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Official Journal of Bangladesh Cardiac Society
We gratefully acknowledge the contribution of the Reviewers of this issue of Bangladesh Heart Journal.
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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). Number of 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.

Case Reports:
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: JPEG 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 up to 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 are appropriate, 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.

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.

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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.


   More than six authors:


2. **Organization as author**

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

4. **Volume with supplement**

5. **Issue with supplement**

6. **Type of article indicated as needed**


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

**Books and Other Monographs**

1. **Personal author(s)**

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

3. **Organization(s) as author**

4. **Chapter in a book**

5. **Conference proceedings**

6. **Dissertation or thesis**

**Other Published Material**

**Newspaper article**

Unpublished Material
In press or Forthcoming


Electronic Material
1. Journal article on the Internet

Article published electronically ahead of the print version:

2. Article with document number in place of traditional pagination:

Article with a Digital Object Identifier (DOI):

3. Monograph on the Internet

3. Homepage/Web site

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 JPEG 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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Professor of Cardiology
Room No. 458, Block B, Anwer Khan Medical College House No. 17, Road No 8, Dhanmondi, Dhaka 1205 Bangladesh.

Papers can also be submitted via the email using the following address:
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Hypertension is a common medical problem encountered in clinical practice in developed, as well as, in developing countries, affecting both urban and rural population. It is one of the leading causes of the cardiovascular complications like myocardial infarction, heart failure and stroke. Moreover, clustering of other risk factors of ischemic heart diseases is associated with hypertension. Almost 1/3rd of end-stage renal disease requiring hemodialysis are due to hypertensive renal diseases. Different clinical trials showed that control of hypertension by life style modification and pharmacological interventions reduce the risk of complications like stroke, myocardial infarction and heart failure. In ASCOT-BPLA trial, amlodipine and perindopril provide mortality and morbidity benefit as compared to beta-blockers and diuretics and this trial influenced the modification of many national and international guidelines. Four drugs like angiotensin converting enzyme inhibitor (ACEi), calcium channel blocker (CCB), angiotensin receptor blocker (ARB) and diuretic are brought in the front line and beta-blocker has been advocated in case of compelling ground. In ADVANCE trial, combination of perindopril and indapamide showed the benefits of blood pressure reduction along with reduction of both microvascular and macrovascular events in diabetic hypertensive patients.

Clinicians know lot about antihypertensive drugs but face problem at what blood pressure threshold, treatment should be started to reach a target blood pressure level as goal of treatment for the clinical benefit of patients. The problem of relationship of J point has also barred the doctors for taking therapeutic measures aggressively because the level of blood pressure achieved at certain point of treatment is not beneficial rather increase the cardiovascular events. Finding of Syst-Eur trial differ with the J- shaped phenomena regarding too much lowering the blood pressure.

Since, 1977, different guidelines have been put forward from different societies and many munities. Recommendation of JNC-7 regarding definition and classification was widely accepted and practiced for more than one era. In 2014, experts panel members appointed to the Eighth Joint National Committee came out with an evidence based guideline for the management of high blood pressure in adults. Nine recommendations were given based on scientific evidence from randomized controlled trial or their own expert opinion. Patients at 60 years or older should have goal blood pressure less than 150/90 mmHg and those aging 30 to 59 years should have blood pressure less than 140/90 mmHg. The same thresholds and goals are recommended for hypertensive adults of less than 60 years having diabetes or non-diabetic chronic kidney diseases.

SPRINT trial was done in hypertensive patients with high cardiovascular risk, elderly patients ged > 75 years, CKD patients, patients with preexisting sub clinical or clinical cardiovascular disease, those with Framingham 10 years cardiovascular risk score of 15% or above, but all were without diabetes. This trial reports considerable reduction of primary outcomes including heart failure by 38%, death from cardiovascular cause by 25%, myocardial infarction by 17%, stroke by 11%, and non-MI acute coronary syndrome by 0%. In this trial, when target blood pressure had been less than 120 mmHg, risk of death from all cause was by 27%. SPRINT trial uses automated office BP measurement that correlates more with home BP measurement than with auscultatory office BP measurement. Intensive BP control reduces the risk of heart failure due to more use of diuretics that drives the beneficial effect i.e. primary composite end point. Hypertension should be early detected by office BP measurement compared and confirmed by out-office BP measurement. Office BP measurement is used for high risk patient like elderly patient without orthostatic hypotension, proteinuric chronic kidney disease, eGFR < 60, patient with cardiovascular diseases risk of
Framingham score more than 15%. Beneficial effect of the SPRINT trial could not be comparable with that of ACCORD trial as SPRINT study did not include diabetic patient. SPRINT research group reported serious adverse events like syncope, injurious fall, bradycardia, but hypotension, electrolyte imbalance, and acute kidney injury or acute renal failure were more concerning in intensive treatment group.

In diabetic patient systolic BP target should be < 140 mmHg but for patient with or without diabetes plus protein-creatinine ratio more than 500 mg/gm. (albumin-creatinine ratio >300 mg/gm.) lower systolic BP target had been proposed for renal protection targeting SBP<130 mmHg.

The HOPE-3 trial showed that fixed-dose treatment with low-dose statin therapy, but not BP agents, is superior to placebo in reducing long-term cardiovascular events in an intermediate-risk population; the trial did not support aggressive lowering of blood pressure. Diastolic BP target should not be less than 70 mmHg because it raises the chance of ischemic heart events or risk of acute renal failure in diabetic patient.

Reduction of BP of in hypertensive patients is desirable up to the optimum level but appropriate measures taken by the clinician depend on one’s judgment in the perspective of the patient’s clinical condition. Results of ongoing and future therapeutic trials will determine the mode of management of hypertension in future.

Further reading:
Abstract:
Hypertension is one and the major non communicable diseases in the world and contributing significantly to the burden of cardiovascular diseases, stroke, kidney failure and premature death. The prevalence of hypertension in Bangladesh varies from 11- 20% and 7% of the death in Bangladesh are due to hypertension related disease. Only 11% of the total cases of hypertension have effective control of blood pressure. Education, social awareness, income, access to physicians may be factors for less adherences to medications and effective control of the disease.

This study was undertaken in a group of population with average to good educational, social and economic background to see the incidence of the disease, its control and associated cardiovascular and metabolic status.

599 working officials above the age of 50 years were evaluated in BIRDEM General Hospital from January 2013 to December 2013 to see the incidences of hypertension, control of blood pressure and cardiac and metabolic status of the population. Total 308 patients in the study population had hypertension and 39 were newly detected. 68.8% of the patients with hypertension have effective control of blood pressure with medications and systolic hypertension was revealed in 8.7% cases. This might be due to increase awareness of the study population related to the educational, economic and social background and this plays an important role in effective control of the disease.

Keywords: Hypertension, Ischemic Heart Disease, Diabetes, Treadmill Test.

Introduction:
Hypertension is one of the major non-communicable diseases (NCDs) in the world, which significantly contributes to the burden of cardiovascular diseases (CVDs), stroke, kidney failure, disability, and premature death.1–3 It is also identified as a global disease burden and is ranked third as a cause of disability-adjusted life-years (DALYs).4

According to World Health Organization (WHO), about 17 million deaths occur worldwide due to CVDs, of which hypertension alone accounts for 9.4 million deaths and 80 % of the CVD-related deaths occurred in the developing countries.5-7 The global prevalence of hypertension is projected to increase from 26 % in 2000 to 29.2 % by 2025 which will be approximately 29 % of the world's population. 5 Although hypertension is more prevalent in developed countries its prevalence is increasing in the low and middle-income countries (LMIC).8-1 Countries in Asia, especially Southeast Asia, are having an increasing burden of hypertension including CVDs.9-11 According to WHO, hypertension has become a significant health concern in the Asian
region, affecting more than 35% of the adult population. The two fast-growing economies, India, and China, have a huge burden of hypertension and are projected to proliferate by 2025. Bangladesh a developing country in South Asia has been experiencing an epidemiologic transition from communicable diseases to NCDs. The exact prevalence of HTN in Bangladesh is not known. Only a limited number of small-scale epidemiological studies are available. The prevalence of HTN was first reported in 1976, as 1.10%. One meta-analysis, a population-based study and a recently published survey found the prevalence of HTN as 11.3%, 18.6%, and 20.1%, respectively. 7% of deaths in Bangladesh are due to HTN-related diseases, which is equivalent to 9.6 million people of age 25 or above. Effective control of blood pressure is found in about 11% of total cases. This may be due to socioeconomic factors, including lack of awareness, high illiteracy rates, low income and difficulty in physician access. This has lead to late HTN detection and lack of compliance in HTN control through pharmacotherapy and lifestyle changes.

At the advent of the new millennium, we are really not aware of our real situation. Large-scale nation-wide survey and clinical research are needed to explore the different aspects of HTN in Bangladesh. This study is undertaken in a group of population having average to good educational, social and financial background to find out the incidence of hypertension along with the pattern of control and associated cardiovascular and metabolic status.

Materials and Methods:
This prospective observational study was conducted in BIRDEM General Hospital during a period of one year from January 2013 to December 2013. 599 Working officials of Bangladesh Bank with 50 years and above were studied during their yearly health checkup. An informed, voluntary written consent was obtained from each participant before enrolment. Detailed medical history and physical examination of the study population were entered in a data sheet. Patients with physical disability and known psychiatric illness were excluded. The population group was screened for Hypertension according to JNC-7 (systolic blood pressure \( \geq \) 140 mmHg and diastolic blood pressure \( \geq \) 90 mmHg or taking antihypertensive medications). During the course of the interview, two measurements of blood pressure on each study participant were measured with the mercury sphygmomanometer by auscultatory method. Study participants were instructed to refrain from drinking any caffeinated beverage and from smoking half an hour preceding the check up. Both blood pressure measurements were obtained after the subject was rest for at least 5 min in a seated position. All blood pressure measurements were made on the left arm of each study subject, using a cuff of appropriate size at the level of the heart. The cuff pressure was inflated 30 mmHg above the level at which the radial pulse disappeared, and then deflated slowly at the rate of about 2 mm Hg/sec. The first (appearance) and the fifth (disappearance) Korotkoff sounds were recorded as indicative of the systolic (SBP) and the diastolic blood pressure (DBP) respectively. The average of two readings of SBP and DBP were used to describe the blood pressure of the participants. In case where the two readings differed by over 10 mm of Hg, a third reading was obtained and the three measurements were averaged. The control of Blood Pressure of each individual was assessed by measuring the Blood Pressure on different settings.

Cardiac Status was evaluated by 12 Lead ECG, Exercise Tolerance Test and Colour Doppler Echocardiography. Ischemic heart disease was considered by the presence of T wave inversion and ST depression and Myocardial Infarction was considered by standard characteristics ST elevation and pathological Q wave in corresponding leads representing a wall in 12 lead ECG. In ETT Bruce Protocol in majority and Modified Bruce in selected cases were carried out to evaluate Ischemic Heart Disease with standard excepted criteria.

Body Mass Index (BMI) of the study population was calculated by measuring the body height and weight with the subject standing motionless on the weighing scale, feet about 15 cm apart and the weight equally distributed on each leg. Subjects were instructed to wear minimum outwear (as culturally appropriate) and no footwear while their weight was being measured. This population group was also assessed for Metabolic Derangement by Fasting Blood Glucose, 2 Hours after Breakfast Blood Glucose, HbA1C and fasting Lipid Profile.

Data was analyzed for mean, percentage, standard deviation, chi square test, multiple correlation and multivariate analysis, by using SPSS-12 Windows. The t-test and chi square test was done for quantitative and qualitative analysis, respectively. P-value <0.05 was considered significant.
Results:
Total 599 persons of above 50 year of age were studied from January 2013 to December 2013 in BIRDEM General Hospital. There were 533 male and 66 female. 297 (49.6%) had the age below 55 years and 302 (50.4%) had age above 55 years. The yearly income was bellow 50,000 taka in 70.78% subjects and 50,000-1,00,000 taka in 28.71% person (Table-1).

Table-I
Age, Sex and Monthly income

<table>
<thead>
<tr>
<th>Variables</th>
<th>Parameters</th>
<th>No of Subjects</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>≤55</td>
<td>297</td>
<td>49.6%</td>
</tr>
<tr>
<td></td>
<td>&gt;55</td>
<td>302</td>
<td>50.4%</td>
</tr>
<tr>
<td>Sex</td>
<td>Male</td>
<td>533</td>
<td>89.0%</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>66</td>
<td>11.0%</td>
</tr>
<tr>
<td>Monthly Income</td>
<td>&lt;50000</td>
<td>424</td>
<td>70.78%</td>
</tr>
<tr>
<td></td>
<td>50000-100000</td>
<td>172</td>
<td>28.71%</td>
</tr>
<tr>
<td></td>
<td>&gt;1000000</td>
<td>3</td>
<td>0.5%</td>
</tr>
</tbody>
</table>

In this study population 308 (51.4%) subjects were found to have Hypertension of which only 30 (9.7%) were newly detected and 278 were previously known case of the hypertension. 68.8% were taking drug regularly and had effective control of Blood Pressure, 12.6% person had no control and only 8.7% had Systolic Hypertension (Table-2).

Table-II
Incidence of Hypertension.

<table>
<thead>
<tr>
<th>Number of population with HTN</th>
<th>308 (51.3%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newly detected HTN</td>
<td>30 (9.7%)</td>
</tr>
<tr>
<td>Known case of HTN</td>
<td>278 (90.3%)</td>
</tr>
<tr>
<td>Controlled HTN</td>
<td>212 (68.8%)</td>
</tr>
<tr>
<td>Uncontrolled HTN</td>
<td>39 (12.6%)</td>
</tr>
<tr>
<td>Systolic HTN</td>
<td>27 (8.7%)</td>
</tr>
</tbody>
</table>

EGC evidence of Ischemic Heart Disease was found in 97 cases of which 17 patients had the evidence of old Myocardial Infarction (Table-3)

Table-III
ECG evidence of ischemic heart disease (97)

<table>
<thead>
<tr>
<th>ECG</th>
<th>With HTN</th>
<th>Without HTN</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial Ischemia</td>
<td>47</td>
<td>33</td>
<td>80</td>
</tr>
<tr>
<td>Old MI</td>
<td>15</td>
<td>02</td>
<td>17</td>
</tr>
</tbody>
</table>

ETT were done in all patients. Symptom limited ETT were done in patients having ECG evidence of Myocardial Ischemia or Infarction. ETT was found positive in 208 out of 502 patients with negative ECG for Ischemic Heart Disease. In ETT positive patients 120 had hypertension (Table-4).

Table-IV
Relation between ECG and ETT

<table>
<thead>
<tr>
<th>ECG</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>ETT</td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>350</td>
</tr>
<tr>
<td>Positive</td>
<td>152</td>
</tr>
<tr>
<td>Total</td>
<td>502</td>
</tr>
</tbody>
</table>

Transthoracic 2D Echocardiography with Color Doppler study were carried out in all patients and regional and global wall motion abnormality were seen in only 16 cases and diastolic dysfunction was present in 127 cases (Table-6).

Table-V
Echocardiography findings

<table>
<thead>
<tr>
<th>Echocardiography</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>RWMA</td>
<td>14</td>
</tr>
<tr>
<td>GWMA</td>
<td>02</td>
</tr>
<tr>
<td>Diastolic Dysfunction</td>
<td>127</td>
</tr>
</tbody>
</table>

470 out of 599 study population had some form glycaemic abnormality. Diabetes Mellitus Type-2 were found in 238 (39.73%), 113 (18.86%) had IGT and 119 (19.86%) had IFG. 129 patients has normal Glycaemic Status.

It was revealed from BMI assessment that 48.4% of the study population was overweight and 12.8% had obesity of different grades (Table-6).
All patients were evaluated for lipid status in fasting state. 326 (54.4%) had raised LDL, 361 (61.3%) low HDL and 289 (48.2%) had raised Triglycerides. Status of the Lipid Profile in the study population is shown in the following bar diagram.

![Bar diagram showing lipid profile](image)

**Fig.-2: Pattern of lipid profile**

### Table VI

**Assessment of BMI**

<table>
<thead>
<tr>
<th>Body Mass Index</th>
<th>Under Weight (&lt;18.5)</th>
<th>Normal (18.5-24.9)</th>
<th>Over Weight (25-29.9)</th>
<th>Obesity G-I (30-34.9)</th>
<th>Obesity G-II (35-39.9)</th>
<th>Obesity G-III (40 and &gt;)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3</td>
<td>229</td>
<td>290</td>
<td>69</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>(0.5%)</td>
<td>(38.2%)</td>
<td>(48.4%)</td>
<td>(11.5%)</td>
<td>(1%)</td>
<td>(0.3%)</td>
</tr>
</tbody>
</table>

### Table VII

**Risk Factors for IHD.**

<table>
<thead>
<tr>
<th>Factor</th>
<th>No (%)</th>
<th>P value</th>
<th>Odds Ratio (95% Class Interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family H/o IHD positive</td>
<td>29 (4.9%)</td>
<td>.003</td>
<td>59.3% (.350-1.005)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (533)</td>
<td>179 (29.9%)</td>
<td>.006</td>
<td></td>
</tr>
<tr>
<td>Female (66)</td>
<td>29 (4.8%)</td>
<td>.001</td>
<td>15.5% (.923- 2.603)</td>
</tr>
<tr>
<td>DM</td>
<td>102 (17.0%)</td>
<td>.001</td>
<td>55.4% (.394-.781)</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>42 (7.0%)</td>
<td>.002</td>
<td>48.5% (.430-.810)</td>
</tr>
<tr>
<td>Obesity-1</td>
<td>46 (7.7%)</td>
<td>.003</td>
<td>62.1% (.442-.872)</td>
</tr>
<tr>
<td>Obesity- 2</td>
<td>00 (0.0%)</td>
<td>HTN</td>
<td></td>
</tr>
<tr>
<td>Tobacco Consumption</td>
<td>50 (8.4%)</td>
<td>.506</td>
<td>33.2%</td>
</tr>
<tr>
<td>Lipids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High Cholesterol</td>
<td>54 (9.0%)</td>
<td>.003</td>
<td>96.8% (.660-1.419)</td>
</tr>
<tr>
<td>High TG</td>
<td>108 (18.0%)</td>
<td>.198</td>
<td>12.5% (.895-1.755)</td>
</tr>
<tr>
<td>High LDL</td>
<td>114(19.0%)</td>
<td>.002</td>
<td>102.4% (.730- 1.436)</td>
</tr>
<tr>
<td>Low HDL</td>
<td>126 (21.0%)</td>
<td>.001</td>
<td>98.0% (.695-1.383)</td>
</tr>
</tbody>
</table>
In table 7 risk factors for IHD were analyzed in patients who had positive ETT. There were 208 (34.72%) individuals who have evidence of IHD. Among them 179 (29.9%) (p=.006) were Male and 29 (4.8%) (p=.001) were Female. 102 (17.0%) (p=.001) had Diabetes Mellitus and 123 (20.5%) (p=.003) had Hypertension. Family history of IHD was positive among 29 (4.9%) (p=.003) individuals of IHD and 50 (8.4%) persons were smoker.

Discussion:
The health status and disease profile of human societies have historically been linked to the level of their economic and social development. With industrialization, the major causes of death and disability, in the more advanced societies, have shifted from a predominance of nutritional deficiencies and infectious diseases, to those classified as chronic diseases such as cardiovascular disease (CVD), cancer, and diabetes. High-fat diets, cigarette smoking, and sedentary lifestyles become more common along with continuous improvement of life expectancy.

Non-communicable diseases become predominant, with the highest mortality caused by atherosclerotic CVD, most frequently ischemic heart disease and athero-thrombotic stroke.

Hypertension is one of the most important preventable causes of premature morbidity and mortality and a major risk factor for ischemic and hemorrhagic stroke, myocardial infarction, heart failure, chronic kidney disease, cognitive decline and premature death. With ageing, systolic hypertension becomes a more significant problem, as a result of progressive stiffening and loss of compliance of larger arteries.

The high current burdens of non-communicable diseases (NCDs) are highlighted by the estimates provided by the Global Burden of Disease Study and in the World Health Report 1999, which indicate that these disorders together contributed to 59% of global mortality (31.7 million deaths) and 43% of the global burden of disease in 1998. Several NCDs such as cardiovascular diseases (CVD), cancers, diabetes, and chronic obstructive pulmonary disease are linked by common lifestyle determinants such as diet, physical activity, and tobacco consumption. These four disorders together contribute to about 50% of global mortality. It is estimated that 30.9% of all deaths in 1998, as well as 10.3% of the total disease related burden, in terms of disability adjusted life year loss (DALY loss) were attributable to CVD.

