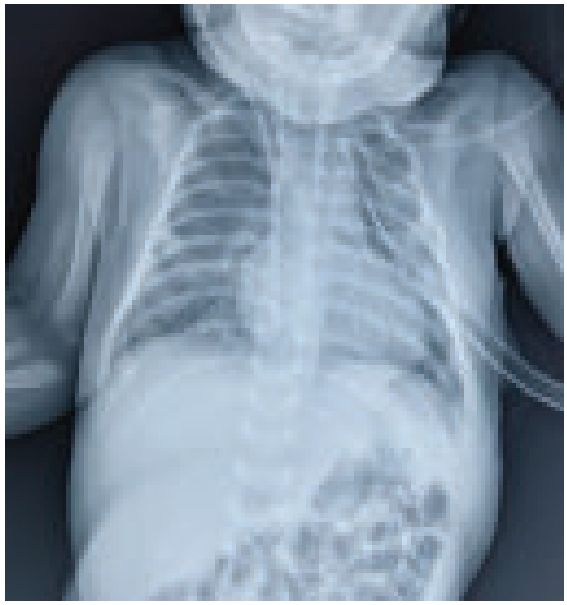

Fig.-2: *Chest X-ray showing cardiomegally*

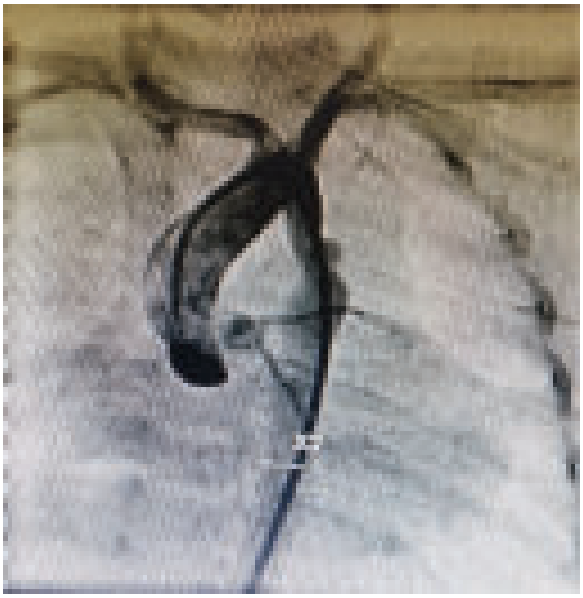

Fig.-3 *Aortogram Showing Critical COA of Aorta*

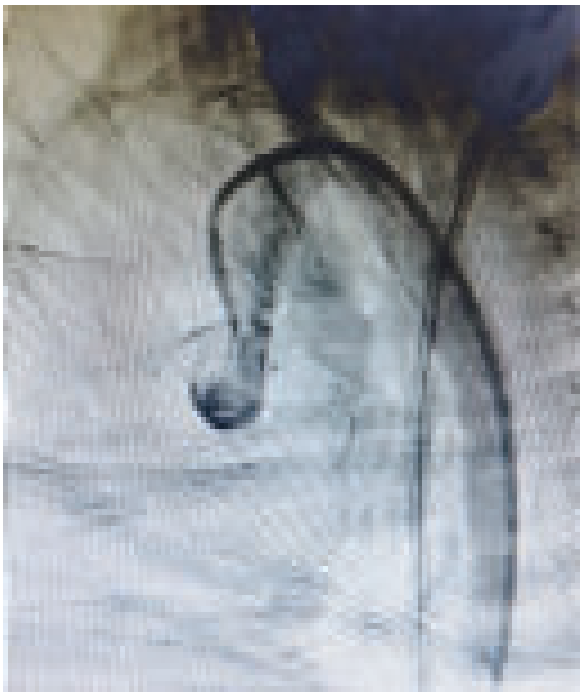

Fig.-4: *Aortogram after Balloon angioplasty showing no Coarctation.*

Procedure:

Hardwires : Standard pediatric drape, Jewsini catheter, Pigtail catheter, Bherman balloon catheter, Tyshak-mini balloon , Piccolo occluder, 4F long delivery system, exchange wire , PTCA all star wire etc.