In this context this observational study were undertaken in a specific population group of age 50 years and above having average to good educational background, social status and income to see the incidence of hypertension along with the state of control, number of medications needed and cardiovascular & metabolic status of the patient.

In this study we had only 39 cases of newly detected hypertension and 68.8% had effective control contrary to the effective control of hypertension in only 11% of the total cases in Bangladesh. This might be due to their increase awareness which is related to their educational, social and economical background leading to good compliance to treatment. Systolic hypertension was revealed in 8.7% of the study population.

Out of 502 patients with normal ECG 152 (31.4%) had positive ETT indicating myocardial ischemia and undoubtedly ETT is a good screening tool for IHD in this group of patients.

Of total 208 ETT positive patients 123 (62.1%) had hypertension and the individual who had raised LDL cholesterol, raised total cholesterol and low HDL cholesterol are more likely to develop IHD (Table 7). Only 8.4% of the study population had the history of smoking or tobacco consumption. This may also be due to increase awareness in this group.

Almost 50% of the study populations were overweight and 39.9% had Diabetes Mellitus. 18.8% had IGT and 19.8% had IFG which are also important risk factors for macrovascular complication.

In conclusion we can draw an inference that education, social and economic status helps to increase the awareness of the patients to adhere to the medications and in other way contributes to the effective control of the disease.

This was a small study in specific group of population having particular social, educational and economic background. So this may not reflect the exact scenario of the disease. We are not really aware of the magnitude of these diseases in our population and large scale nationwide study is needed to find it out.

References:


Abstract:
Background: Electrocardiographic diagnosis of a posterior wall myocardial infarction is difficult to accomplish by the standard 12-lead ECG. Early detection of posterior wall involvement in an inferior myocardial infarction is of paramount importance for the therapeutic outcome. The aim of this study is to assess the role of ST segment elevation in posterior wall leads (V₇, V₈, V₉) on the admission ECG of acute inferior myocardial infarction, for the diagnosis of posterior wall myocardial infarction and the identification of infarct related artery as well as in-hospital outcome following thrombolysis.

Methods: A total of 90 patients with acute inferior MI were enrolled by purposive sampling. On the basis of ST segment elevation in posterior leads (V₇, V₈, V₉), study subjects were categorized into two groups: 45 patients of acute inferior MI with ST segment elevation in posterior leads as group I and 45 patients of acute inferior MI without ST segment elevation in posterior leads as group II. Coronary angiography was done during index hospital admission. Interpretation of coronary angiogram was done by visual estimation by two cardiologists to assess the severity of coronary artery disease. Severity of coronary stenosis was graded according to the number of major epicardial vessel with significant stenosis by vessel score and Friesinger score. After CAG, patients were evaluated for in hospital adverse outcome like heart block, cardiogenic shock, arrhythmia, and death.

Results: Patients of PMI and non PMI groups were similar in terms of age and sex. Smoking and dyslipidemia (p=0.05) were significantly higher in PMI group. Mean RBS and Troponin-I difference were significantly (p<0.05) higher in group I. Majority of patients had ejection fraction 45-55% in both groups. Patients in group I showed more normal LVEF, than group II, which was statistically significant. This study provided the evidence that the ST segment elevation in posterior leads associated with more left circumflex (LCX) and posterior left ventricular brass (PLVB) involvement. Majority of the patients had vessel score 2, Friesinger score 5-10 in group I and vessel score 1, Friesinger score 1-4 in group II.

Conclusion: ST segment elevation in posterior chest leads (V₇, V₈, V₉) were associated with more in-hospital adverse outcome than those who had inferior MI alone. This group of patients had more PLVB involvement. Recording of posterior precordial leads appear to be beneficial for risk stratification and to locate the site of lesion in patients admitted with acute inferior myocardial infarction. Since it is inexpensive method, it may be used in any hospital.

Key words: Myocardial Infarction, ECG, Thrombolytic Therapy, Angiography

Introduction
The presentation of acute myocardial infarction is different depending on the coronary artery involved. Inferior wall myocardial infarction results from either right coronary (RCA) or left circumflex coronary artery (LCX) occlusion. Electrocardiographic diagnosis of a posterior wall myocardial infarction is difficult to accomplish by the standard 12-lead ECG, especially during the acute phase. Although an infarction involving the posterior wall...
might occur as an isolated event, it is more often associated with an inferior myocardial infarction. 2

Diagnosis of a posterior wall infarction in the acute setting is typically based on the ECG detection of ST segment depression in leads V₁ to V₃. 3 However, these changes are relatively insensitive and not specific for the diagnosis of an acute posterolateral infarction, since they may also represent inferoseptal infarction, anterior ischemia or non-Q wave myocardial infarction. 4 Taking into account the fact that the benefit of thrombolytic therapy is proportional to the amount of jeopardized myocardium, it becomes obvious that the early detection of posterior wall involvement in an inferior myocardial infarction is of paramount importance for the therapeutic outcome. Studies have shown that posterior ECG leads (V₇, V₈, V₉) can identify patients with posterior wall infarction. 5

Acute PMI has been reported to represent 15-20% of acute myocardial infarction – the vast majority occurring with acute infarction of the inferior or lateral wall of the left ventricle. The additional lead ECG, using left posterior chest leads, has increased the rate of isolated PMI diagnosis from “very rare” to a 3-11% range among all patients with AMI. 6 Rapid recognition of acute posterior myocardial infarction is of clinical importance for several reasons. Firstly, patients with acute inferior or lateral wall myocardial infarction who also have posterior involvement are experiencing a large sized infarct. Secondly, the use of acute therapies including treatments aimed at urgent revascularization may benefit patients with acute infero-posterior myocardial infarction, more than patients with an isolated infarct of a single wall. Lastly, isolated, acute PMI, if not clinically recognized as a transmural infarction, likely will not receive appropriate therapy, including thrombolytic agent or urgent angioplasty. 6

Materials and method:
This prospective observational study was conducted in the department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh. Objective of the study was to evaluate in-hospital outcome and angiographic findings in acute inferior myocardial infarction with ST segment elevation in posterior leads (V₇, V₈, V₉) following thrombolytic therapy. Considering inclusion and exclusion criteria 90 patient of acute inferior myocardial infarction with or without ST segment elevation in posterior leads (V₇, V₈, V₉) treated with thrombolytic therapy who subsequently underwent coronary angiogram were included in this study by purposive sampling. 45 patients were in group I with acute inferior myocardial infarction with ST-segment elevation in (V₇, V₈, V₉) leads and 45 patients were in group II with acute inferior myocardial infarction without ST-segment elevation in (V₇, V₈, V₉) leads. 12 lead ECG with right precordial and posterior leads (V₇, V₈, V₉). Within index hospital admission, the enrolled patients underwent coronary angiography (CAG), and coronary artery lesions were correlated with ECG findings. CAG was analysed by visual estimation. Angiographic severity of coronary artery disease was assessed by Vessel score and Friesinger score.

Vessel Score: This is number of vessels with a significant stenosis (for left main coronary artery 50% or greater and for others 70% or greater reduction in luminal diameter). Left main coronary artery will be scored as single vessel disease. 7

Score 0 = no vessel involvement.
Score 1 = single vessel involvement.
Score 2 = double vessel involvement.
Score 3 = triple vessel involvement.

Friesinger score: Friesinger index is a score ranges from 0 to 15. Each of the three main coronary arteries is scored separately from 0 to 5. 8

Score 0: no arteriographic abnormality.
Score 1: trivial irregularities (lesion from 1-29%).
Score 2: localized 30-68% luminal narrowing.
Score 3: Multiple 30-68% luminal narrowing of same vessel.
Score 4: 69-100% luminal narrowing without 100% occlusion of proximal segments.
Score 5: Total obstruction of proximal segment of a vessel.

Statistical methods:
The collected data were checked and coded manually and then entered into computer. The numerical data obtained from the study were analyzed and significance of difference were estimated by using statistical methods.

The data obtained were expressed in frequency, percentage, and mean ± standard deviation as applicable. Comparison between groups was done by chi-square test, Student’s test or others as applicable. Computer based SPSS (Statistical package for social science) program was used for data analysis. All p values of < 0.05 were accepted as statistically significant.

Results
Mean age was found 51.64±8.28 and 51.22±9.11 years in group I and II respectively (p>0.05). it was observed that in group I, maximum 19(42.2%) patients age
belonged to 41-50 years and in group II 17(37.8%) patients age belonged to 51-60 years. Male were predominant in both groups, 40(88.9%) in group I and 39(86.7%) in group II. It was observed that chest pain, shortness of breath, syncope, nausea, vomiting and sweating had 86.7% vs. 77.8%, 48.9% vs. 53.3%, 7% vs. 2.2%, 24.4% vs. 20.0%, 26.7% vs. 24.4% and 60.0% vs. 60.0% in group I and group II respectively. Back pain was significantly higher (55.6%) in group I than group II (6.7%). Regarding risk factors smoking, HTN, DM, dyslipidaemia and obesity was 33(73.3%) vs. 19(42.2%), 20(44.4%) vs.19(42.2%), 20(44.4%) vs. 24(53.3%), 32(71.1%) vs. 19(42.2%) and 5(11.1%) vs. 2 (4.4%) in group I and II respectively. Smoking and Dyslipidemia difference was statistically significant (p<0.05). Mean RBS, creatinine and Troponin I was found 11.17±3.87 vs. 9.57±3.6 mmol/L, 1.04±0.17 vs. 1.07±0.26 mg/dl and 55.16±48.97 vs. 28.8±29.54 ng/ml in group I and II respectively. Mean RBS and Troponin-I difference was statistically significant (p<0.05) between two groups. Majorit y patients had ejection fraction 45-54 in both groups, which was 26(57.8%) in group I and 25(55.6%) in group II. Normal wall motion was found 15(33.3%) in group I and 9(20.0%) in group II. Inferior wall motion abnormality more marked, 38 (84.4%) in group II than 12(26.7%) in group I. While, Inferior and posterior wall motion abnormality more marked, in group I, 18(40.0%) than group II, 4(8.9%). Mitral regurgitation was found 12(26.7%) in group I and 5(11.1%) in group II. Ejection fraction, and wall motion abnormality were statistically significant (p<0.05) between two groups (Table I).

In group I majority 17(37.8%) patients had normal RCA and in group II 31(68.9%) patients had mid RCA. The difference was statistically significant (p<0.05). Majority patients had normal LAD in both groups, which was 26(57.8%) in group I and 24(53.3%) in group II. The difference was not statistically significant (p>0.05). In group I, majority 29(64.4%) patients had mid LCX and in group II 27(60.0%) patients had normal LCX. The difference was statistically significant (p<0.05). PLVB more involved in group I 34(75.5%) than group II 15 (13.3%) (Table II). In group I, 71.1% patients had right coronary dominant vessel, 17.8% had left coronary dominant vessel and 11.1% had co-dominant vessel. In group II, most patients (95.5%) also had right coronary dominant vessel, 4.4% had co-dominant vessel (Table III).

In group I majority 22(48.9%) patients had vessel score 2 and in group II 25(55.6%) patients had vessel score 1. In group I majority 22(48.9%) patients had 5-10(moderate) friesinger score and in group II 23(51.1%) patients had 1-4 (mild) friesinger score. The difference was statistically significant (p>0.05) (Table IV). Regarding patent IRA of the study patients it was observed that in RCA, TIMI (2, 3) was found 39 in group I and 22 in group II, it also found in LCX, TIMI (2,3) was found 34 in group I and 43 in group II. This signifies that patent coronary artery more involved in group I than group II. So group I is more benefited from thrombolytic therapy (Table V). More than two third (66.7%) patients had complication in group I and 10(22.2%) patients in group II. No complication patients was found 15(33.3%) in group I and 35(77.8%)
in group II. Among complication, post infarct angina was found 8(17.8%) in group I and 3(6.7%) in group II. Majority (6.7%) patients had A/V block in group II and 2(4.4%) in group I. Re-infarction was found 2(4.4%) in group I but not found in group II. Atrial fibrillation (AF) was found 6(13.3%) in group I and 2(4.4%) in group II. Killip II heart failure was 7(15.6%) in group I and 2(4.4%) in group II. Cardiogenic shock was 5(11.1%) in group I and 1(2.2%) in group II. Complication and no complication difference was statistically significant (p<0.05), as both arrhythmias, heart failure were significantly higher in group I than in group II (Table VI).

### Table-II

**Distribution by site of coronary artery lesion (n=90)**

<table>
<thead>
<tr>
<th>Site</th>
<th>Group I(n=45)</th>
<th>Group II(n=45)</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>RCA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>17</td>
<td>37.8</td>
<td>5</td>
</tr>
<tr>
<td>Proximal</td>
<td>8</td>
<td>17.8</td>
<td>1</td>
</tr>
<tr>
<td>Mid</td>
<td>15</td>
<td>33.3</td>
<td>31</td>
</tr>
<tr>
<td>Distal</td>
<td>5</td>
<td>11.1</td>
<td>8</td>
</tr>
<tr>
<td>LAD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>26</td>
<td>57.8</td>
<td>24</td>
</tr>
<tr>
<td>Proximal</td>
<td>8</td>
<td>17.8</td>
<td>8</td>
</tr>
<tr>
<td>Mid</td>
<td>11</td>
<td>24.4</td>
<td>12</td>
</tr>
<tr>
<td>Distal</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>LCX</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>7</td>
<td>15.6</td>
<td>27</td>
</tr>
<tr>
<td>Proximal</td>
<td>2</td>
<td>4.4</td>
<td>9</td>
</tr>
<tr>
<td>Mid</td>
<td>29</td>
<td>64.4</td>
<td>9</td>
</tr>
<tr>
<td>Distal</td>
<td>7</td>
<td>15.6</td>
<td>0</td>
</tr>
<tr>
<td>OM</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Table-III

**Distribution of coronary dominance between two groups (n=90)**

<table>
<thead>
<tr>
<th>Dominant Vessel</th>
<th>Group I(n=45)</th>
<th>Group II(n=45)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Right dominant</td>
<td>32</td>
<td>71.1</td>
</tr>
<tr>
<td>Left dominant</td>
<td>8</td>
<td>17.8</td>
</tr>
<tr>
<td>Co-dominant</td>
<td>5</td>
<td>11.1</td>
</tr>
</tbody>
</table>

### Table-IV

**Comparison of coronary angiographic severity between two groups (n=90)**

<table>
<thead>
<tr>
<th>Coronary angiographic severity</th>
<th>Group I(n=45)</th>
<th>Group II(n=45)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vessel score</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Score 0</td>
<td>4</td>
<td>8.9</td>
<td>5</td>
</tr>
<tr>
<td>Score 1</td>
<td>15</td>
<td>33.3</td>
<td>25</td>
</tr>
<tr>
<td>Score 2</td>
<td>22</td>
<td>48.9</td>
<td>12</td>
</tr>
<tr>
<td>Score 3</td>
<td>4</td>
<td>8.9</td>
<td>3</td>
</tr>
<tr>
<td>Friesinger score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 ( Normal)</td>
<td>3</td>
<td>6.7</td>
<td>3</td>
</tr>
<tr>
<td>1-4 (Mild)</td>
<td>13</td>
<td>28.9</td>
<td>23</td>
</tr>
<tr>
<td>5-10 (Moderate)</td>
<td>22</td>
<td>48.9</td>
<td>14</td>
</tr>
<tr>
<td>11-15 (Severe)</td>
<td>7</td>
<td>15.6</td>
<td>5</td>
</tr>
</tbody>
</table>
Discussion:
The mean age was found 51.64±8.28 years in group I and 51.22±9.11 years in group II. Maximum (42.2%) patients were in 5th decade in group I and most (37.8%) of the group II patients were in 6th decade. However, no statistical significant mean age difference was found between two groups of patients (p>0.05). Similarly, another study showed the mean age was found 50±8 years in group I and 53±5 years in group II, which is closely resembled with the current study.

Among the studied patients, male were predominant in both groups, 88.9% in group I and 86.7% in group II and male to female ratio was 7.2:1, which is consistent with the result of a study where the percentage of male patient were 73.68% and 84.6% in group I and group II respectively.

The important risk factors in studied patients were, history of smoking (73.3%) in group I and (42.2%) in group II, followed by hypertension (44.4%) and (42.2%) in group I and group II respectively. DM was found (44.4%) in group I and (53.3%) in group II. Dyslipidemia was found (71.1%) and (42.2%) in group I and group II respectively. Family history of IHD was found (26.7%) in group I and (22.2%) in group II. Obesity was found (11.1%) in group II. Contraceptives was found (2.2%) in group II but not found in group I. Smoking and Dyslipidemia difference were significantly (p<0.05) higher in group I but others risk factor were almost similar between two groups. Studies done by others also reported similar data.

In this study it is found that, extensive MI presenting with ST segment elevation in the posterior leads were associated with more favourable effect from thrombolytic

<table>
<thead>
<tr>
<th>Table-V</th>
<th>Distribution according to patent infarct related artery (IRA) (n=90)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patent IRA</td>
<td>TIMI 0,1 (n=45)</td>
</tr>
<tr>
<td></td>
<td>Group I</td>
</tr>
<tr>
<td>RCA</td>
<td>06</td>
</tr>
<tr>
<td>LCX</td>
<td>11</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table-VI</th>
<th>Distribution of in-hospital outcome (n=90)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital outcome</td>
<td>Group I (n=45)</td>
</tr>
<tr>
<td>Complication</td>
<td>n</td>
</tr>
<tr>
<td>No complication</td>
<td>30</td>
</tr>
<tr>
<td>Post infarction angina</td>
<td>8</td>
</tr>
<tr>
<td>A/V block</td>
<td>3</td>
</tr>
<tr>
<td>1&lt;sup&gt;o&lt;/sup&gt; HB</td>
<td>1</td>
</tr>
<tr>
<td>2&lt;sup&gt;o&lt;/sup&gt; HB</td>
<td>0</td>
</tr>
<tr>
<td>CHB</td>
<td>2</td>
</tr>
<tr>
<td>Re-infarction</td>
<td>2</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>11</td>
</tr>
<tr>
<td>Atrial fibrillation (AF)</td>
<td>6</td>
</tr>
<tr>
<td>Ventricular tachycardia (VT)</td>
<td>3</td>
</tr>
<tr>
<td>Ventricular fibrillation (VF)</td>
<td>2</td>
</tr>
<tr>
<td>Heart failure</td>
<td>122</td>
</tr>
<tr>
<td>Killip I</td>
<td>5</td>
</tr>
<tr>
<td>Killip II</td>
<td>4</td>
</tr>
<tr>
<td>Killip III</td>
<td>1</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>5</td>
</tr>
<tr>
<td>Death</td>
<td>0</td>
</tr>
</tbody>
</table>
Coronary angiogram was performed in all study population during index hospital admission. Coronary angiographic severity was assessed by vessel score and Friesinger score. The possible vessel score ranges from zero to three vessel disease. In group I majority (48.9%) patients had vessel score 2 and in group II 55.6% patients vessel score 1. In group I majority (48.9%) patients had friesinger score 5-10 (moderate) and in group II 51.1% patients had friesinger score 1-4 (mild). Another author observed that, Friesinger score 0-4 indicated less extensive disease and Friesinger score e" 5 indicated extensive coronary atherosclerosis. The vessel score and friesinger score were statistically significant.

Regarding the patent IRA of the study patients, it was observed that in RCA, TIMI (2, 3) was found 39 patients in group I and 22 patients in group II, it also found in LCX, TIMI (2,3) was 34 patients in group I and 43 patients in group II. This signifies that patents coronary artery more involved in group I than group II. So group I is more benefited from thrombolytic therapy. Another author showed patent IRA in RCA, TIMI (2, 3) 52(54.74%) in group I and 54(83.08%) in group II. In LCX, TIMI (2,3) was found 41(43.16%) in group I and 8(12.3%) patients in group II, which support the current study.

About the hospital outcome more than two third (66.7%) of patients had complications in group I and 22.2% in group II. In this study, more than one complication developed in single patient. Another study showed 63.0% patients had complications in group I and 38.0% in group II, which is similar with the current study. Complications were significantly higher in group I, which were arrhythmias and heart failure.

In general, the 12-lead ECG is less sensitive in identifying left circumflex occlusion. Huey, et al., 1988 found that 52% of patients with acute MI from left circumflex disease did not show any ST segment elevation, while other investigators reported that acute left circumflex occlusion either does not bring about any changes at all in the standard 12-lead ECG or generates only ST depression in the precordial leads (Jacobs, et al., 2000). Scintigraphic studies showed that thallium myocardial perfusion defects in posterolateral segments are relatively specific for left circumflex occlusion (Newman, et al., 1983). Thus, posterior leads may contribute to the regional diagnosis of an acute inferior MI. 1998 study claims that an increase in posterior lead sensitivity from 57.7% to 59.7% could lead to a beneficial clinical outcome. In another study showed that the criterion of ST segment e" 0.5 mm in the 15 lead ECG (12 classic and
V₇, V₈, V₉) can improve the sensitivity of the diagnosis of acute coronary syndromes attributed to left circumflex occlusion by at least 94%.

Conclusion:
From this study, it may be concluded that patients with ST segment elevation in posterior chest leads (V₇, V₈, V₉) were associated with more in-hospital adverse outcome than those who had inferior MI alone. This group of patients had more PLVB involvement.

Recording of posterior precordial leads appear to be beneficial for risk stratification and to locate the site of lesion in patients admitted with acute inferior myocardial infarction. Since it is inexpensive method, it may be used in any hospital.

Conflict of interest: None.

References:
Role of Diabetic Dyslipidemia on Coronary Atherosclerotic Severity in Acute Coronary Syndrome

Golam Mahfuz Rabbani¹, Afzalur Rahman², Anisur Rahman Khan³, Nur Hossain⁴, Muhammad Badrul Alam⁵, Amiruzzaman Khan⁶, Khondoker Asaduzzaman⁷

Abstract:
Aims: To evaluate the association of coronary atherosclerotic severity in diabetic dyslipidemic patients of acute coronary syndrome.
Methods: This was a cross sectional comparative analytical study, done in the Department of Cardiology, Sir Salimullah Medical College Mitford Hospital and Ibrahim Cardiac Hospital & Research Institute, Dhaka, during September 2009 to August 2010.
Results: Most of the patients (57.5%) were in the age range of 40-70 years. Atherosclerotic severity in diabetic ACS patients was significantly higher with low HDL, high TC/HDL and high LDL/HDL ratio. Low HDL, high TC/HDL and high LDL/HDL ratio are indicators of the extent and severity of coronary artery disease. More frequent dyslipidemia in diabetic ACS patients were low HDL and it was about 54%.
Conclusion: Atherosclerotic severity in diabetic acute coronary syndrome patients was significantly more in dyslipidemic group than non dyslipidemic group.
Keywords: Diabetic Dyslipidemia, Acute Coronary Syndrome, Coronary Atherosclerosis

Introduction:
By the year 2025, there will be more than 300 million type 2 diabetes sufferer worldwide. This epidemic will be followed by a wave of cardiovascular disease. As because, diabetes is in fact a serious vascular disease with poor prognosis.¹

¹ Acute coronary syndrome (ACS) is one of the life-threatening manifestations of coronary artery disease, includes from unstable angina, acute myocardial infarction (non–ST elevation and ST elevation) and sudden cardiac death.²

Diabetic patients presenting with an acute coronary syndrome are more likely to have a larger culprit lesion, higher incidence of multivessel disease and left main stem disease (Morgan et al, 2004). Dyslipidemia is an abnormal amount of lipids e.g. total cholesterol e” 200 mg/dl and/or LDL-cholesterol e” 130 mg/dl and/or HDL-cholesterol d” 36 mg/dl and/or triglyceride e” 150 mg/dl) in the blood.³

³ The relationship between coronary artery disease (CAD) and diabetic dyslipidemia in acute coronary syndromes has been rarely demonstrated in clinical and epidemiological studies. The extent of CAD in subtypes of patients with DM is unknown.²

As morbidity and mortality of diabetic ACS patient is more due to severity of coronary atherosclerosis, so we set out

(Original Article) Role of Diabetic Dyslipidemia on Coronary Atherosclerotic Severity in Acute Coronary Syndrome

1. Junior Consultant, Department of Cardiology, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh.
2. Director and Professor, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh.
3. Associate Professor, Department of Cardiology, Mugda Medical College, Dhaka, Bangladesh.
4. Professor of Cardiology, Medinova Medical Services, Dhaka, Bangladesh.
5. Professor, Department of Cardiology, Sir Salimullah Medical College, Dhaka, Bangladesh.
6. Associate Professor, Department of Cardiology, Sir Salimullah Medical College, Dhaka, Bangladesh.
7. Assistant Professor, Department of Cardiology, Sir Salimullah Medical College, Dhaka, Bangladesh.

Address of Correspondence: Dr. Khondoker Asaduzzaman, Assistant Professor, Department of Cardiology, Sir Salimullah Medical College, Dhaka, Bangladesh. E-mail: asadareen@yahoo.com

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to examine if patients with dyslipidemia have more severe CAD when compared to nondyslipidemic patients.

**Patients and Methods:**
This was a cross sectional comparative analytical study, done in the Department of Cardiology, Sir Salimullah Medical College Mitford Hospital and Ibrahim Cardiac Hospital & Research Institute, Dhaka during September 2009 to August 2010. All diabetic acute coronary syndrome patients admitted in those hospitals during the specified period were included in the study considering the inclusion and exclusion criteria.

The diagnosis of ACS was made in patients who presented with retrosternal chest pain of 20 minutes or more with electrocardiographic changes (T wave inversion > 1.0mm, ST-segment depression > 1 mm and or ST-segment elevation > 1 mm) or Troponin-I levels > 0.06 ng/ml.

Type 2 diabetes was diagnosed by taking documented past history of DM, RBS >11.1 mmol/L or FBS > 7 mmol/L or Hba1c was > 7 mmol/L. Serum lipid profile (total cholesterol, LDL-cholesterol, HDL-cholesterol, and triglyceride) was measured as early as possible (within 24 hours).

When serum lipid profile showed total cholesterol > 200 mg/dl and/or LDL-cholesterol > 130 mg/dl and/or HDL-cholesterol < 36 mg/dl and/or triglyceride > 150 mg/dl then they were considered as dyslipidemic patients and if total cholesterol <200 mg/dl, LDL-Cholesterol <130 mg/dl, HDL-Cholesterol >36 mg/dl and triglyceride <150 mg/dl then they were considered as nondyslipidemic patients.

All patients underwent coronary angiography. Two criteria were considered for coronary artery disease: percentage of obstruction and number of vessels affected. Where the main artery lesion were scored as for the presence of lesions (>50%) in the proximal-3, middle -2 and distal segment-1; as well as for the degree of obstruction (<50% = 1; 50-70% = 2; >70% = 3). The secondary artery lesion were scored only as for the degree of obstruction (50-70% =1; >70% = 2). Score was also divided into mild (when score <9), moderate (when score 9 to 17) and severe (when score >17).

**Statistical Analysis:**
Data were analyzed by the SPSS version 11.5 software program. Confidence interval was set at 95% level. Results were considered to be statistically significant if p value was < 0.05, very significant if p value was <0.01, and highly significant when p value was <0.001.

**Results:**
The study was done in Cardiology Department of Sir Salimullah Medical College and Mitford Hospital, Dhaka, with the collaboration of Ibrahim Cardiac Hospital & Research Institute, Dhaka, during the period of September 2009 to August 2010. This study was carried out to identify the association of dyslipidemia with severity of coronary atherosclerosis in diabetic acute coronary syndrome patients.

A total of 123 patients were included in this study. Out of 123 patients 31 were non-dyslipidemic and 92 were dyslipidemic diabetic ACS patients. There was no significant difference (p=0.06) between the mean age of dyslipidemic (57 years) and nondyslipidemic (53 years) patients. No significant difference (p=0.104) in BMI was observed between these two groups.

Normal epicardial coronary artery and single vessel disease (SVD) were more in nondyslipidemic patients compared to dyslipidemic patients (22.6% vs 0% and 64.5% vs 14.1%). On other hand double vessel disease (DVD) and triple vessel disease (TVD) were more in dyslipidemic patients group compared to nondyslipidemic patients group. So, the difference between the two group was highly significant (p = 0.0001).

Type A, type B and type C lesions (all types) were more in dyslipidemic patients compared to nondyslipidemic patients (55.4%, 76.1%, 51.1% vs. 48.4%, 35.5%, 22.6%). However, type A lesion was not significantly different between two groups (p=0.496) but type B lesion and type C lesion were highly significant between the two groups (p=0.0001, p=0.006).

Mean percentage of stenosis in three main coronary arteries (LAD, RCA, and LCX) were significantly higher in dyslipidemic group than that of nondyslipidemic group (p=0.0001, p=0.01 and p=0.0001). No left main (LM) disease found in nondyslipidemic patients but 12% dyslipidemic patients had LM disease, So the difference was significant (p<0.05). LAD, LCX & RCA lesions were significantly more in dyslipidemic compared to nondyslipidemic patients (p<0.05). The difference were highly significant especially in LAD and RCA.

Table- II shows that mild score was more in nondyslipidemic patients compared to dyslipidemic patients (90.3% vs. 7.6%); while moderate and severe score were more in dyslipidemic compared to
nondyslipidemic patients group (65.2% vs. 6.5% and 27.2% vs. 3.2%). So the difference between the two group was highly significant (p=0.0001).

The above table (III) showed that there was negative correlation between atherosclerotic lesion score and HDL cholesterol ("r" = -0.557 and p = 0.0001), so this correlation is highly significant. Atherosclerotic lesion score and TC/HDL ratio had positive correlation ("r" = 0.444 and p = 0.0001) also the same positive correlation was existed between atherosclerotic lesion score and LDL/HDL ratio ("r" = 0.395 and p = 0.0001). So, HDL cholesterol, TC/HDL ratio and LDL/HDL ratio were highly significant. But other lipid fractions had no significant correlation.

The risk of severe atherosclerotic disease was higher in dyslipidemic patients than nondyslipidemic patients, as Odds ratio was 40.76 (>1). So, dyslipidemia was significant risk factor for severe atherosclerotic disease.

The risk of multivessel disease was higher in dyslipidemic patients than nondyslipidemic patients, as Odds ratio was 41.01 (>1). So, dyslipidemia was significant risk factor for multivessel disease.

Table-I
Atherosclerosis lesion score between non-dyslipidemic and dyslipidemic patients

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Non-dyslipidemic (n=31) Mean±SD</th>
<th>Dyslipidemic (n=92) Mean±SD</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesion score</td>
<td>4.81±4.08</td>
<td>13.98±4.37</td>
<td>-10.277</td>
<td>0.0001*</td>
</tr>
</tbody>
</table>

Data were analysed by using student’s t-test. The table (I) shows that atherosclerotic lesion score was more in dyslipidemic patients group than nondyslipidemic patients group (13.98±4.37 vs. 4.81±4.08), which was highly significant (p =0.0001).

Table-II
Atherosclerosis lesion score severity between nondyslipidemic and dyslipidemic patients

<table>
<thead>
<tr>
<th>Lesion Score</th>
<th>Nondyslipidemic</th>
<th>Dyslipidemic</th>
<th>χ² - value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
<td>(n=31) No. (%)</td>
<td>(n=92)No.(%)</td>
<td>value</td>
<td>p-value</td>
</tr>
<tr>
<td>Mild Score</td>
<td>28(90.3)</td>
<td>7(7.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate Score</td>
<td>2(6.5)</td>
<td>60(65.2)</td>
<td>77.926*</td>
<td>0.0001*</td>
</tr>
<tr>
<td>Severe Score</td>
<td>1(3.2)</td>
<td>25(27.2)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data were analyzed by using Chi-square(÷²) Test; Mild Score = Score < 9. Moderate Score = Score 9 to 17. Severe Score = Score > 17.

Table-III
Correlation between different lipid component and atherosclerotic lesion score

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Correlation Co-efficient “r”</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>-.063</td>
<td>.492 ns</td>
</tr>
<tr>
<td>HDL</td>
<td>-.557</td>
<td>.0001*</td>
</tr>
<tr>
<td>LDL</td>
<td>.004</td>
<td>.963ns</td>
</tr>
<tr>
<td>Score</td>
<td>TG</td>
<td>-.010</td>
</tr>
<tr>
<td></td>
<td>TC/HDL ratio</td>
<td>.444</td>
</tr>
<tr>
<td></td>
<td>LDL/HDL ratio</td>
<td>.395</td>
</tr>
<tr>
<td></td>
<td>NHDL ratio</td>
<td>.065</td>
</tr>
</tbody>
</table>
Discussion:
Diabetes is in epidemic proportion in our country and over half (75%) of all diabetic patients die of coronary artery disease presenting as acute coronary syndrome. Tight glycemic control have benefit on microvascular disease but it show no benefit on macrovascular disease, such as coronary artery disease. Increased incidence of hypertension, insulin resistance and adverse lipid profile in diabetic patients predisposes to coronary artery disease. An atherogenic pattern of lipoprotein changes (low HDL, high TG and small size LDL) is often present for years prior to development of fasting hyperglycemia and diagnosis of type 2 diabetes mellitus. In the presence of hyperglycaemia LDL becomes glycosylated and is poorly recognized by the low density lipoprotein receptor. It is scavenged by tissue macrophages creating the foam cell, a constituent of the atherosclerotic plaque. So, lipid status is more important in CAD of diabetic patient.

This cross-sectional analytical study revealed that dyslipidemia in diabetic patients were associated with higher risk of development of severe coronary atherosclerosis in terms of number of vessel involvement, type of lesions, percentage of obstruction and lesion score.

In this study 97 (78.86%) patients were male and 26 (21.14%) patients were female. Male female ratio was 3.73:1. Female patients comprise a small part of the present study. In Bangladesh, almost all of the study reported an overwhelming majority of male patients. This study also showed that mean age of both group was around 56 years. Hochman et al. (1988) also showed that fewer women than men had myocardial infarction (36.6 percent vs. 47.6 percent, P<0.001) and their average age was 56.

The analysis showed that most patients in this study population had dyslipidemia 74.8% (especially low HDL 53.6%). Nasir et al (2008) also showed that dyslipidemia in diabetic patient 76%, which is almost similar to my study.

In this study in general, dyslipidemic patients had more multivessel disease and complex type B and type C lesions. Left main disease was found only in dyslipidemic group. Morgan et al. showed that diabetic patients have a higher incidence of multivessel disease, left main stemdisease and complex type C lesion.

In this study, the mean levels of lipid variables did allow the discrimination between the presence and absence of multivessel coronary artery disease. The analysis of HDL showed that this variable decreased with the number of vessels affected. Patients with single-vessel disease had HDL levels of 40.65±8.51 md/dl, whereas those with multivessel disease had levels of 31.21±9.28 md/dl (p<0.0001). Several studies agree that low levels of HDL are correlated with the presence and severity of CAD. TC/HDL and LDL/HDL were higher in the multivessel groups when compared to the single vessel group (6.00 vs. 4.62 and 3.38 vs. 2.81). Similarly TC/HDL and LDL/HDL were higher in multivessel coronary artery disease, showed by Penalva et al.

In addition, the analysis of severity of coronary artery disease using the score system showed that the variable HDL, TC/HDL and LDL/HDL were also significantly correlated with the atherosclerotic lesion score. HDL had negative correlation (inversely related) with atherosclerotic lesion score. TC/HDL and LDL/HDL had positive correlation with atherosclerotic lesion score. Similar relationship with low HDL shown by Murphy et al. (2007) and positive correlation of TC/HDL and LDL/HDL with coronary lesion score were showed by Penalva et al. (2008).

Therefore, this finding reinforces the importance of the measurement of HDL, TC/HDL ratio and LDL/HDL ratio. They are indicators of extent and severity of the coronary artery disease. Penalva et al (2008) thus suggesting that the imbalance between TC and HDL levels plays a more important role in the pathophysiology of atherogenesis.

It is important to consider that the atheroprotective function of HDL is not restricted to reverse cholesterol transport, but can also transport antioxidant enzymes, break down oxidized lipid fractions, and neutralize their proinflammatory effects.

Patients of diabetic acute coronary syndrome was my study population and low HDL, high TC/HDL and LDL/HDL ratio were the markers of severe coronary artery disease. Fresco et al. reported that lowering lipid level occur 24 hours after an attack of acute coronary syndrome. So it is important to measure lipid profile at admission to know the exact value and identify patients of higher potential risk.

Conclusion and Recommendation:
Atherosclerotic severity in diabetic acute coronary syndrome (ACS) patients were significantly more in dyslipidemic group.

The findings of this study emphasize the need for assessment of the lipid profile of diabetic ACS patients at admission, so to identify patients of higher potential risk.
Multicentred broad based study in Bangladesh will help to support the findings and observations of this study.

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Comparison of Short-Term Clinical Outcome in In-Hospital Patients of ST Elevation versus Non ST Elevation Myocardial Infarction

Aparna Rahman¹, M.M. Zahurul Alam Khan², Md Zahid Alam³, Shabnam Jahan Hoque⁴, Tunaggina Afrin Khan⁵, Mohammad Shakhawat Hossain⁶

Abstract
Objective: To compare short term clinical outcome in hospital patient of ST Elevation versus Non ST Elevation Myocardial Infarction.

Methodology: This cross sectional observational study was carried out enrolling 100 subjects with ST elevation and Non ST elevation Myocardial Infarction, in the Department of Cardiology, BIRDEM General Hospital, Shahbagh, Dhaka, over a period of six months from January 2012 to June 2012.

Results: Mean age and gender difference was significant between STEMI and non-STEMI. Most common short term clinical outcome was heart failure (80.95% vs 75.68%). Atrial fibrillation was observed in (4.76% vs 3.44%), VT (2.38% vs 1.72%), cardiogenic shock (31.03% vs 17.24%), hypotension (76.19% vs 58.62%), reinfarction (2.38% vs 00%) and death (14.28% vs 5.17%) were observed among ST and Non ST elevation MI respectively. Statistical analysis revealed that all the parameters of short term outcome had significant difference except atrial fibrillation and VT.

Conclusion: It could be concluded that short term outcome were relatively worse in ST elevated MI and to be managed with all possible therapeutic modules.

Key words : STEMI, NSTEMI, Myocardial infarction

Introduction:
Acute coronary syndrome (ACS) refers to a constellation of clinical symptoms caused by acute myocardial ischemia.¹ Owing to their higher risk for cardiac death or ischemic complications, patients with ACS must be identified among the patients with non-traumatic chest symptoms presenting for emergency evaluation.², ³ In practice, the terms suspected or possible ACS are often used by medical personnel early in the process of evaluation to describe patients for whom the symptom complex is consistent with ACS but the diagnosis has not yet been conclusively established.⁴, ⁵ Patients with ACS are subdivided into two major categories; Unstable angina and acute myocardial infarction. New ST-segment elevation / new onset LBBB on the ECG is diagnostic of acute ST-elevation myocardial infarction (STE-MI),and ST segment depression, T-wave changes or no ECG abnormalities are cases of non-ST elevation MI. The term STE-ACS encompasses only STEMI.⁶-⁹

Unstable angina and NSTEMI are considered to be closely related conditions, sharing a common pathogenesis and clinical presentation but differing in severity.¹ Specifically, NSTEMI is distinguished from unstable angina by ischemia sufficiently severe in intensity and duration to cause irreversible myocardial damage (myocyte necrosis), recognized by the elevation of biomarkers of myocardial injury.¹⁰ The majority of patients with ST-segment elevation ultimately develop a Q-wave AMI (QMI), whereas a minority develops a non Q-wave AMI (Non-QMI).¹¹

Most patients with NSTEMI do not reveal a Q wave in the 12 lead electrocardiogram (ECG) and are subsequently referred to as having sustained Non-QMI; only a minority of NSTEMI patients develop a Q wave and are later diagnosed as QMI. It is important to recognize that ACS is a complex syndrome with a heterogeneous etiology.⁵

1. Senior Medical Officer, BIRDEM, Dhaka, Bangladesh.
2. Prof. of Cardiology BIRDEM, Dhaka, Bangladesh.
3. Asso. Prof. of Cardiology BIRDEM, Dhaka, Bangladesh.
4. Junior Consultant of Cardiology BIRDEM, Dhaka, Bangladesh.
5. Specialist of Cardiology, United Hospital, Dhaka, Bangladesh.
6. Senior Medical Officer, SMO BIRDEM, Dhaka, Bangladesh.
Address of Correspondence: Dr. Aparna Rahman, Senior Medical Officer, BIRDEM, Shahbag, Dhaka, Bangladesh. E-mail: aparnadr28@gmail.com

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The prognosis of patients with STEMI has improved considerably over the last decade. The introduction of new therapeutic modalities, including invasive cardiac procedures and new medications, probably play a major role in the favorable outcome of this patients.12,13 In different studies on the prognosis of STEMI versus non-STEMI have shown different results. Some studies have shown that patients with non-STEMI have a relatively better in-hospital course and a lower early mortality rate.10 Also, patients with non-STEMI have a relatively high prevalence of spontaneous infarct artery reperfusion, smaller infarct size, and relatively low in-hospital mortality, but a higher rate of post-infarction recurrent ischemic events.