Aortogram showed Critical coarctation of aorta . So, Balloon dilatation was performed with Tyshak mini 5x2 & followed by 8x2 balloon. Post Balloon Aortogram showed a very Large PDA out of our surprise. We tried to close the PDA subsequently with 8/6 Piccolo occluder. But PDA was larger than the device, Considering 2.4kg baby weight , passing a delivery sheath of 6F size through RFV/RFA was avoided.

As the Patient developed tachypnea and respiratory distress on the procedure table, we decided to refer the case to the cardiac surgeon. Doing PDA ligation on a 2.4kg baby was difficult but the case was accepted by our surgical team. PDA ligation was performed on next day; PDA size was equal to aorta as mentioned in surgical note.

During the postoperative period, the patient tolerated the procedure well and was on ventilator support till the next morning. Patient developed left lung collapse but recovered with Chest Physiotherapy (CPT) and gradually stepped down to CPAP then to headbox O2 supply to Room air . After Surgery improvements were also noted in the heart rate (from 150–170 beats/min to 120 beats/min), as well as in the respiratory rate (from 70 to 40 breaths/min) . There was no chest indrawing so breastfeeding was started. The patient was discharged on the 5th postoperative day.

Discussion:

The management of patients presenting with coarctation in neonates is revolutionized by the invention of prostaglandin E and its use to maintain and restore the patency of the ductus[1,2,3]. Neonates presenting with preductal coarctation have heart failure, shock, and deteriorating renal function, which could be reversed by maintaining the lower body circulation through the ductus[1,2,3,4,5]. Any neonate or infant presented with shock within the first few weeks of life and in whom lower limb pulses are absent, it should be consider to start prostaglandin along with other resuscitative maneuvers until expert assistance is available[1,2].

Coarctation of aorta can occur as an isolated defect or in association with a patent ductus arteriosus (PDA), It can be a discrete or long segment defect associated with a variable degree of hypoplasia of the isthmus or transverse arch[1]. In the present case, coarctation was discrete and preductal in location . The indications for balloon angioplasty of the coarctation of aorta are 1. Native or recurrent obstruction with a gradient of >20 mmHg. 2. Coarctation where there is left ventricular hypertrophy or systemic hypertension. But in neonate pre ductal significant coarctation is a medical emergency and should be treated immediately[1-5].

In neonates and infants < 1 year of age with native coarctation surgical resection and repair is recommended(1-5). Transcatheter therapy is the treatment of choice in >1 year age with a well-developed isthmus. Balloon angioplasty is one of the modalities of transcatheter treatment[6-7]. A balloon, 2-3 times the diameter of the coarctation segment but not exceeding the diameter of the adjacent arch proximal to the narrowed segment is selected and inflated across the coarctation site [8-11].

In this case, coarctation had a pinhole opening only and the diameter was 3 mm. So, gradual dilatation of the coarctation area was performed with 5x2 & followed by up to 8x2 Tyshak mini balloon. Stent implantation in the coarctation area is another modality of treatment for older children, adolescents, and adults. Published reports of balloon angioplasty demonstrate that this procedure results in short-term effective relief of gradient in 75-90% of patients and low mortality of 0.7-02.5%[12-14]. Long-term follow-up revealed re-stenosis in 25-36% cases[15-18]. VACA (Valvuloplasty and Angioplasty for Congenital Anomalies) registry data reported suboptimal outcomes in 19% of native and 25% of recurrent lesions[18]. The study was conducted among 970 patients from 25 centers. The major drawback of angioplasty alone is the recoil of the vessel wall with recurrence of stenosis [(19)]. Balloon angioplasty may cause aortic wall dissection in 1-4% of patients and aneurysm formation in 4-11.5% of patients [(19-20)] . Another study reports that the immediate gradient reduction was similar in both surgery and angioplasty case [(21)] . But in our country, since 1998 most of the surgeons refused to do a surgical repair of coarctation on neonates and infants. So balloon angioplasty was performed as a life-saving intervention and the excellent outcome encouraged us to take it as first-choice therapy.

Conclusion

We treated a full-term infant with symptomatic COA and a Large PDA successfully with a Hybrid procedure, Balloon angioplasty and Surgical PDA ligation . Thus, this hybrid approach might be a useful treatment option for Critical COA and Large PDA in country where surgical treatment is not available in most of the centers. °

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