Methodology:
This prospective observational study was done in the Department of Cardiology, BIRDEM General Hospital, Shahbagh, Dhaka during the period of January 2012 to June 2012 with the general objective to compare short term clinical outcome in hospital patient with ST Elevation versus non ST Elevation Myocardial Infarction. During the study period 100 consecutive subjects aged 25-75 years suffering from STEMI and non-STEMI who presented with chest discomfort, palpitation or shortness of breath with either ECG change (ST elevation / depression, T wave changes) or raised Troponin I were enrolled. Patient with chronic stable angina, unstable angina, non-cardiac chest pain, congenital or valvular cases and shortness of breath other than ischemic heart disease were excluded from our study. Study subjects were collected from admitted patient in CCU referred from emergency department and also from in-patient department of the respective disciplinewith acute coronary syndrome.

The objective of the study was discussed in details with the patients or their attendants before their decision to enroll themselves into the study. Clinical examination, laboratory tests, X-ray, ECG and Echocardiography were done and data collected. Demographic information was prospectively recorded including the subject’s age, gender, medical and clinical history, clinical examination and follow up of clinical conditions during hospital stay were assessed and study was conducted. Data were analyzed by using SPSS version 13. Categorical data were expressed as frequency and percentage and continuous data were expressed as mean ±SD. Comparison of mean between two groups were done by Students t test. The level of significance was set at 0.5.

Result:
Total 100 cases of STEMI and non-STEMI were evaluated after hospital admission of which 42 with STEMI and 58 with NSTEMI. The male female ratio 1:2 (STEMI) and 1:1.3 (NSTEMI). The mean age of STEMI and non-STEMI groups were 48.36±10.18 and 51.29±11.55 years respectively. Majority of (16% & 19%) the respondents (STEMI vs Non-STEMI) were found in the age group of 50-59. Mean age difference was significant between STEMI and non-STEMI. (Table – I)

Clinical findings of the study subjects (n=100) are shown in Table II
Tachycardia was observed in 57.14% STEMI and 62.06% non-STEMI subjects. Bradycardia was seen in 19.04% STEMI and 10.34% non-STEMI subjects. Hypotension was higher among nonSTEMI (58.62%) than STEMI (17.19%). About 38.09% subjects with STEMI and 58.62% subjects with non STEMI had edema. JVP was raised among non STEMI (44.82%) than STEMI(38.09%). Bilateral basal crepitation was observed in most (38.09%, 34.48% in STEMI and non STEMI) of the subjects. Except edema, there was significant difference in different signs between STEMI and non-STEMI.

ECG findings of the study subjects (n=100)) are shown in Table-III
ST elevation was seen in all STEMI subjects. ST depression was observed in 60.36% subjects with NSTEMI. Arrhythmia (STEMI vs non STEMI 14.28%, 5.17%) was also evident in ECG.

Echocardiographic findings of the study subjects (n=100) are shown in Table-IV
Most common Echocardiographic findings of the subjects were regional wall motion abnormalities (ST vs non ST, 100%, 68.96%). Majority (ST vs non ST, 9.52%, 27.58%) of the subjects had d<40-49% LV dysfunction. Only 19.04% in STEMI and 24.23% in non STEMI had e60% LV ejection fraction. Significant difference in findings was observed between two groups except normal LV function.

Short term clinical outcome of the study subjects(n=100) are shown in Table-V.
Most common short term clinical outcome was heart failure (ST vs non ST, 80.95% vs 75.68%). Atrial fibrillation was observed in (ST vs non ST, 4.76% vs 3.44%), VT (ST vs non ST, 2.38% vs 1.72%), cardiogenic shock (ST vs non ST, 31.03% vs 17.24%), hypotension (ST vs non ST, 76.19% vs 58.62%), reinfarction (ST vs non ST, 2.38% vs 0%) and death (ST vs non ST, 14.28% vs 5.17%) were observed among the study subjects. Statistical analysis revealed that all the parameters of short term outcome had significant difference except atrial fibrillation and VT.
### Table-I

**Age distribution of the study (n= 100)**

<table>
<thead>
<tr>
<th>Age group (Years)</th>
<th>STEMI (n=42) n (%)</th>
<th>Non-STEMI (n=58) n (%)</th>
<th>p valuen (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-39</td>
<td>06 (6)</td>
<td>11 (11)</td>
<td></td>
</tr>
<tr>
<td>40-49</td>
<td>08 (8)</td>
<td>14 (14)</td>
<td></td>
</tr>
<tr>
<td>50-59</td>
<td>16 (16)</td>
<td>19 (19)</td>
<td></td>
</tr>
<tr>
<td>60 and above</td>
<td>12 (12)</td>
<td>14 (14)</td>
<td></td>
</tr>
<tr>
<td>Mean± SD</td>
<td>48.36±10.18</td>
<td>51.29±11.55</td>
<td>0.024</td>
</tr>
<tr>
<td>Age range</td>
<td>33-68</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table-II

**Clinical findings of the study subjects (n= 100)**

<table>
<thead>
<tr>
<th>Signs</th>
<th>STEMI (n=42) n (%)</th>
<th>Non-STEMI (n=58) n (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tachycardia</td>
<td>24 (57.14)</td>
<td>36 (62.06)</td>
<td>0.001</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>08 (19.04)</td>
<td>06 (10.34)</td>
<td>0.001</td>
</tr>
<tr>
<td>Irregular</td>
<td>06 (14.28)</td>
<td>03 (05.17)</td>
<td>0.023</td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>04 (09.52)</td>
<td>07 (12.06)</td>
<td>0.034</td>
</tr>
<tr>
<td>Hypotension (SBP&lt; 90 mm of Hg)</td>
<td>32 (17.19)</td>
<td>34 (58.62)</td>
<td>0.047</td>
</tr>
<tr>
<td>Presence of edema</td>
<td>20 (47.61)</td>
<td>34 (58.62)</td>
<td>0.056</td>
</tr>
<tr>
<td>Presence of raised JVP</td>
<td>16 (38.09)</td>
<td>26 (44.82)</td>
<td>0.045</td>
</tr>
<tr>
<td>Crepitation in lung</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal zone</td>
<td>10 (23.80)</td>
<td>16 (27.58)</td>
<td>0.031</td>
</tr>
<tr>
<td>Basal and mid zones</td>
<td>16 (38.09)</td>
<td>20 (34.48)</td>
<td>0.037</td>
</tr>
<tr>
<td>Whole lung</td>
<td>06 (14.28)</td>
<td>04 (06.89)</td>
<td>0.022</td>
</tr>
</tbody>
</table>

### Table-III

**ECG findings of the study subjects (n= 100)**

<table>
<thead>
<tr>
<th>ECG findings</th>
<th>STEMI (n=42) n (%)</th>
<th>Non-STEMI (n=58) n (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST elevation</td>
<td>42 (100)</td>
<td>00</td>
<td>0.001</td>
</tr>
<tr>
<td>ST depression</td>
<td>00</td>
<td>35 (60.36)</td>
<td>0.001</td>
</tr>
<tr>
<td>Within normal limit</td>
<td>00</td>
<td>20 (34.48)</td>
<td>0.001</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>06 (14.28)</td>
<td>03 (05.17)</td>
<td>0.011</td>
</tr>
</tbody>
</table>

### Table-IV

**Echocardiographic findings of the study subjects (n= 100)**

<table>
<thead>
<tr>
<th>ECHO findings (Common)</th>
<th>STEMI (n=42) n (%)</th>
<th>Non-STEMI (n=58) n (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regional wall motion abnormality</td>
<td>42 (100)</td>
<td>40 (68.96)</td>
<td>0.001</td>
</tr>
<tr>
<td>LV function</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal ≥ 60%</td>
<td>08 (19.04)</td>
<td>14 (24.13)</td>
<td>0.648</td>
</tr>
<tr>
<td>Fair ≤ 50-59%</td>
<td>08 (19.04)</td>
<td>10 (17.28)</td>
<td>0.001</td>
</tr>
<tr>
<td>Mild ≤ 40-49%</td>
<td>04 (09.52)</td>
<td>16 (27.58)</td>
<td>0.029</td>
</tr>
<tr>
<td>Moderate ≤ 30-39%</td>
<td>10 (23.80)</td>
<td>08 (13.79)</td>
<td>0.011</td>
</tr>
<tr>
<td>Severe ≤ 30%</td>
<td>12 (28.57)</td>
<td>10 (17.28)</td>
<td>0.032</td>
</tr>
</tbody>
</table>
Discussion:
Myocardial infarction comprises a group of symptoms attributed to obstruction of the coronary arteries. The most common symptom prompting diagnosis of myocardial infarction is chest pain, often radiating of the left arm or angle of the jaw, pressure-like in character, and associated with nausea and sweating. Myocardial infarction usually consists of ST elevation myocardial infarction and non ST elevation myocardial infarction. These types are named according to the appearance of the electrocardiogram (ECG/EKG) as non-ST segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI). Both ST elevation myocardial infarction and non ST elevation myocardial infarction causes significant mortality and morbidity in acute phase as well as in chronic course of disease. With the aim to compare short term clinical outcome in hospital patient with ST Elevated versus Non ST elevated Myocardial Infarction, this present study was carried enrolling 100 subjects in the Department of Cardiology, BIRDEM General Hospital, Dhaka. The findings of the study are discussed on basis of related previous study concerning the chief objective of the study.

It was observed that mean age of STEMI and non-STEMI groups were 48.36±10.18 and 51.29±11.55 years respectively with a age range from 33 to 68 years. Majority of (16%, 19%) the respondents (STEMI vs Non-STEMI) were found in the age group of 50-59. STEMI vs Non-STEMI subjects were found in 12% and 14% cases respectively above 60 years age group. Mean age difference was significant between STEMI and non-STEMI.Burazeri et al (2007) found that mean age of the study subjects with STEMI was 59.1±8.7 years in their study. In STEMI group male female ratio (1:2). In non-STEMI group male female ratio (1:1.3). Chi-square test revealed significant difference in gender between two groups. Female predominance in our study may be due to consecutive selection of study subjects and small sample size.

Tachycardia was observed in 57.14% STEMI and 62.06% non STEMI subjects. Bradycardia was seen in 19.04% STEMI and 10.34% non STEMI subjects. Woo et al reported that cardinal sign of decreased blood flow to the heart was chest pain experienced as tightness around the chest. This was associated with shortness of breath. Some reported palpitations, anxiety or a sense of impending doom and a feeling of being acutely ill. Other studies also revealed the similar comparable sign and symptoms. Previous studies revealed potential complications included pulmonary edema and myocardial reinfarction. Our present study revealed Hypotension was higher among non STEMI (58.62%) than STEMI (17.19%). About 38.09% subjects with STEMI and 58.62% subjects with non STEMI had edema. JVP was raised among non STEMI (44.82%) than STEMI (38.09%). Bilateral basal crepitation was observed in most (38.09%, 34.48% in STEMI and non STEMI) of the subjects. Except oedema, there was significant difference in different signs between STEMI and non STEMI.

ST elevation was seen in all STEMI subjects. ST depression was observed in 60.36% subjects with NSTEMI. Arrhythmia (STEMI vs non STEMI 14.28%, 5.17%) was also evident in ECG. Most common Echocardiographic findings of the subjects were regional wall motion abnormalities (ST vs non ST, 100%, 68.96%). Majority (ST vs non ST, 9.52%, 27.58%) of the subjects had ≥40-49% LV dysfunction. Only 19.04% in STEMI and 24.23% in non STEMI had ≥60% LV dysfunction. Significant difference in findings was observed between two groups except normal LV function. Cannon et al (2002)7

| Table-V | In hospital short term clinical outcome of the study subjects |
|------------------------|------------------------|------------------------|------------------------|
| Short term clinical outcome | STEMI | Non-STEMI | p-value |
| Heart failure | 34 (80.95) | 44 (75.86) | 0.011 |
| Atrial fibrillation | 02 (4.76) | 02 (3.44) | 0.351 |
| SVT | 01 (2.38) | 00 | 0.001 |
| VT | 01 (2.38) | 01 (1.72) | 0.424 |
| VF | 02 (4.76) | 00 | 0.028 |
| Cardiogenic-shock(Defined as persistently low SBP< 90 mm of Hg with features of tissue hypoperfusion) | 18 (31.03) | 10 (17.24) | 0.001 |
| Post-infarct angina | 06 (14.28) | 02 (3.44) | 0.034 |
| Hypotension (Defined as SBP<90 mm of Hg) | 32 (76.19) | 34 (58.62) | 0.012 |
| Reinfarction | 01 (2.38) | 00 | 0.033 |
| Death | 06 (14.28) | 03 (5.17) | 0.027 |

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observed the same findings in subjects with ACS. The incidence rates of STEMI were 21%, whereas the incidence rates of NSTEMI was 32% (McManus et al 2011)\(^1\) according to ECG. Most common Echocardiographic findings of the subjects were regional wall motion abnormalities (100%). Majority (34%) of the subjects had d'41-50% LV dysfunction. Only 11% had e'56% LV dysfunction.

**Conclusion:**
From the study result it could be concluded that short term complications were relative grave in ST elevated MI. So, subjects with MI who had ST elevation should be paid extra attention during early management. However further study with a comparative prospective design is required to solve these questions.

**References:**


Abstract:
Evaluation of different morphology of premature ventricular contraction (PVC) in 12-lead ECG might reflect the presence or absence of myocardial diseases and determine PVC foci. It is important for ablation procedure and it can help in pre-procedural planning and potentially may improve ablation outcome.

Methods and Results-In this study, 12-lead Electrocardiogram (ECG) of 50 patients with or without structural cardiac diseases, who had experienced PVC, were obtained. PVC QRS duration, contour, pattern, unifocal or multifocal and different morphology in various lead were evaluated. PVC-QRS morphology of 50 ECGs showed QRS duration 140ms was 60%, >140ms was 24%, >160ms was 16%. QRS notching <40ms was 42%, >40ms was 16%, smooth contour was 42%. The morphology of PVCs in lead V1, RBBB morphology was 36%, LBBB morphology was 64%; in lead V1 & V2, high r 8%, low r 4%. QRS wave polarity in lead I negative (QS, Qr, or rS wave pattern) 28%, positive (R-wave pattern) 52%; in lead II, III, avF, positive 76%. Of these RR’ or Rr’ pattern 20%, R pattern 56%. Negative 24%. QRS transition in chest lead, 16% transition occur at V4–V5, 48% at V3-V4, 4% at V2-V3, 36% at V1-V2 level. The pattern of PVCs were bigeminy 24%, trigeminy 6%, couplet 4%, salvos 12%, R on T 2%, VT 6%. Of the 32 PVCs originating from the RVOT, 8 were classified as of free-wall origin, 24 of septal, 14 of left, 26 of right, 4 of proximal, and 2 of distal origin. Of the 6 PVCs originating from the LVOT, 4 were originated from the LVOT close to the left coronary cusp and 2 were originated from the LVOT close to the right coronary cusp. Of the 12 PVCs originated from LV fascicle, 12 of posterior fascicle origin and none from anterior fascicle origin.

Conclusion-12-lead ECG is a simple, inexpensive and noninvasive tool to detect PVCs and facilitate their localization. By evaluating morphology of PVC, we can also predict the structural and functional state of heart.

Keywords: Electrocardiogram, Cardiac Arrhythmia, Premature Ventricular Beats,
effective non-invasive diagnostic method for determining arrhythmias. The morphological features of the premature ventricular complex (PVC) have been cited as a clue to the presence or absence of underlying cardiac disease. Scherf and Schott recognized that PVCs with exceptionally wide QRS complexes frequently occurred in diseased hearts. Solloff found that PVCs with a bizarre and distorted configuration were highly suggestive of underlying myocardial disease in contrast to those with the "classic" smooth pattern. Morphology is also useful to localize the site of origin of PVCs before ablation procedure where indicated. This helps not only in pre-procedural planning, but also can potentially improve ablation outcomes.

PVCs may originate from various foci. If PVC focus is in right ventricle, it would appear as Left Bundle Branch Block (LBBB) and if it is in left ventricle, it would appear as Right Bundle Branch Block (RBBB) because in this state left ventricle would depolarize earlier. In general there are three common regions are defined for PVC foci: Right Ventricular Outflow Tract (RVOT), Left Ventricular Outflow Tract (LVOT) and Aortic Cusp (AC). Many researches for determining various divisions of idiopathic VT or PVC foci have been developed so far, including idiopathic ventricular tachycardia (IVT) consist of RVOT VT/PVC, Idiopathic Left Ventricular Tachycardia (ILVT), Idiopathic Propranolol sensitive VT (IPVT), LVOT VT/PVC[8] and AC[6]. It has been reported that 60-80% of the idiopathic tachycardia in normal hearts arise from the RVOT and 10% of them arise from LVOT[9]. RVOT VT/PVC is more common in females at age 30 to 50 years old[10]. It shows wide QRS complex and LBBB pattern with inferior axis, whereas LVOT VT/PVC usually shows RBBB morphology in lead V1 with wide monophasic R-wave in precordial leads. Morphologic explanations of ECG characteristics are useful for differentiating of VT/PVC arising from the AC region. VT/PVC originated from the left coronary cusp produces multiphasic QRS morphology with an M or W configuration in lead V1 with a preexcitation transition no later than V2. A left bundle pattern with a wide small R wave in lead V2 and a preexcitation transition usually at V3 is revealed in PVCs with a right coronary cusp origin[5].

Kamakura et al.[12] proposed the method to estimate the origin of VT/PVC from the RVOT and LVOT by using indexes obtained from 12-lead ECG. They classified PVC/VT from the RVOT into 8 subdivisions by using 3-dimensional anatomic relation: anterioposterior, right-left, and superior-inferior. The features they used for estimating the origin of PVC/VT consisted of morphology, amplitude, duration and polarity of QRS complex. To distinguish LVOT from RVOT region, they showed that R/S amplitude ratio in lead V3 is a helpful index. If the ratio of R/S amplitude in V3 is equal or higher than 1, the PVC/VT stems from LVOT zone, otherwise arises from RVOT.

The purpose of this study is to find out the various morphology and foci of PVCs using 12-lead ECG.

Materials and methods:

Data collection
12 lead ECGs of the 50 patients with or without structural cardiac disease, who had experienced PVC, were obtained. The data was collected from National Institute of Cardiovascular Diseases (NICVD) arrhythmia clinic.

PVC detection
First of all, PVCs were recognized and distinguished from the normal beats. Because of their greatness in height, depth and length, PVCs could easily be detected. They were characterized by-

- Duration of more than 120 msec
- Bizarre morphology that does not resemble usual aberration (i.e. a typical right or left bundle branch block).
- T wave in the opposite direction from the main QRS vector.
- A fully compensatory pause.

QRS duration and morphology
In this study we detected the duration, contour, pattern (bigemini, trigemini, quadrigeminy, couplet, triplet, salvoes), unifocal or multifocal and various morphology of PVCs in different leads like lead I,II,III,AVF,V1 and QRS transition in chest lead.

Presence or absence of notching in PVC was also detected. Notching in QRS complex was determined as a tri-phasic R or Q wave with an interval greater than 40 msec between the first and second peak of the QRS complex. Existence of notching is considered when notching is observed in more than three of the six limb leads. When notching occurred near the summit, the non-dominant peak was measured.

Classification of anatomical site of PVCs by 12-lead ECGs
According to the morphology of different lead of 12-lead ECG PVCs were originated from the LVOT, RVOT (free wall, septal origin, right, left side, proximal, distal origin), basal RV, LV fascicles and aortic cusp (Right and Left coronary cusp). Figure 1 shows a sample of 12-lead ECG of patients with PVC originating from RVOT septum.
Results:
The electrocardiographic characteristics of PVC-QRS morphology of 50 ECGs are shown in Table 1. QRSd ≤140ms was 60%, >140ms was 24%, >160ms was 16%. QRS notching <40ms was 42%, >40ms was 16%, smooth contour was 42%. Table II. Showed the morphology of PVCs in lead V1, V2 and lead 1 (n=50). In lead V1, RBBB morphology was 36%, LBBB morphology was 64%; in lead V1 & V2, high r 8%, low r 4%. QRS wave polarity in lead I negative (QS, Qr, or rS wave pattern) 28%, positive (R-wave pattern) 52%. Table III. Showed the morphology of PVCs in lead II, III, avF (n=50). Positive 76%. Of these RR’ or Rr’ pattern 20%, R pattern 56%. Negative24%.Table IV. Showed QRS transition in chest lead (n=50). 16% transition occur at V4 –V5, 48% at V3-V4, 4% at V2-V3, 36% at V1-V2 level. Table V. showed the pattern of PVCs (n=50). Bigeminy 24%, Trigeminy 6%, Couplet 4%, Salvos 12%, R on T 2%, VT 6%. The sites of origin of all 50 PVCs are shown in Table VI. Of the 32 PVCs originating from the RVOT, 8 were classified as of free-wall origin, 24 of septal, 14 of left, 26 of right, 4 of proximal, and 2 of distal origin. Of the 6 PVCs originating from the LVOT, 4 were originated from the LVOT close to the left coronary cusp and 2 from the LVOT close to the right coronary cusp. Of the 12 PVCs originated from LV fascicle, 12 of posterior fascicle origin and none from anterior fascicle origin.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Number</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS duration (ms)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 140</td>
<td>30</td>
<td>60%</td>
</tr>
<tr>
<td>&gt; 140</td>
<td>12</td>
<td>24%</td>
</tr>
<tr>
<td>&gt; 160</td>
<td>8</td>
<td>16%</td>
</tr>
<tr>
<td>QRS notching (ms)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 40</td>
<td>21</td>
<td>42%</td>
</tr>
<tr>
<td>&gt; 40</td>
<td>8</td>
<td>16%</td>
</tr>
<tr>
<td>Smooth contour</td>
<td>21</td>
<td>42%</td>
</tr>
</tbody>
</table>

PVC, premature ventricular complex; ECG, electrocardiogram; < 40ms =narrow notching; > 40ms = broad notching.
Table-II

Morphology of PVCs in lead V1 and lead 1 (n=50).

<table>
<thead>
<tr>
<th>Morphology in V1 &amp; V2</th>
<th>Number</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBBB</td>
<td>18</td>
<td>36</td>
</tr>
<tr>
<td>LBBB</td>
<td>32</td>
<td>64</td>
</tr>
<tr>
<td>High r</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Low r</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Morphology in 1</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>14</td>
<td>28</td>
</tr>
<tr>
<td>Positive</td>
<td>26</td>
<td>52</td>
</tr>
</tbody>
</table>

LBBB, Left bundle branch; RBBB, Right bundle branch; High r means initial r-wave amplitude 0.2 mV in both lead; Low r means r-wave amplitude, 0.2 mV in 1 or both leads.

Table-III

Morphology of PVCs in lead II, III, avF (n=50).

<table>
<thead>
<tr>
<th>Morphology in II, III, avF</th>
<th>Number</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>38</td>
<td>76</td>
</tr>
<tr>
<td>RR' or Rr' pattern</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>R pattern</td>
<td>28</td>
<td>56</td>
</tr>
<tr>
<td>Negative</td>
<td>12</td>
<td>24</td>
</tr>
</tbody>
</table>

Table-IV

QRS transition in chest lead (n=50).

<table>
<thead>
<tr>
<th>QRS transition</th>
<th>Number</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V4 – V5</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>V3 – V4</td>
<td>24</td>
<td>48</td>
</tr>
<tr>
<td>V2 – V3</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>V1 – V2</td>
<td>18</td>
<td>36</td>
</tr>
</tbody>
</table>

Table-V

Pattern of PVCs (n=50).

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Number</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bigeminy</td>
<td>12</td>
<td>24</td>
</tr>
<tr>
<td>Trigeminy</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Couplet</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Salvos</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>R on T</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>VT 3</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

Table-VI

Number of PVC origins. (n=50).

<table>
<thead>
<tr>
<th>Site of origin</th>
<th>Number</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV outflow tract</td>
<td>32</td>
<td>64.0</td>
</tr>
<tr>
<td>Free wall side</td>
<td>8</td>
<td>16.0</td>
</tr>
<tr>
<td>Septum side</td>
<td>24</td>
<td>48.0</td>
</tr>
<tr>
<td>Left side</td>
<td>14</td>
<td>28.0</td>
</tr>
<tr>
<td>Right side</td>
<td>26</td>
<td>52.0</td>
</tr>
<tr>
<td>Proximal side below PV</td>
<td>4</td>
<td>8.0</td>
</tr>
<tr>
<td>Distal side below PV</td>
<td>2</td>
<td>4.0</td>
</tr>
<tr>
<td>LV outflow tract</td>
<td>6</td>
<td>12.0</td>
</tr>
<tr>
<td>Left coronary cusp</td>
<td>4</td>
<td>8.0</td>
</tr>
<tr>
<td>Right coronary cusp</td>
<td>2</td>
<td>4.0</td>
</tr>
<tr>
<td>Fascicular PVC</td>
<td>12</td>
<td>24.0</td>
</tr>
<tr>
<td>Posterior fascicle</td>
<td>12</td>
<td>24.0</td>
</tr>
<tr>
<td>Anterior fascicle</td>
<td>0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

RV, right ventricle; LV, left ventricle; PV, pulmonary valve.

Discussion:

This study shows a method to identify PVCs and determine their duration, pattern, and morphology of PVCs in different lead of 12-lead ECG. Through which foci of PVCs can also be determined.

In our study, total 50 ECGs were evaluated. Among them QRSd d” 140ms was 60%, >140ms was 24%, >160ms was 16%. QRS notching <40ms was 42%, >40ms was 16%, smooth contour was 42%. Broad notches or shelves coupled with QRS duration >160 msec is a useful discriminator between the presence and absence of heart disease, as suggested by other studies [4, 5]. These PVCs might serve as a reliable marker for a particular structural and functional state of a nonspecifically diseased myocardium: dilated and globally hypokinetic. On the other hand, PVCs (with smooth contour or narrow notches as well as QRS duration < 160 msec) is more likely to identify patients with normal-size hearts with normal or near-normal left ventricular function despite the presence of underlying cardiac disease.

We also evaluated morphology of PVCs in different leads of 12-lead ECG, like lead I, II, III, avF, V1 and QRS transition in precordial leads. Through which location of PVCs origins can be determined.

In our study, 32 PVCs (64%) were originated from RVOT, which is consistent with previous study. RVOT PVC is associated with a characteristic ECG morphology of LBBB in lead V1 with inferior axis. The QRS duration and the QRS wave morphology in leads II and III is informative. If the QRS duration is >140 ms, the origin is likely to be on the free-wall side. If it is d”140 ms, the origin is likely to be on the septal side (diagnostic accuracy: 80%). If the RR’ or Rr’ wave pattern is observed in lead II and III, the origin is likely to be on the free-wall side. If the R-wave pattern was seen in leads II and III, the origin was likely to be on the septal side (diagnostic accuracy: 86%). QRS transition at V4-V5, origin...
is free-wall RVOT and transition at V3-V4, origin is septal RVOT. The QRS wave polarity of lead I is another useful index. If lead I showed negative polarity (QS, Qr, or Rs wave pattern), the origin is likely to be on the left side. If lead I showed positive polarity (R-wave pattern), the origin is likely to be on the right side (diagnostic accuracy: 83%). If the initial r-wave amplitude in leads V1 and V2 is high (>0.2 mV in both leads), the origin is likely to be on the proximal side. If the initial r-wave amplitude in V1 and V2 is low (<0.2 mV in one or both leads), the origin is likely to be on the distal side (diagnostic accuracy: 66%)².

LVOT PVC is suggested by RBBB morphology in lead V1 with inferior axis or LBBB morphology with inferior axis with small R-waves in V1 and early precordial transition (R/S = 1 by V2 or V3) [11]. In our study, 6 PVCs (12%) were originated from LVOT, which is also consistent with previous study [9]. Among them 4 were originated from the LVOT close to the left coronary cusp and 2 were originated from the LVOT close to the right coronary cusp. Aortic sinus cusp origin is sometimes difficult to differentiate from RVOT PVC because both are so close to each other. VT/PVC originated from the left coronary cusp produces multiphasic QRS morphology with an M or W configuration in lead V1 with a precordial transition no later than V2. A left bundle pattern with a wide small R-wave in lead V2 and a precordial transition usually at V3 is revealed in PVCs with a right coronary cusp origin⁵. The RBBB QRS configuration with a left superior axis, suggesting an exit site from the posterior fascicle and RBBB/right inferior axis, suggesting an exit site from anterior fascicle. In our study, we found of the 12 PVCs originated from LV fascicle, 12 from posterior fascicle origin and none from anterior fascicle origin.

We also found several pattern of PVCs like bigeminy, trigeminy, couplet, salvos, R on T, VT in our study.

The clinical value of studying morphology of PVCs in 12-lead ECG is a cost effective and noninvasive means of risk stratifying patients early during the initial evaluation period when used in conjunction with the history and physical examination. It can also serve to prompt additional caution when contemplating the use of drugs that significantly impair ventricular function, including certain antiarrhythmic agents. Studying morphology of PVCs in different lead of 12-lead ECG also helps determining their foci and can be important for ablation procedure and may help to improve ablation outcome.

References:
Abstract:
Like elsewhere, cardiovascular disease (CVD) is an increasingly important cause of morbidity and mortality in Bangladesh. Over the past few decades, because of epidemiological transition, the prevailing disease pattern in this country changed from predominantly communicable to predominantly non-communicable disease, CVD contributes to the latter a lot. Actually, CVD particularly coronary artery disease (CAD) is getting epidemic proportion day by day. Hypertension and heart failure are on the rise, whereas the prevalence of acute rheumatic fever is declining. However, despite some efforts, reliable data concerning various aspects of CVD is inadequate at present. The current prevalence of hypertension, CAD, rheumatic fever and rheumatic heart disease and stroke may be 20-25%, 4-6%, <1/1000, 0.3-1.0% respectively. Besides conventional risk factors for different CVD, genetic predisposition and some novel issues like high salt intake, arsenicosis, hypovitaminosis D and air pollution may play important role in the aetiopathogenesis of CVD in this population. Formulation of appropriate policy and more emphasis on preventive strategy may help combat CVD in Bangladesh.

Keywords: Coronary Artery Disease, Hypertension, Rheumatic Heart Diseases, Bangladesh.

Introduction:
CVD is a major public health problem throughout the world. It is the number one cause of morbidity and mortality world-wide. The economic impact of different types of CVD is enormous. Traditionally, Bangladesh is a developing country burdened with communicable diseases. However, like many other low-income countries in the world, she has been experiencing epidemiological transition; the prevailing disease pattern is changing from communicable diseases to non-communicable diseases (NCD). Small pox, once upon a time a regular epidemic, has been eradicated. Cholera is no longer a major threat to our health. The major causes of death in Bangladesh gradually shifted from acute infectious and parasitic diseases to NCDs. In 1986, NCDs represented only 8% of total deaths compared to 52% of deaths due to communicable diseases, whereas in 2014, NCDs are estimated to account for 59% of total deaths; CVD is the single-most important contributor, and is responsible for 17% of total mortality. Despite this paradigm shift, little is known regarding the epidemiological pattern and underlying pathophysiology of CVD in Bangladesh. Recognizing these limitations, the present review has been planned to compile the available data on this important public health issue. This review will make a basis for future research and would be a valuable source of information.

Methods:
Data was collected from the available articles searched via PubMed, Google Scholar and BanglaJOL supported by the International Network for the Availability of Scientific Publications up to December, 2016. Besides this, local journals which were not available online but recognized by the Bangladesh Medical and Dental Council were searched as well. Also, some information was collected from personal communication with responsible persons.
Epidemiology of CVD

CVD is the number one killer worldwide.\textsuperscript{5,6} According to the Heart Disease and Stroke Statistics — 2016 update by the American Heart Association, heart disease and stroke continue to be the top two killers worldwide. As of 2013, 31% of all deaths were from CVD, with 80% occurring in low- and middle-income countries; stroke accounted for 11.8% of all deaths. The burden of CVD, especially the CAD is increasing at a greater rate in South Asia than in any other region globally. The prevalence of CVD in India has been estimated to be nearly 3% in 2000, and up to 10% in recent years, indicating rising prevalence.\textsuperscript{7,8} Also, data from the Registrar-General of India shows that CVD is the top killer of Indians, accounting for 23% of all deaths in 2010-2013 as compared to 20% in 2004-2006.\textsuperscript{9} Among the NCDs, CVD is probably the most important cause of mortality and morbidity in Bangladesh. In 2014, NCDs represented 59% of the total deaths; CVD was the single-most important contributor, being responsible for 17% of the country’s deaths.\textsuperscript{4} According to the Health Bulletin 2015,\textsuperscript{10} CVD and stroke together was the topmost cause of death in Upazila, District and Medical College Hospitals, and was responsible for 17.78%, 21.83% and 16.32% deaths respectively in 2014. Also, stroke and acute myocardial infarction together was the topmost cause of admission of the indoor patients in Medical College Hospitals across the country in the same year.\textsuperscript{10}

The exact prevalence of CVD in Bangladesh is not known. Probably the first attempt to determine the prevalence of heart disease was made by Malik et al. in a survey amongst 7062 people of different age groups in Dacca City and in a village; the surgery revealed the prevalence of 2.92%.\textsuperscript{11} Self-reported prevalence of heart disease among the 25 to 64-year-old respondents were 5.3% to 66.3% in males and 7.8% to 77.7% in females in another study in 2005.\textsuperscript{12} The wide range of prevalence is presumably due to differences in study design and methodology.

The prevalence of CAD in Bangladesh has been reported to be 0.33% to 19.6% in different studies.\textsuperscript{11,13-6} A recent study from rural Bangladesh demonstrated a dramatic increase in CVD, and the age-standardized CVD mortality increased by 30-fold (from 16 deaths per 100,000 to 483 deaths per 100,000) among males and 47-fold (from 7 deaths per 100,000 to 330 deaths per 100,000) in females.\textsuperscript{1}

Like CAD, hypertension is an increasingly important medical and public health problem in Bangladesh. The reported prevalence varies widely from 1.21% to 32%,\textsuperscript{11,16,32-35} (Table 2). According to the Bangladesh NCD Risk Factor Survey 2010,\textsuperscript{27} the prevalence of hypertension is 17.9% in general, 18.5% in men and 17.3% in women. On the other hand, overall, age-standardized prevalence of prehypertension and hypertension were 27.1 and 24.4%, respectively, in a recently published analysis based on the nationwide population-based 2011 Bangladesh Demographic and Health Survey (BDHS).\textsuperscript{29} Even higher prevalence of hypertension of 40% (95% confidence interval (CI) 38-42%) was found in a population-based study involving 3096 adults aged >30 years from rural Bangladesh.\textsuperscript{33} A recently-published meta-analysis concerning risk factors for CVD in Bangladesh found the prevalence of hypertension to be 15.1%.\textsuperscript{34}

### Table-I

**Prevalence of coronary artery disease in Bangladesh.**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Place</th>
<th>Age, year</th>
<th>Diagnostic criteria</th>
<th>No. screened</th>
<th>Prevalence (%)</th>
<th>Type of study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malik A\textsuperscript{11}</td>
<td>1976</td>
<td>Urban and rural</td>
<td>15-74</td>
<td>ECG, chest X-ray</td>
<td>7062</td>
<td>0.33</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Zaman, et al.\textsuperscript{13}</td>
<td>2007</td>
<td>Rural</td>
<td>&gt;20</td>
<td>Pathological Q wave or current medication</td>
<td>447</td>
<td>3.4: male 4.6, female 2.7</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Ahsan et al.\textsuperscript{14}</td>
<td>2009</td>
<td>Urban; UGC Employee</td>
<td>Mean age 44 8</td>
<td>Not defined; ECG and echo were used</td>
<td>163</td>
<td>20.9</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Sayeed et al.\textsuperscript{15}</td>
<td>2010</td>
<td>Rural</td>
<td>≥20</td>
<td>1) H/o angina plus ECG +ve; 2) post-MI with Q or non-Q MI; 3) diagnosis by a cardiologist</td>
<td>768</td>
<td>1.85</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Parr et al.\textsuperscript{16}</td>
<td>2011</td>
<td>Urban and rural</td>
<td>&gt;25</td>
<td>Self-reported</td>
<td>8591</td>
<td>5.1; urban 6.0, rural 4.7</td>
<td>Cross sectional</td>
</tr>
</tbody>
</table>

UGC: University Grants Commission
### Table-II

**Prevalence of hypertension in Bangladesh.**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Place</th>
<th>Age, year</th>
<th>Diagnostic criteria (BP in mmHg)</th>
<th>No. screensed</th>
<th>Prevalence (%)</th>
<th>Type of study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malik A(^{11})</td>
<td>1976</td>
<td>Urban and rural</td>
<td>15-74</td>
<td>Not mentioned</td>
<td>7062</td>
<td>1.21</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Ullah W.(^{18})</td>
<td>1976</td>
<td>Rural</td>
<td>&gt;20</td>
<td>Not mentioned</td>
<td>17569</td>
<td>2.6</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Islam et al.(^{19})</td>
<td>1983</td>
<td>Rural</td>
<td></td>
<td>dBP &gt;90</td>
<td>5026</td>
<td>6.70</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Sayeed et al.(^{20})</td>
<td>1994</td>
<td>Rural</td>
<td>&gt;15</td>
<td>sBP &gt;140 and dBP &gt;90</td>
<td>1005</td>
<td>sHTN 10.5; dHTN 9.0</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Sayeed et al.(^{21})</td>
<td>2002</td>
<td>Urban and rural</td>
<td>≥20</td>
<td>sBP ≥140 and dBP ≥90</td>
<td>2361</td>
<td>sHTN 14.4; dHTN 9.1</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Sayeed et al.(^{22})</td>
<td>2003</td>
<td>Rural</td>
<td>≥20</td>
<td>Not mentioned</td>
<td>4923</td>
<td>Male 15.7; female 22.5</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Zaman et al.(^{23})</td>
<td>2004</td>
<td>Rural</td>
<td>≥20</td>
<td>sBP ≥140 + dBP ≥90 + medication</td>
<td>1271</td>
<td>17.8</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Chen et al.(^{24})</td>
<td>2006</td>
<td>Rural</td>
<td>≥18</td>
<td>sBP ≥140 or dBP ≥90 or, medication</td>
<td>11116</td>
<td>13.3</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Van Minh et al.(^{12})</td>
<td>2008</td>
<td>Rural</td>
<td>25-64</td>
<td>Self-reported</td>
<td>7153</td>
<td>HSID: 14.6; WATCH: 17.1; Matlab: 10.6</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Bangladesh NCD Risk Factor Survey 2010(^{27})</td>
<td>2010</td>
<td>Urban and rural</td>
<td>≥25</td>
<td>BP ≥140/90 or, medication</td>
<td>9,275</td>
<td>17.9</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Parr et al.(^{19})</td>
<td>2011</td>
<td>Urban and rural</td>
<td>&gt;25</td>
<td>Self-reported</td>
<td>8591</td>
<td>13.60</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Cravedi et al.(^{28})</td>
<td>2012</td>
<td>Rural</td>
<td>&gt;18</td>
<td>By clinical staff</td>
<td>1518</td>
<td>18.5</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>BDHS 2011(^{29})</td>
<td>2013</td>
<td>Urban and rural</td>
<td>≥35</td>
<td>Pre-HTN: sBP 120-139 + dBP 80-89; HTN: sBP ≥140 + dBP ≥90 or, medication</td>
<td>17,964</td>
<td>In male: Pre-HTN 27, HTN 19; In females: Pre-HTN 28, HTN 32</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Bhowmik et al.(^{30})</td>
<td>2013</td>
<td>Rural</td>
<td>≥20</td>
<td>sBP ≥140 and dBP ≥90</td>
<td>4757</td>
<td>1999: 14.3; 2004: 18.4; 2009: 14.0</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Neupane et al.(^{31})</td>
<td>2014</td>
<td>Urban and rural</td>
<td></td>
<td>Pre-HTN: sBP 120-139 + dBP 80-89; HTN: sBP ≥140 + dBP ≥90 or, medication</td>
<td>17.9</td>
<td>Meta-analysis</td>
<td></td>
</tr>
<tr>
<td>Khanam et al.(^{32})</td>
<td>2015</td>
<td>Rural</td>
<td>≥25</td>
<td>Pre-HTN: sBP 120-139 + dBP 80-89; HTN: sBP ≥140 + dBP ≥90 or, medication</td>
<td>6,094</td>
<td>Pre-HTN 31.9; HTN 16.0</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Islam et al.(^{33})</td>
<td>2016</td>
<td>Rural</td>
<td>≥30</td>
<td>sBP ≥140 and dBP ≥90 or, self-reported</td>
<td>3096</td>
<td>40</td>
<td>Cross sectional</td>
</tr>
<tr>
<td>Fatema et al.(^{34})</td>
<td>2016</td>
<td>Urban and rural</td>
<td></td>
<td>Different</td>
<td>Different</td>
<td>15.1</td>
<td>Meta-analysis</td>
</tr>
</tbody>
</table>

BP, blood pressure; sBP, systolic blood pressure; dBP, diastolic blood pressure; HTN, hypertension; sHTN, systolic hypertension; dHTN, diastolic hypertension; BDHS, Bangladesh Demographic and Health Survey; NCD, non-communicable diseases; HEALS, Health Effects of Arsenic Longitudinal Study; HSID, Health System and Infectious Disease; WATCH, Woman Abuse Tracking in Clinics and Hospitals
Rheumatic fever (RF) and rheumatic heart disease (RHD) are common CVD in Bangladesh. Data regarding the incidence and prevalence of these conditions vary widely.\textsuperscript{36-42} (Table 3). However, over the past 3 decades, there is a declining trend of acute RF in the country. However, chronic RHD continues to be an important public health problem here. Current prevalence of RF and RHD may be <1/1000.\textsuperscript{43} Recently, conventional and portable echocardiography is being used increasingly in studies concerning RF and RHD, as a result, more and more subclinical cases of RHD are being diagnosed. So, the prevalence of RF and RHD estimated so far may not be accurate, and the true prevalence of RHD may be much higher in Bangladesh as well.

Data regarding the incidence and prevalence of heart failure at the community level in Bangladesh are almost non-existing. In a hospital-based retrospective study at a tertiary cardiac hospital in Dhaka City\textsuperscript{44}, about one-seventh (1970 out of 14009) of the patients admitted between January 2005 and August 2006 had heart failure. Majority (35.79%) had CAD as the principal etiological factor, whereas hypertension was the primary risk factor for HF in 29.14% of cases. In another hospital-based study conducted in the National Institute of Cardiovascular Diseases (NICVD), Dhaka in 2009 involving 780 patients, 27.25% had heart failure.\textsuperscript{45}

Little is known regarding the incidence and prevalence of congenital heart disease (CHD) in Bangladesh. A proportion of CHD in children may remain undetected unless specific efforts are made to diagnose them. In a prospective, hospital based study conducted over January 2006 to December 2008 in the Pediatric Cardiology unit of Combined Military Hospital (CMH) Dhaka, 142 babies out of 5668 live birth had CHD, giving an incidence of 25/1000 live births. Most common CHDs were atrial septal defect (ASD, 26%), ventricular septal defect (VSD, 16.9%), patent ductus arteriosus (PDA, 18%), tetralogy of Fallot (TOF, 14%), and pulmonary stenosis (PS, 7.75%).\textsuperscript{46} Another study\textsuperscript{47} conducted in Dhaka Shishu Hospital from January 2008 to December

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Place</th>
<th>Age, years</th>
<th>Echo used or not</th>
<th>No. screened</th>
<th>Prevalence (per 1000)</th>
<th>Type of study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malik\textsuperscript{35}</td>
<td>1976</td>
<td>Urban and rural</td>
<td>Different ages</td>
<td>No</td>
<td>7062</td>
<td>7.5, combined RF and RHD</td>
<td>Community project</td>
</tr>
<tr>
<td>Ahmed et al.\textsuperscript{36}</td>
<td>1991</td>
<td>Rural</td>
<td>5-15</td>
<td>Yes, in selected cases</td>
<td>5923</td>
<td>RF 1.2; RHD 1.3</td>
<td>Community project</td>
</tr>
<tr>
<td>Haque et al.\textsuperscript{37}</td>
<td>1992</td>
<td>Urban and rural</td>
<td>5-15</td>
<td>Yes, in selected cases</td>
<td>Urban 9875, rural 5923</td>
<td>3.6, combined RF and RHD</td>
<td>School and house to house survey</td>
</tr>
<tr>
<td>Banoo et al.\textsuperscript{38}</td>
<td>1984-85</td>
<td>Urban</td>
<td>4-17</td>
<td>No</td>
<td>4349</td>
<td>RF 43.9; RHD 5.05</td>
<td>School survey</td>
</tr>
<tr>
<td>Mahmud et al.\textsuperscript{39}</td>
<td>1989</td>
<td>Urban</td>
<td>5-18</td>
<td>Yes, in selected cases</td>
<td>5011</td>
<td>RF 0.85; RHD 2.8</td>
<td>School survey</td>
</tr>
<tr>
<td>Begum et al.\textsuperscript{40}</td>
<td>1990-91</td>
<td>Urban</td>
<td>5-18</td>
<td>Yes, in selected cases</td>
<td>10538</td>
<td>RF 2.37; RHD 0.189</td>
<td>School survey</td>
</tr>
<tr>
<td>Majumder et al.\textsuperscript{41}</td>
<td>2004</td>
<td>Rural</td>
<td>5-16</td>
<td>No</td>
<td>947</td>
<td>RF 4.22; RHD 0</td>
<td>School survey</td>
</tr>
<tr>
<td>Zaman et al.\textsuperscript{42}</td>
<td>2005</td>
<td>Urban and rural</td>
<td>5-19</td>
<td>Yes</td>
<td>56827</td>
<td>RF 0.6; RHD 0.3</td>
<td>Cross-sectional survey</td>
</tr>
</tbody>
</table>

RF, rheumatic fever; RHD, rheumatic heart disease

Table-III

Prevalence of RF and RHD in Bangladesh.
2009 prospectively and from January 1998 to December 1999 retrospectively, involved subjects aging from 1st day of life to 12 years of age. Majority were acyanotic congenital heart disease (75% and 78.5% in the past and present respectively); VSD was the commonest lesion (32.7% and 26.9% respectively), followed by ASD (25.6% and 21.2% respectively). TOF was the commonest cyanotic lesion both in the past and present. VSD (42.6%) was the commonest type of congenital heart disease reported in another prospective 1-year study among the admitted children (newborn to 12 years) in the Department of Paediatrics of Rajshahi Medical College & Hospital. Other major types were TOF (18.3%), ASD (14.8%), and PDA (7.8%). A more recent retrospective study from 2010 to 2013 conducted in Sir Salimullah Medical College Hospital, Dhaka, demonstrated that out of 6520 cases of live births, 196 had CHD giving the incidence 30/1000 live births. Among the congenital heart lesions, the prevalence of ASD, VSD, PDA, TOF and transposition of great arteries (TGA) were 20.41%, 13.78%, 10.71%, 8.67% and 4.59% respectively.

At present, there are probably no available data regarding arrhythmias, cardiomyopathies and peripheral arterial disease in Bangladesh.

Data regarding prevalence of stroke in Bangladesh is inadequate. The prevalence of stroke has been estimated from a community study involving 15,627 participants aged >40 years with an overall prevalence of stroke of 0.30%. Stroke prevalences were reported as 0.20%, 0.30%, 0.20%, 1.00%, and 1.00% for the age groups of 40–49 years, 50–59 years, 60–69 years, 70–79 years, and 80 years and above, respectively. In a recently published study, the prevalence of stroke in rural population aged >30 years has been found to be 0.94% in general, 1.45% in male and 0.45% in female.

Data regarding the prevalence of CVDs in Bangladesh are insufficient and not homogeneous. Well-designed epidemiological studies are needed to generate reliable and up to date data which can be applied in formulation and implementation of healthcare policies at national level. Realizing these limitations the estimated current prevalence of different CVDs in the country is shown in Table 4.

### Risk factors of CVD in Bangladesh

#### Ethnicity and genetics

Ethnicity is an important determinant of prevalence of CVD specially CAD. When compared to other ethnicities, South Asians i.e. individuals originally from India, Pakistan, Nepal, Bangladesh and Sri Lanka, have a high prevalence of CAD and associated risk factors. South Asians have a 3 to 5-fold increased risk of myocardial infarction. South Asians also present with more severe disease and at an earlier age than Caucasians. The London Life Sciences Population Study (LOLIPOP) and the Pakistan Risk of Myocardial Infarction Study (PROMIS) have given important insights into the genetics associated with the undue susceptibility of the South Asians to cardiometabolic conditions including CAD. Over 25 cosmopolitan loci for CAD and type 2 diabetes have already been discovered showing that there are genetic risk factors for cardio-metabolic conditions that apply to people of South Asian ancestry and to people of European ancestry. Bangladeshis appear to share with other South Asian populations the same susceptibility to CAD; however, the probability of existence of an even more prone ‘Bangladeshi ethnicity’ in not impossible. In concert with this concept, the initial analyses of the ongoing Bangladesh Risk of Acute Vascular Events (BRAVE) study indicates that Bangladeshis are genetically distinct from major non-South Asian

### Table IV

<table>
<thead>
<tr>
<th>Disease</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>20-25% in adults</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>4-6% in adults</td>
</tr>
<tr>
<td>Rheumatic fever/Rheumatic heart disease</td>
<td>&lt;1/1000 in children and young adults</td>
</tr>
<tr>
<td>Congenital heart disease</td>
<td>25-30/1000 live births</td>
</tr>
<tr>
<td>Stroke</td>
<td>0.3-1.0% in adults</td>
</tr>
<tr>
<td>Heart failure, arrhythmias, cardiomyopathies and peripheral arterial disease</td>
<td>No available data</td>
</tr>
</tbody>
</table>
ethnicities, as well as distinct from other South Asian ethnicities and were perhaps genetically closest to (though still distinct from) Sri Lankan Tamils.\textsuperscript{54} There may also be genetic susceptibility to hypertension. The association between angiotensin converting enzyme (ACE) gene polymorphism and blood pressure has been studied inadequately in Bangladeshi population.\textsuperscript{55,56} In 2002, Morshed et al. found positive association between ACE insertion/ deletion (I/D) polymorphism and hypertension in Bangladeshi population. Among the three ACE I/D variants, the DD genotype was associated with the highest value of both mean systolic and mean diastolic blood pressure ($p = <0.05$) in men. In the overall population, blood pressure was highest in DD, intermediate in ID, and the least in II subjects. Further research is needed to clarify this relationship.

\textit{Diabetes mellitus}

Diabetes has become a national health concern in Bangladesh. The BDHS 2011 showed the overall, age-standardized prevalence of diabetes and pre-diabetes to be 9.7\% and 22.4\%, respectively; among urban residents, the age-adjusted prevalence of diabetes was 15.2\% compared with 8.3\% in rural residents.\textsuperscript{67,68} The prevalence of diabetes mellitus was 7.4\% (95\% CI 7.2-7.7\%) in a recently published review involving 51,252 participants, and also there was an increasing trend of diabetes prevalence among urban and rural population in Bangladesh.\textsuperscript{69}

According to the International Diabetes Federation assumption in 2010, the explosion in diabetes prevalence will place Bangladesh among the top 7 countries in terms of the number of people living with diabetes in 2030.\textsuperscript{70}

\textit{Smoking and smokeless tobacco use}

Tobacco use is quite common in Bangladesh. Bangladesh is one of the top 10 countries that make up two-thirds of the world population of smokers.\textsuperscript{71} According to the Bangladesh NCD risk factor survey 2010, the prevalence is 51.0\% for any form of tobacco, 26.2\% for smoking and 31.7\% for smokeless tobacco (SLT).\textsuperscript{27} Current tobacco use is 43.3\% in Bangladesh; exclusively smoking is 16.1\%, exclusively using SLT 20.3\%, and dual use of smoking and SLT is 6.8\% according to the Global Adult Tobacco Survey (GATS).\textsuperscript{72} The prevalence of smoking among men in Bangladesh is higher than the world average of daily smoking among men (37\% vs. 31.1\%).\textsuperscript{73} According to a proportional mortality study, smoking causes about 25\% of all deaths in Bangladeshi men aged between 25 to 69 years and an average loss of 7 years of life per smoker.\textsuperscript{74} However, currently published research does not provide conclusive evidence regarding the association between SLT use and CAD. In a recently published systematic review, 9 studies found no statistically significant positive association between SLT use and CAD, while 9 studies did find a positive association.\textsuperscript{75}

\textit{Dyslipidaemia}

The excess burden of CAD among South Asians appears to be primarily due to dyslipidemia that is characterized by high levels of apolipoprotein (apo) B, triglycerides (TG), and lipoprotein (Lp)(a); borderline high levels of low-density lipoprotein cholesterol (LDL-C); and low levels of high-density lipoprotein cholesterol (HDL-C) and apoA1.\textsuperscript{76} Liberal use of saturated fats and trans fats, deep frying, reuse of cooking oil, and overcooking leading to destruction of folates may all contribute to dyslipidaemia in this population.\textsuperscript{78} Studies exclusively related to dyslipidaemia are sparse in Bangladesh. A study\textsuperscript{79} involving 51,353 predominantly urban population during 2005-2011 demonstrated significantly higher mean serum levels of total cholesterol (TC), LDL-C, TG, LDL to HDL cholesterol ratio and TC to HDL-C ratio among younger adults aged 30-39 years compared to other age groups, regardless of sex, which may lead to microvascular complications. Another study\textsuperscript{80} involving 320 individuals found rising trend of dyslipidaemia in sub-urban population; prevalence of dyslipidaemia was 16.6\% in general and 22.2\% in males and 15.9\% in females. TC was high (>240 mg/dl) in 16.9\%, LDL-C was high (>160 mg/dl) in 15.7\%, HDL-C was low (<40 mg/dl) in 8.8\%, and TG was high (>200 mg/dl) in 17.8\% and very high (>350 mg/dl) in 2.0\% population. Women had significantly higher TC and LDL-C in comparison to men above 40 years. Contrary to the popular belief, dyslipidaemia is common in rural people as well.\textsuperscript{81} Studies are needed to determine the lipoprotein profile of the population for better understanding of the contribution of dyslipidaemia to the aetiopathogenesis of CVD.

\textit{Lifestyle related factors}

As a result of socioeconomic transition, lifestyle, as well as, the dietary pattern is changing in Bangladesh. Increasing prevalence of obesity, tobacco use, high intake of processed foods and less physical activity accompany this transition.

Prevalence of overweight and obesity is increasing. In general, 21.5\% adults (male 21\%, female 22\%) have
Body-mass index (BMI) ≥25 kg/m²; increased waist circumference is alarming especially in women (33.7%). In a population-based, cross-sectional survey conducted in 2009 involving 2293 subjects aged ≥20 years from rural Bangladesh, the age-standardized prevalence of overweight (BMI 23-24.9 kg/m²) and obesity (BMI ≥25 kg/m²) were 17.7% (95% confidence interval (CI): 16.1, -19.2%) and 26.2% (95% CI: 24.4-27.9%), respectively. The age-standardized prevalence of central obesity based on waist circumference (male ≥90 & female ≥80 cm) and waist hip ratio (male ≥0.90 and female ≥0.80) were 39.8% (95% CI: 37.9, 41.7%) and 71.6% (95% CI: 69.8, 73.4%) respectively. The prevalence of central obesity was more in female than male. Both total obesity and abdominal adiposity were associated with development of CAD in Bangladeshi population.

Childhood obesity is a growing concern in this population. A recent review showed an increasing trend in childhood obesity over time in Bangladesh; prevalence ranged from less than 1% to 17.9% based on different reference standards, with higher percentage amongst urban children. In a recent countrywide cross-sectional study, from June to September 2009 among 10,135 students of 6 to 15-year age group from both the urban and rural schools, 3.5% were obese, 9.5% were overweight and 17.6% were overweight. The proportion of obese and overweight students were greater among the students from urban schools (5.6% and 10.6% respectively) compared to the students from rural schools (1.2% and 8.6% respectively) (Risk difference, RD = 4.3, 95% CI = 3.6, 5.0; RD = 2.0, 95% CI = 0.1, 3.1).

A recent study found a high prevalence of overall and central obesity in adolescent girls in Bangladeshi population; the prevalence of obesity and overweight were 23% and 14%, whereas the prevalence of central obesity was 26%. Around 14% of girls in the normal weight group were centrally obese.

The prevalence of metabolic syndrome is also high in Bangladesh. In a recent population-based cross-sectional study involving 2,293 randomly selected participants (aged ≥20 years) in a rural community in Bangladesh, the age-adjusted prevalence of metabolic syndrome was 30.7% (males 30.5%; females 30.5%) using the National Cholesterol Education Programme (NCEP) Adult Treatment Panel III (ATP III) definition, and 24.5% (males 19.2%, females 27.5%) using the International Diabetes Federation (IDF) definition. In another study, the prevalence of metabolic syndrome was found to be 20.7%, 11.2% and 8.6% following ATP III, IDF and by the World Health Organization (WHO) definitions, respectively. Metabolic syndrome is probably commoner in women. The prevalence of metabolic syndrome was found to be 31.25% (NCEP ATP III modified) in 1485 rural women of Bangladesh aged ≥15 years.

Sedentary life style may have an association with CAD. Bangladesh NCD Risk Factor Survey 2010 found low level of physical activity (<600 metabolic equivalent-minutes) per week. Future research is needed to determine the association of physical inactivity to the high incidence of CAD in Bangladesh.

Dietary pattern may play role in aetiopathogenesis of CVD. Like many other developing countries, socioeconomic transition is accompanied by a changing dietary pattern in Bangladesh. A prospective cohort analyses in 11,116 participants enrolled in the Health Effects of Arsenic Study in Araihazar, Bangladesh, with a follow-up of average 6.6 years, an animal protein-rich diet in rural Bangladesh was associated with increased risk of CVD mortality, especially among smokers. Diets were classified in patterns: (i) a “balanced” pattern, comprised of steamed rice, red meat, fish, fruit and vegetables; (ii) an “animal protein” diet, which was more heavily weighted towards eggs, milk, red meat, poultry, bread, and vegetables; and (iii) a “gourd and root vegetable” diet that heavily relied on a variety of gourds, radishes, pumpkin, sweet potato, and spinach. ‘Western’ dietary pattern was associated with greater longitudinal increase in blood pressure in comparison to the ‘gourd vegetable’ dietary pattern and the ‘balanced’ dietary pattern. Similar observations were found in a previous study. In the participants (n=1149) randomly selected from the Health Effects of Arsenic Longitudinal Study, a gourd/root vegetable diet in this Bangladeshi population positively correlated with carotid intima-media thickness a validated surrogate marker of preclinical atherosclerosis, while a balanced diet was associated with decreased intima-media thickness.

The average Bangladeshi eats a total of 126 g of fruit and vegetables daily, which is far below the minimum daily consumption of 400 g of vegetables and fruit recommended by Food and Agriculture Organization of the United Nations and the WHO. Bangladesh NCD Risk Factor Survey 2010 revealed 95.7% people consume inadequate fruit and/or vegetables (<5 servings per day). A more recent study using spot urine analysis found very high average sodium intake of
Considering these data, salt intake in this country appears to be much higher than what is recommended by the WHO (sodium chloride <5 g/day, sodium <2 g/day) or the 2015–2020 Dietary Guidelines for Americans (sodium <2.3 g/day in general and children ages >14 years, and <1.5 g/day for individuals with prehypertension and hypertension). Extra salt intake along with age, BMI, physical inactivity, tobacco use and family history of stroke/CVD was found to have significant relationship with hypertension and pre-hypertension in a cross-sectional survey involving participants aged e” 25 years in an urban area in Dhaka between June to December 2012. Also, more than 35 million people in coastal Bangladesh are vulnerable to increasing freshwater salinization; elevated salinity in drinking water has been found to be associated with higher BP in young coastal populations. The overall risk perception regarding excessive salt consumption is low and there is widespread belief that the cooking process can render the salt harmless. High salt intake presumably contributes to hypertension, which is an established risk factor for CAD.

**Low-Birth Weight and Childhood Malnutrition**

The developmental origin theory of CAD proposes that undernutrition in utero permanently changes body functions and metabolism leading to an increased risk of CAD in adult life. However, a recently published study involving German youths aged 3-18 years did not find significant association between birth weight and traditional cardiovascular risk factors. Low birth weight (<2,500 g) affects 36% of infants in Bangladesh, more than twice of 15% threshold that indicates a public health burden. Also, <1% of infants are born with very low birth weight (<1,500 g). Research is needed to explore association, if any, between the two public health problems i.e. low birth weight and CAD in this community. Under-nutrition during childhood, adolescence, or your adulthood is related to CAD and stroke in adult life. Despite the progress achieved, rates of malnutrition in Bangladesh are among the highest in the world; more than 54% of preschool-age children, equivalent to more than 9.5 million children, are stunted, 56% are underweight and more than 17% are wasted. An analysis revealed that among the children under five years of age 16% were severely stunted, 25% moderately stunted, 3% severely wasted and 14% were moderately wasted; furthermore, 11% of the children were severely underweight and 28% were moderately underweight. Such a high prevalence of low-birth weight childhood under-nutrition may facilitate development of CAD in adult in Bangladeshi population.

**Hypovitaminosis D**

Role of Vitamin D in cardiovascular health is of much interest at present. Experimental, as well as, some observational studies suggest that vitamin D and its metabolites are integrally related to blood pressure and the renin-angiotensin system. Vitamin D insufficiency affects almost 50% of the population worldwide. Few studies have been carried out to determine the prevalence of hypovitaminosis D in Bangladesh. In a recently published study involving husbands of pregnant women in Dhaka, vitamin D deficiency was prevalent in both men and women but men had substantially higher circulating 25-hydroxycholecalciferol (25(OH)D) concentrations and lower risk of vitamin D deficiency than their pregnant spouses; gender-related lifestyle factors, rather than ethnic or environmental factors likely explain the high risk of vitamin D deficiency among women of reproductive age in Bangladesh. Vitamin D deficiency was found prevalent in young infants in rural Bangladesh. High prevalence of suboptimal serum 25(OH)D level (<25 nmol/l) was described in lactating women of low socioeconomic status and those wearing Shari, a traditional ladies wear. In another survey of women aged 18–60 years, serum 25(OH)D levels were <40 nmol/l in 78% of 36 university students and 83% of 30 veiled women. Further research is needed to evaluate the association, if any, between vitamin D deficiency and CAD in Bangladesh.

**Chronic arsenicosis**

Arsenic contamination of groundwater in Bangladesh has been recognized as a massive public health hazard. Positive association has been found between chronic arsenic exposure and CVD, ECG abnormalities, hypertension, and stroke. Chronic arsenic exposure may facilitate systemic inflammation and vascular endothelial dysfunction, which may, in turn, increase the risk of CVD. The Health Effects of Arsenic Longitudinal Study in Bangladesh (2007–2008) has reported positive association between arsenic exposure from drinking water and plasma levels of markers of systemic inflammation and endothelial dysfunction. In the same population, positive association has been found between inorganic arsenic exposure from drinking water and risk of hypertension, and more recently, increased cardiovascular mortality. Further basic, as
well as, clinical research is needed to better define the role of arsenicosis in the aetiopathogenesis of CVD in Bangladeshi population.

**Air Pollution**

In the recent years, air pollution has been suggested to contribute to cardiovascular illness. The overall evidence is consistent with a causal relationship between exposure to particulate matter <2.5 µm in diameter (PM$_{2.5}$) and cardiovascular morbidity and mortality. The role of air pollution is a significant problem specially in the urban areas of Bangladesh with marked temporal and directional variations in particulate matter concentrations. A study to evaluate the emissions and air quality in megacities found Dhaka to have the poorest air quality in respect of total suspended particles (TSP), sulfur dioxide (SO$_2$), and nitrogen dioxide (NO$_2$) among the megacities, and the pollutant levels were far beyond the WHO standard. One recent study involving Dhaka City found elevated concentrations of the number, surface and mass distributions of particulate matters; fine particles (0.5–1.0 µm) derived from vehicle emissions were dominating the aerosol particles number concentrations. Investigation of sources of atmospheric aerosol at urban and semi-urban areas in Bangladesh revealed soil dust, road dust, cement, sea salt, motor vehicles and biomass burning to be the main sources of air pollution. Vehicular emissions and emission from brick kiln are the major contributors to air pollution in Dhaka especially during dry seasons, while contribution from emissions from metal smelters increases during rainy seasons. In rural areas, indoor air pollution from the combustion of traditional biomass fuels is a significant public health problem in many developing countries, including Bangladesh. One study found the major constituent of the particulate matter in rural air was carbonaceous matter. Chronic exposure to the particulate matter in indoor air from combustion of traditional biomass fuels may be a contributor to the CAD in rural women who are especially concerned with cooking. In a retrospective cohort study in Matlab, Bangladesh, household solid-fuel use was associated with increased respiratory mortality and non-significantly increased risk of cardiovascular mortality. Further research is needed to elucidate the role of air pollution to the aetiopathogenesis of CVD in Bangladeshi population.

**Cardiovascular Care in the Past**

Traditionally, cardiac diseases were treated by physicians specialized in medicine. In the Government sector, the cardiac patients were managed in the medicine outpatient and inpatient departments; the facilities were limited to the medical college hospitals, the formerly Institute of Postgraduate Medicine & Research (IPGMR), and the Combined Military Hospital (CMH). The first coronary care unit was established at IPGMR where electrocardiogram (ECG) and phono-cardiographs were available. First integrated cardiovascular care started in this country with the establishment of the then Institute of Cardiovascular Diseases (ICVD), later named as the National Institute of Cardiovascular Diseases (NICVD) in 1978, and formally in April 1981. Generous technical and financial cooperation was provided by the Government of Japan in the advent of cardiovascular care in Bangladesh. Besides medical management, invasive diagnostic and therapeutic modalities were started there. First permanent pacemaker implantation was done in 1981. Since the introduction of angioplasty by Gruentzig in 1977, Bangladesh took more than a decade to make appropriate utilization of this technology. Percutaneous transluminal coronary angioplasty (PTCA) was introduced in ICVD by foreign experts in 1987; the first case was failed, while 1 case was successfully done in 1990. First PTCA was done by Bangladeshi team in 1995, first coronary stenting in 1997. First pulmonary valvuloplasty was done in 1987, while percutaneous transluminal mitral commissurotomy (PTMC) was introduced in 1996. For the first time, few cases of closed mitral commissurotomy (CMC) were done in IPGMR in 1973 by Prof. Ali Ashraf, and then in National Institute of Diseases of Chest & Hospital (NIDCH) in 1979 by Prof. SR Khan. (Ahmed NU. Professor of Cardiac Surgery. Personal communication, 25 Dec 2013). First open heart surgery was done in NICVD in 1981, while coronary artery bypass grafting (CABG) was done in NICVD for the first time in 1985. CMC started in NICVD in 1980, and subsequently, a good number of cases were done at low cost with good results. Under the patronization of Dr. Rafique Ahmed of USA, non-pharmacological management of cardiac arrhythmia started in Bangladesh; an electrophysiology (EP) lab was established in NICVD, and the first EP study was done in July 2004. Also under his supervision, automated implantable cardioverter-defibrillator (AICD) was implanted for the first time in NICVD in 2005. First device closure of PDA was done in 2006 in the same institute.

**Present Status of Cardiovascular Care Facilities**

After the 80s, cardiovascular care facilities in Bangladesh have increased steadily. At present, a good number of institutions are rendering cardiovascular care throughout the country; also, they are becoming decentralized. The cardiac care institutions in Bangladesh are public, private and autonomous. Some are dedicated cardiac institutions, while others are in fact multi-speciality institutions having cardiac care facilities. At present, cardiovascular intervention and surgical facilities are available approximately in 30 and 25 institutions respectively. There are 39 catheterization laboratories as in November, 2016. Almost all conventional noninvasive modalities including ECG, echocardiography,
exercise tolerance test are being more widely used for diagnosis of cardiac diseases. Side by side, invasive modalities are becoming more and more available; facilities for coronary angiogram and cardiac catheterization are rapidly expanding. More sophisticated diagnostic facilities including computed tomography (CT) coronary angiography, transesophageal and 3-dimensional echocardiography, electrophysiological study, fractional flow reserve, and optical coherence tomography are available in selected centres.

Besides diagnostic modalities and techniques, treatment facilities have also increased in numbers. Besides percutaneous coronary intervention (PCI), and PTMC, radiofrequency ablation for arrhythmias, AICD implantation, biventricular pacing, and device closure of shunt lesions are also available in practice. Different types of CABGs including OBCAB, minicab, midCAB, valve surgeries, surgery of congenital heart lesions, and importantly, vascular surgeries are in rapid evolution.

Classically, severe cases of mitral stenosis with suitable valve morphology were managed by surgery i.e. CMC. However, open mitral commissurotomy (OMC) is being used for suitable, complicated cases, like before. On the other hand, for mitral valve replacement, some bioprostheses were used initially, which were later replaced by metallo-prostheses. Initial ball-and-case metallic valves are out of use now-a-days, majority of the prostheses used today are bileaflet valves. In the recent years, primary angioplasty is being performed in Government, as well as, in private centers.

At present, no complete registry concerning CVD and cardiovascular care including interventions exist in Bangladesh. Hence, the available data are incomplete. Recently, a registry has been formulated and maintained by the Bangladesh Association of Cardiovascular and Thoracic Anaesthesiologists (BACTA); the purpose is to maintain data regarding the cardiovascular and thoracic operations done throughout the country. A registry involving

### Table-V

*Catheterization laboratory procedures performed in the NICVD in 2001-2016. (Rahman SW. Statistics Officer, NICVD. Personal communication. 28 February 2017)*

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NICVD, National Institute of Cardiovascular Diseases; PCI, Percutaneous coronary intervention; PTMC, Percutaneous transvenous mitral commissurotomy; EPS, Electrophysiological study; RFA, Radiofrequency ablation; PPM, Permanent pacemaking; TPM, Temporary pacemaking.
Table VI
Cardiovascular surgeries done in the NICVD in 2001-2016. (Rahman SW. Statistics Officer, NICVD. Personal communication. 28 February 2017)

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<tr>
<td>2013</td>
<td>147</td>
<td>293</td>
<td>450</td>
</tr>
<tr>
<td>2014</td>
<td>103</td>
<td>310</td>
<td>492</td>
</tr>
<tr>
<td>2015</td>
<td>147</td>
<td>239</td>
<td>393</td>
</tr>
<tr>
<td>2016</td>
<td>206</td>
<td>226</td>
<td>464</td>
</tr>
</tbody>
</table>

NICVD, National Institute of Cardiovascular Diseases; CABG, Coronary artery bypass graft;

Table VII
Cardiac surgeries done in Bangladesh in 2015.145-6

<table>
<thead>
<tr>
<th>Type of surgery</th>
<th>2014</th>
<th>2015</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CABG</td>
<td>4809</td>
<td>5305</td>
</tr>
<tr>
<td>CABG+Valve/ASD/VSD</td>
<td>77</td>
<td>83</td>
</tr>
<tr>
<td>Valvular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MVR</td>
<td>535</td>
<td>509</td>
</tr>
<tr>
<td>AVR</td>
<td>248</td>
<td>251</td>
</tr>
<tr>
<td>DVR</td>
<td>172</td>
<td>151</td>
</tr>
<tr>
<td>Congenital</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASD</td>
<td>789</td>
<td>779</td>
</tr>
<tr>
<td>VSD</td>
<td>456</td>
<td>405</td>
</tr>
<tr>
<td>TOF (Total correction)</td>
<td>358</td>
<td>390</td>
</tr>
<tr>
<td>BT shunt</td>
<td>23</td>
<td>33</td>
</tr>
<tr>
<td>PDA</td>
<td>165</td>
<td>207</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>378</td>
<td>1221</td>
</tr>
<tr>
<td>Total</td>
<td>8060</td>
<td>9094</td>
</tr>
</tbody>
</table>

CABG, Coronary artery bypass graft; Valve, Valvular surgery; ASD, Atrial septal defect; VSD, Ventricular septal defect; MVR, Mitral valve replacement; AVR, Aortic valve replacement; DVR, Double valve replacement; TOF, Tetralogy of Fallot; BT, Blalock–Taussig shunt; PDA, Patent ductus arteriosus.
the cardiac interventions done is under construction on behalf of the Bangladesh Cardiac Society.

Data available from NICVD and the registry maintained by BACTA are being presented here. (Table 5 to 8)

Besides curative services, there are some efforts to ensure preventive and promotive services.

In the current Health, Population and Nutrition Sector Development Program (HPNSDP) 2011-2016, control of NCDs, including CVDs is one of the topmost priority areas of healthcare in the country.\textsuperscript{10} Government has formulated National NCD Strategy and plan of action. Different non-Government organizations, including WHO are playing important role in this regard as well.

The BRAVE [Bangladesh Risk of Acute Vascular Events] study

Despite the enormous magnitude of CVD in Bangladesh, the volume and quality of research related to this issue is limited. Also, many aspects of CVD in Bangladesh, including the undue prevalence of CAD in this population, are unknown. The Bangladesh Risk of Acute Vascular Events (BRAVE) study is an epidemiological bioresource established to examine environmental, genetic, lifestyle and biochemical determinants of CAD among the Bangladeshi population.\textsuperscript{54} This study is a joint collaboration of Cambridge University of UK, International Centre for Diarrhoal Disease Research, Bangladesh (ICDDR,B) and the NICVD in Dhaka of Bangladesh. The study was established in 2011 by the Department of Public Health and Primary Care at the University of Cambridge (the study’s international coordinating centre), in collaboration with the Chronic NCD Unit at ICDDR,B and at NICVD in Bangladesh. By early 2015, the ongoing BRAVE study had recruited over 5000 confirmed first-ever myocardial infarction cases, and over 5000 controls “frequency-matched” by age and sex. Initial analyses indicate that Bangladeshis are genetically distinct from major non-South Asian ethnicities, as well as distinct from other South Asian ethnicities. Also, several environmental contaminants (e.g. arsenic in the blood) and nutritional elements (e.g. zinc deficiency) are emerging as important drivers for heart attacks in this population.

DNA genotyping data of 5755 subjects of the BRAVE study have been used in a recent study also involving the participants of the PROMIS study, and the participants belonging to the European ancestry.\textsuperscript{147} The study demonstrates that carriers of loss-of-function mutations in ANGPTL4 gene had triglyceride levels that were lower than those among noncarriers; these mutations were also associated with protection from CAD.

Future Directions

Data related to different aspects of CVD in Bangladesh are inadequate. Large, preferably nationwide epidemiological and clinical studies should be carried out to gain reliable information on this important public health issue. CVD prevention should be integrated with primary health care. Cardiovascular health promotion should be part of the national media strategy and the health education curriculum. The public health approach should target population-wide lifestyle intervention, screening for high blood pressure, diabetes and dyslipidaemia. Healthy lifestyles including consumption of heart-healthy diets, avoidance to smoking and smokeless tobacco, moderation of salt intake and increased physical activity, should be promoted. Limitations can be placed on the concentrations of salt, sugar, trans-fats and saturated fats in manufactured food products. Food labeling should also be introduced to facilitate informed choice by consumers. Food adulteration should be dealt rigorously. Provision of safe, arsenic-free water and food should be ensured. Necessary legislative and administrative steps should be taken to reduce air pollution. Policy change should address urban planning, transport and preservation of environment. Special attention should be given to stop malnutrition and under nutrition in fetal and neonatal life through nutrition programmes. Public awareness should be created to avoid childhood obesity. If indicated by further research, vitamin D deficiency may be avoided by fortification of food. Further research, may be in collaboration with international organizations, should be undertaken to explore the still-unidentified risk factors of CVD unique to this nation.

Renovation of National Centre for Control of Rheumatic Fever and Heart Diseases (NCCRF&HD) may be done to boost up research in RF and chronic RHD and render point-of-care services involving medical, interventional, as well as, surgical modalities of treatment. Appropriate guidelines should be formulated in relation to RHD to bring about uniformity and rationality in existing practice.

Conclusion:

CVD is a major public health burden in Bangladesh. Besides the well-known risk factors, genetic factors and some emerging risk factors unique to this population may play an important role in CVD. At the advent of the new millennium, more and more information is becoming available; however, presumably much is still unknown.
We have no more time to lapse. Large-scale, preferably, nation-wide survey and clinical research should be conducted to determine the different aspects of CAD in Bangladesh. The information available hereby, would help to formulate national policy to combat the deadly epidemic more efficiently in future. This information would be used to formulate national cardiovascular guidelines for early detection and prevention of CVD with top importance.

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Case Report

Situs Inversus Totalis: A Rare Congenital Anomaly and the Presence of COPD Giving a Concept of Dextroposition with Limb Lead Reversal in ECG

Khandker Md. Nurus Sabah¹, Abdul Wadud Chowdhury², Mohammad Shahidul Islam³, Mohammad Gaffar Amin¹, Md. Azizul Hassan Khandakar⁴, Shamima Kawser⁵

Abstract:
Situs inversus totalis is the mirror-image of normal position of the thoracic and abdominal viscera. It may be detected incidentally when the patient seek medical attention for other medical illness. From medico-legal points of view, this rare disorder is important in many ways for a junior physician to prevent a big mishap, especially surgical. Here, we report a case, who was 55-year-old, seeking medical attention for infective exacerbation of chronic obstructive pulmonary disease incidentally diagnosed as a case of situs inversus totalis.

Key words: Situs inversus, Dextrocardia, ECG.

Introduction:
Situs inversus totalis, also familiar as dextrocardia with situs inversus, situs transverses or opposites is a congenital condition in which the visceral organs of chest and abdomen are reversed from their normal position, giving a mirror-image of normal. It is first described by Matthew Ballie in 1788¹. For normal cardiovascular and abdominal visceral functioning, it may not be detected in whole life, may be detected incidentally when they seek medical attention for an unrelated condition, in post-mortem or while dissection of cadaver in anatomy dissection teaching class. Last year, a 55-year-old farmer came to us with the infective exacerbation of chronic obstructive pulmonary disease. Incidentally, situs inversus totalis was identified.

Case Presentation:
A 55-year-old Bangladeshi, chain-smoker, farmer presented with high-grade fever, breathlessness for 1 week. He gave history of dry cough and mild short of breath for last 2 years. Now he had fever, that came without chills and rigor, maximum temperature 103°F, persisted most of the time of day and it subsided after taking anti-pyretic drugs. Initially he had mild short of breath. Later it progressed day by day. On admission, patient was toxic, having respiratory distress with respiratory rate 34 breaths/min with wheeze heard with unaided ear, blood pressure – 140/90 mm Hg, pulse – 140 beats/ min and regular, nicotine staining in nails of hands. There was no clubbed finger, cyanosis, Examination of chest revealed features of chronic obstructive pulmonary disease. Examination of precordium revealed apex beat not felt, heart sound was very distant, more prominent on right side of sternum. Clinical presentation and examination findings were suggestive of infective exacerbation of chronic obstructive lung disease.

Complete blood count revealed high WBC count with neutrophilic leucocytosis. ESR was 40 mm in 1st hour. There were normal electrolytes and slightly raised serum creatinine (1.49 mg/dl).

Electrocardiography showed heart rate 140 per minute with sinus rhythm, right axis deviation, prominently

1. Assistant Professor, Department of Cardiology, Dhaka Medical College, Dhaka, Bangladesh.
2. Professor & Head of Department, Department of Cardiology, Dhaka Medical College, Dhaka, Bangladesh.
3. Registrar, Department of Medicine, Anwer Khan Modern Medical College, Dhaka, Bangladesh.
5. Associate Professor, Dr. Sirajul Islam Medical College, Dhaka, Bangladesh.

Address of Correspondence: Dr. Khandker Md. Nurus Sabah, Assistant Professor, Department of Cardiology, Dhaka Medical College Hospital, Dhaka, Bangladesh. E-mail: sabahkmn09@gmail.com
negative P wave, QRS complexes and T wave in lead I, normal P wave in inferior leads, flattened P wave in aVL, positive QRS complexes with upright P and flattened T wave in aVR, absence of R wave and prominent of S wave in chest leads (Figure I). Initially it was thought to be due to erroneous positional change of limb leads. After checking the position of limb leads, it was taken and revealed the same findings. Later chest radiography revealed dextrocardia with the cardiac apex pointing to the right side, the aortic arch and stomach bubble located on the right and features of chronic obstructive pulmonary disease with hyperinflated lung shadow, low flat diaphragm, widening of intercostals space and horizontal ribs. Colour Doppler Echocardiography mentioned situs inversus with dextrocardia with L-loop ventricle and L-normal great vessels, normal size cardiac chambers and normal ejection fraction.

USG of whole abdomen revealed mirror-image anatomy of the abdominal viscera with liver on left side and spleen on right side and no other abnormality. Radiography of paranasal sinus revealed no abnormality. All the findings were suggestive of situs inversus totalis. For COPD, he was treated according to the hospital protocol and discharged on 7th day of hospitalization after clinical improvement.

It showed heart rate 140 per minute with sinus rhythm, right axis deviation, prominently negative P wave, QRS complexes and T wave in lead I, normal P wave in inferior leads, flattened P wave in aVL, positive QRS complexes with upright P and flattened T wave in aVR, absence of R wave and prominent of S wave in chest leads (though it is not a good quality of electrocardiography).

**Discussion:**

Situs inversus totalis is a rare congenital anomaly, occurs approximately 2 in 10,000 births\(^2\). Unfortunately, there is no registry in Bangladesh. Only few cases were reported \(^3\), \(^4\), \(^5\), \(^6\).

This positional anomaly is important for a physician as – 1) to prevent a surgical disaster in diagnosis and/or surgical intervention following failure to identify the reversed anatomy or an atypical history as cholecystitis causes left upper quadrant pain 2) for the prevention of false-negative reporting by the radiologist resulting from inattention to labelling or false-labelling by a technician 3) to avoid misleading electrocardiographic findings 4) to evaluate all cardiac structure in an organized way as a small proportion have some form of congenital heart disease 5) to identify another disease ‘kartagener syndrome’ having bronchiectasis, sinusitis and situs inversus.

This disease is transmitted usually but not invariably in an autosomal recessive manner. It is found in X-linked pattern of inheritance and in identical twins \(^7\), \(^8\). Abnormalities in Lefty gene, nodal gene, ZIC 3, ACVR2B and Ptxz gene is identified but not obvious culprit for this left-right asymmetry \(^9\), \(^10\). Still research is going on. Some predisposing factors- materanal diabetes, family history of malformations, cocaine use during the 2 months before conception and through the first trimester or being a conjoined twin may increase the risk of heterotaxy mentioned in the review of the data from the Baltimore-Washington Infant Study \(^11\), \(^12\).
Many people with situs inversus totalis are unaware of their visceral positional abnormality in whole life as they are phenotypically unimpaired and lead normal healthy life that was seen in this case though it depends on associated congenital defects and the presence of cardiovascular compromise. Congenital heart disease is observed in only 3% cases of situs inversus totalis. Of these patients, 80% have a right-sided aortic arch. In 20% cases, kartagener syndrome is seen; however, only 50% of patients with kartagener syndrome have situs inversus. Here, It is better to mention that in dextrocardia, the arrangements of the position of the abdominal viscera may be normal (situs solitus), reversed (situs inversus) and indeterminate (situs ambiguous) in 34.4%, 39.2% and 26.4% cases respectively.

It may be recognized first by using radiography or ultrasonography. Computed tomography scanning is the preferred examination for the details of visceral organ position, cardiac apical position and great vessels branching and the degree of confidence is high. In this case, ultrasonography had being preferred option and further no question aroused.

Sometimes, electrocardiography gives a clue to a diagnosis of dextrocardia. It shows inverted P, QRS and T wave in lead I & aVL, positive deflection in aVR with decreasing amplitude of R wave from lead V_1 through V_6. A same feature is seen in case of reversal of the arm leads except decreasing R wave amplitude in V_1 - V_6. When chronic obstructive lung disease is present, right axis deviation, poor R wave progression is seen in V_1 through V_6. In this case, after getting ECG report, it was thought to be a case of COPD with reversal of limb leads. Later, this concept proved to be a wrong one when repeat ECG with correcting limb lead position, chest radiography and echocardiography was done.

Many of the junior physicians can miss this easily if he or she doesn’t look this carefully. This case report emphasizes to know this rare anomaly when dextrocardia is present with situs inversus.

Conclusions:
It is high-lighted to know this and keep in mind of a physician for correct diagnosis and for the prevention of surgical mishap. If there is any doubt after getting electrocardiography or radiological reports, it is best to examine again bedside and consult with concerning specialist, even with technician or repeat the test.

Acknowledgement
We acknowledge all the clinical staff of Department of Cardiology, Dhaka Medical College Hospital for cordial co-operation in this case.

References:


**Case Report**

**Lead-Reversal ECG Simulating Myocardial Infarction – A Case Report and Literature Review**

Mohammad Gaffar Amin¹, Abdul Wadud Chowdhury², Mohsin Ahmed³, Khandker Md Nurus Sabah⁴, Hasna Fahmima Haque⁵, Syed Rezwan Kabir⁶, Kazi Nazrul Islam⁷, Mohammad Abaye Deen Saleh⁸

**Abstract:**
Electrocardiogram (ECG) is one of the most important and time-tested tool for appropriate interpretation and diagnosis of coronary artery disease (CAD) and other structural & electrical cardiac abnormalities. Erroneous misplacement of surface ECG-leads (lead reversal) may result in wrong interpretation as well as unnecessary investigations, admission to hospital and improper treatment. Early & prompt recognition of lead-reversal is imperative to avoid inappropriate & harmful therapeutic interventions to the patients. This case-report focuses on an interesting scenario of ECG-lead misplacement, which will aid in detection, exclusion and further prevention of ECG lead misplacement.

**Key words:** Electrocardiogram diagnosis, Myocardial infarction

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**Introduction**

The electrocardiogram (ECG) is one of the most important and most frequently requested investigations in the management of patients in medical practice. It is both inexpensive and very efficacious in many different clinical scenarios. Proper & meticulous recording is of utmost importance in order to ensure an accurate interpretation of this time-tested diagnostic tool. Due to structural variation of different health-care programs, ECGs may not be recorded by expert technicians at all times; this leads to multiple errors in interpreting results. ECG-lead misplacement or reversal, that occurs in 0.4–4% of all 12-lead surface ECGs acquired in various clinical settings, results in erroneous interpretation as well as unnecessary investigations, admission to hospital and improper treatment.¹,² As a result incorrect ECG-Lead placement can result in significant harm to the patient if essential treatment is withheld or unnecessary treatment is delivered solely on the basis of ECG findings.² An interesting case of ECG-lead misplacement is presented here, which would have resulted in significant harm to the patient had it not been identified in time.

**Case report**

A 30 years old young man attended the cardiac outpatient department (OPD) of Dhaka Medical College Hospital (DMCH) with the complaints of non-specific chest-pain and occasional palpitation for two months. The pain was burning in nature, centered to lower chest and upper abdomen, non-radiating, occurring several times a day having no obvious precipitating factors but used to relieve by taking antacids & PPIs. The pain was associated with occasional palpitation which was of short duration & self-limiting. He complained of abdominal fullness but no history of nausea or vomiting. He was non-smoker & non-alcoholic. His father died of cardiac disease at 65 years of age. The patient was hemodynamically stable with pulse 80 beats/min, regular, normal in volume & character and BP 110/70mmHg. His precordial examination was normal and other systems revealed no abnormality. To evaluate the chest pain the attending doctor suggested a 12 lead
surface ECG (Figure 1) and later labeled the ECG as old myocardial infarction (MI) involving the inferior wall of heart. He immediately referred the patient to cardiac in-patient department (IPD) for further management. The attending consultant cardiologist reviewed the ECG thoroughly and found that the OPD-doctor interpreted the ECG on the basis of deep QS complexes & inverted T waves in leads II,III & aVF but he missed the inverted p waves in those leads; he also missed the completely inverted lead I and completely upright lead aVR (P wave, QRS complex and T wave). The attending consultant advised a repeat & supervised ECG (Figure 2) which was found completely normal. Transthoracic echocardiography was done later, which was unremarkable. The patient’s initial ECG abnormalities were finally diagnosed as erroneous misplacement of limb leads of ECG and he was sent to Gastro-enterology department for further management.

Fig.-1: Initial ECG done at Cardiac OPD of DMCH

Fig.-2: Repeat supervised ECG done at Cardiac IPD of DMCH
Discussion:
Cardiovascular diseases are the leading cause of death worldwide, accounting for 30% of all deaths. Of these, 42% are due to coronary artery disease (CAD). Proper ECG recording facilitates appropriate interpretation and diagnosis of CAD and other structural & electrical cardiac abnormalities. Electrode misplacements can lead to morphological changes on ECG that may potentially be interpreted as ischemic or arrhythmogenic in origin or may conceal the ECG features altogether. Therefore, recognition of the patterns seen in improper lead positioning is essential to avoid incorrect diagnoses and unnecessary treatments. The frequency of electrode misplacement has been shown to increase with increasing acuity of patient care and urgency of recording. Rudiger et al. reported a 0.4% rate of lead misplacement on ECGs performed on patients attending cardiac outpatient clinics and a 10-fold increased rate (4%) among patients in intensive care units. Several key findings on an ECG can help clinicians identify potential signs of electrode misplacements. In order to systematically identify these telltale clues, mnemonics to remember common errors and recognize their findings have been previously proposed. The REVERSE mnemonic is one such tool that outlines the most frequent abnormal findings on ECG (Table 1).

The 12-lead ECG is recorded by placing 10 electrodes (4 limb & 6 precordial) on predetermined anatomical locations. The terms electrode and lead are often used interchangeably. However, electrodes attach to the skin while leads are the vectors between the electrodes. The four limb electrodes [right arm (RA), left arm (LA), right leg (RL), left leg (LL)] generate six limb leads (leads I, II, III, aVR, aVL, and aVF). The right leg electrode (RL) is the grounding electrode and can be placed anywhere on the body without affecting the ECG appearance. ECG findings due to limb electrodes misplacement can be difficult to detect despite producing characteristic appearances in most cases (Table 2).

With reversal of the RA and LL electrodes, Einthoven’s triangle rotates 180 degrees vertically, from normal, around an axis formed by aVL (Figure 3 & Figure 4).

<table>
<thead>
<tr>
<th>Abnormal Finding</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>R wave is positive in lead aVR (P wave also positive)</td>
<td>Reversal of left arm and right arm electrodes</td>
</tr>
<tr>
<td>Extreme axis deviation: QRS axis between +180° and -90°</td>
<td>Reversal of right leg and left arm or right arm electrodes</td>
</tr>
<tr>
<td>Negative R wave in lead I, positive R wave in aVF</td>
<td></td>
</tr>
<tr>
<td>Very low (&lt;0.1 mV) amplitude in an isolated limb lead (isolated “flat” lead)</td>
<td>Reversal of right left and left arm or right arm electrodes</td>
</tr>
<tr>
<td>Exchanged amplitude of the P waves (P wave in lead I Greager)</td>
<td>Reversal of left arm and left leg electrodes</td>
</tr>
<tr>
<td>R wave abnormal progression in the precordial leads (predominant R wave in V1, predominant S wave in V6)</td>
<td>Reversal of precordial electrodes (V1 through V6)</td>
</tr>
<tr>
<td>Eliminate noise and interference (artifact mimicking tachycardias or ST-T changes)</td>
<td></td>
</tr>
</tbody>
</table>

Table-II
ECG changes resulting from interchange of the limb leads.

<table>
<thead>
<tr>
<th>Leads</th>
<th>RA-LA reversal</th>
<th>RA-LL reversal</th>
<th>RA-RL reversal</th>
<th>LA-LR reversal</th>
<th>LA-LL reversal</th>
<th>LL-RL reversal</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Inverted</td>
<td>Inverted III</td>
<td>Inverted III</td>
<td>II</td>
<td>II</td>
<td>Unchanged</td>
</tr>
<tr>
<td>II</td>
<td>Inverted II</td>
<td>Unchanged</td>
<td>Flat line</td>
<td>Unchanged</td>
<td>I</td>
<td>Unchanged</td>
</tr>
<tr>
<td>III</td>
<td>Inverted I</td>
<td>Inverted I</td>
<td>Flat line</td>
<td>Unchanged</td>
<td>Inverted III</td>
<td>Unchanged</td>
</tr>
<tr>
<td>aVR</td>
<td>aVL</td>
<td>aVF</td>
<td>Same as aVF (1/2 size III)</td>
<td>Inverted II</td>
<td>Unchanged</td>
<td>Unchanged</td>
</tr>
<tr>
<td>aVL</td>
<td>aVR</td>
<td>Unchanged</td>
<td>Inverted III</td>
<td>Same as aVF (1/2 size II)</td>
<td>aVF</td>
<td>Unchanged</td>
</tr>
<tr>
<td>aVF</td>
<td>Unchanged</td>
<td>Same as aVF (1/2 size III)</td>
<td>Same as aVF (1/2 size II)</td>
<td>aVL</td>
<td>Unchanged</td>
<td></td>
</tr>
</tbody>
</table>

RA, right arm; LA, left arm; RL, right leg; LL, left leg. This describes what the ECG output actually represents for each case of reversal. For example, in RA-LA reversal lead II actually represents lead III. Note: aVR and aVF are never the same in correct electrode placement.
This produces the following effects on the ECG (Figure 5): 11, 13, 14

- Lead II becomes inverted.
- Leads I and III become inverted and switch places.
- Leads aVR and aVF switch places; as a result Lead aVR becomes upright & Lead aVF becomes inverted.
- Lead aVL is unchanged.

These ECG changes can closely mimic a chronic phase inferior myocardial infarction due to inverted T waves and QS complexes in leads II, III and aVF. However, a high index of suspicion is required to detect lead reversal, as lead I and aVR will not be inverted from baseline in an inferior myocardial infarction. 15

In our case, the initial ECG of the patient done in cardiac OPD showed deep QS complexes & inverted T waves along with inverted p waves in leads II, III & aVF; lead I was also completely inverted and lead aVR was completely upright (P wave, QRS complex and T wave). These findings were consistent with reversal of right arm and left leg electrodes. However the subsequent supervised ECG done at CCU was completely normal which confirmed the diagnosis of an interesting case of ECG limb-leads reversal.

**Fig.-3:** Normal Einthoven’s triangle formed by six limb leads

**Fig.-4:** Rotation of Einthoven’s triangle due to RA-LL reversal

**Fig.-5:** 12 lead ECG depicting right arm and left leg reversal.
Conclusion:
ECG lead misplacement is a common and under-reported technical error. Most ECG interpretation books devote little if any space to this extremely important topic. Early & prompt recognition is imperative to avoid unnecessary, inappropriate & harmful therapeutic interventions to the patients. The 12-lead ECGs should always be interpreted in the context of patient’s history and clinical findings not relying solely on the ECG tracing. Measures like - ongoing education, obtaining repeat & supervised 12-lead ECG, double checking of lead placement by two staff members, reviewing old ECGs of the same patient and preserving ECG-strips containing the error for training purpose should be encouraged to aid in the detection, exclusion and further prevention of lead misplacement.

References:
Abstract:
Mitral stenosis is a valvular heart disease caused by a number of diseases. Chronic rheumatic fever is the most important cause. Among rare causes, some rheumatoid diseases like SLE may involve cardiovascular system causing Libman-Sacks endocarditis, pericardial diseases and other valvular lesions mostly associated with positive antiphospholipid and anticardiolipin antibody. Here, we presented a case of rheumatic mitral valvular heart disease having systemic lupus erythematosus but negative antiphospholipid and anticardiolipin antibody. Keywords: Rheumatic fever, Systemic lupus erythematosus, Mitral stenosis

Case Report
A Lady with Systemic Lupus Erythematosus and Mitral Stenosis
Muhammad Badrul Alam1, Sania Hoque2, Amiruzzaman Khan3, Md. Zakir Hossain4, Khondoker Asaduzzaman5

Introduction:
Rheumatic fever (ARF) is the most important cause of valvular heart diseases, but there are some rheumatoid diseases where heart valves are also involved. Systemic lupus erythematosus (SLE) is one of the chronic systemic autoimmune disease, associated with valvular heart diseases, Libman-Sacks lesions, serositis, pericardial disease, venous and arterial thrombosis. All these manifestations are mostly associated with antiphospholipid antibodies. In a study conducted on echocardiography of SLE patients, variable valvular diseases such as mitral stenosis, mitral valve thickening or vegetation, mitral valve prolapsed, mitral, aortic, and tricuspid regurgitation; were reported. Here we reported a known case of rheumatic MS with incidental findings of SLE with negative antiphospholipid and anticardiolipin antibody.

History: A young lady of 34 years, housewife, normotensive, nondiabetic, admitted into CCU of Mitford Hospital on 9th November 2015 with history of shortness of breath for 6 years, palpitation for 2 years, multiple joint pain & oral ulcer, skin rashes on face, arm & legs for 2 months (Fig: 1). She gave history of marked weight loss but there was no history of fever, spontaneous abortion, bleeding, venous thrombosis or convulsion. She had history suggestive of rheumatic fever 22 years back and repeated hospital admission without any improvement.

Fig-1: Characteristic rashes of SLE.

Clinical examination revealed tachycardia, dyspnoea, anaemia, oedema, skin rashes over face, legs, arms, raised JVP, apex beat in left 5th intercostal space tapping in nature, palpable P2, epigastric pulsation with left parasternal lift were present. There was loud first heart sound in mitral area with mid diastolic murmur and apansystolic murmur over left lower sternal edge. Liver was palpable and shifting dullness was present. Her both knees, ankles and small joints of both hands were swollen and tender. Investigations showed: Hb%-9.8gm/dl, anti-
nuclear Ab(ANA): positive, anti-ds DNA: positive, APL Ab: negative, anti-cardiolipin-Ab: negative, CCr-30.21ml/min, ECG: sinus Tachycardia (Fig:2), pericardial effusion on X-Ray chest P/A view (Fig:3). Echocardiography revealed severe mitral stenosis (MVA-0.59cm²) with thickening and calcification of both AML and PML with diastolic doming of AML with fused both Commisures, moderate subvalvular changes, moderate pericardial effusion (23mm) with moderate pulmonary hypertension (PASP-51mmHg) without any thrombus (Fig:4).

Fig.-2: ECG: Sinus tachycardia

Fig-3: X-Ray chest P/A view: Features of pericardial effusion.

Fig-4: Echocardiography (2D, M-Mode and Doppler): Severe MS (MVA-0.59cm²), subvalvular changes and pericardial effusion.
After diagnosis of SLE, hydroxychloroquine was added, subsequently patient developed visual impairment, fundoscopic examination found macular pigmentations.

Discussion:
Our patient was a known case of rheumatic valvular heart disease for last 22 years, as there was maculopapular skin rashes, investigations regarding SLE was done. SLE is an autoantibody and immune complex disorder, with immunoglobulin and complement deposition in involved organs, including the heart. The serologic findings may be detectable years before clinical disease manifests. In this patient valvular manifestations are mostly due to rheumatic fever because echocardiography showed mitral valve was severely stenosed (MVA-0.6cm²), both AML and PML were thickened and calcified with diastolic doming of AML, both commissures were fused, moderate subvalvular changes without any thrombus, all these findings are in favour of chronic rheumatic heart disease, whereas in SLE echocardiographic findings include diffuse thickening of valve leaflets with minimal subvalvular changes.

Transthoracic and transesophageal echocardiography had shown that valvular involvement is greater in SLE patients who have positive antiphospholipid antibodies. A number of other manifestations may occur in SLE, most of which are associated with antiphospholipid and anticardiolipin antibodies, such as venous and arterial thrombosis, recurrent fetal loss, pulmonary hypertension, endocardial disease seizures, and migraine. But in our patient antiphospholipid and anticardiolipin antibody were negative.

Studies of patients with SLE have reported a 25% incidence of clinically evident pericarditis, a 50% incidence of pericardial effusion (detected by echocardiography), and 80% incidence of pericardial abnormalities at autopsy. Here, in our patient, we also found moderate pericardial effusion (23mm) may be due pericarditis.

Over 95% of patients with SLE have a positive ANA; however, even high titers of ANA are not diagnostic of SLE. Anti–double-stranded DNA is more specific for SLE but is present in 50% to 70% of patients with idiopathic SLE, often in those with glomerulonephritis. Our case had strongly positive ANA and anti ds-DNA with impaired creatinine clearance (30.21ml/min).

There are reports of mitral and aortic valve replacement in patients with SLE. Valve repair has also been described. Our further planning for management of this patient is mitral valve repair &/or replacement.

We also found moderate pulmonary hypertension in this case, which is common in SLE but significant pulmonary hypertension is less common.

According to ACR/SLICC revised criteria for diagnosis of SLE, our patient had 9 points out of 16 (malar rash-2 points, oral ulcer-1 point, pericarditis-1 point, anemia-1 point, high titre ANA-2 points, positive anti ds-DNA-2 points and diagnosed a case of definite SLE.

The scope of investigations like renal biopsy, SPECT or endomyocardial biopsy were limited in this case, to find out further involvement of myocardium and kidney.

Our patient developed macular pigmentation on starting hydroxychloroquine that need further evaluation.

Conclusion: An uncommon case of SLE without positive antiphospholipid and anticardiolipin antibody along with rheumatic origin of mitral stenosis is presented here. It is recommended that two different entity of mitral valvular disease can co-exist.

References:


