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ISSN Print : 2617-5177
Online : 2617-5185

# Journal of Invasive and Clinical Cardiology

Volume 05 Issue 01 January 2023

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**Case Report**

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# Journal of Invasive and Clinical Cardiology




The Journal is published in the year 2021 by Professor (Dr.) Md. Afzalur Rahman on behalf of Journal of Invasive and Clinical Cardiology from Asian Colour Printing, 130, DIT Extension Road, Fakirerpool, Dhaka, Phone: 49357726, 58313186.





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# Journal of Invasive and Clinical Cardiology

## INSTRUCTION TO AUTHORS

**A. Introduction**

The Journal of Invasive and Clinical Cardiology is a biannual, peer-reviewed journal and aims to publish work of the highest quality from all sub-specialties of Cardiology. The aim of the publication is to promote research 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. 3 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.

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

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Only case reports of exceptional quality will be published in the case report format. The text should not exceed 1500 words and is arranged as introduction, case report and discussion. Include a brief abstract of about 150 words. Number of tables/figures should be limited to 3. Include up to 15 most recent references. The patient's written consent, or that of the legal guardian, to publication must be obtained.

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

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Letters commenting upon recent articles in Journal of Invasive and Clinical Cardiology 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.

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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 Journal of Invasive and Clinical Cardiology 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 Journal of Invasive and Clinical Cardiology and suggestions may be made to the authors by the Editorial Board to improve the scientific value of the journal.

**D. Editorial Process**

Journal of Invasive and Clinical Cardiology 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. Journal of Invasive and Clinical Cardiology follows the guidelines on editorial independence produced by the International Committee of Medical Journal Editors (ICMJE). All manuscripts correctly submitted to Journal of Invasive and Clinical Cardiology 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 Journal of Invasive and Clinical Cardiology, 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 Journal of Invasive and Clinical Cardiology 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 lowercase 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 U.S. National Library of Medicine (NLM) available at: https://www.nlm.nih.gov/mesh.

d. *Abbreviations*

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

**e.** *Acknowledgements*

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

f. *Units*

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

g. *Figures and graphics*

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

h. *Tables*

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

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

i. *References*

The authors are responsible for the accuracy and completeness of the references and their citations in the text.

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

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

Examples of correct forms of references are given below:

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

1. *Standard journal article*

   List the first six authors followed by et al.

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

   More than six authors:

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

2. *Organization as author*

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

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

   Vallancien G, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1,274 European men suffering

from lower urinary tract symptoms. J Urol. 2003;169(6):2257-61.

4. *Volume with supplement*

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

5. *Issue with supplement*

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

6. *Type of article indicated as needed*

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

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

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

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

**Books and Other Monographs**

1. *Personal author(s)*

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

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

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

3. *Organization(s) as author*

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

4. *Chapter in a book*

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

5. *Conference proceedings*

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

6. *Dissertation or thesis*

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

**Other Published Material**

*Newspaper article*

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

**Unpublished Material**

*In press or Forthcoming*

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

**Electronic Material**

1. *Journal article on the Internet*

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

Article published electronically ahead of the print version:

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

Article with document number in place of traditional pagination:

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

Article with a Digital Object Identifier (DOI):

Zhang M, Holman CD, Price SD, Sanfilippo FM, Preen DB, Bulsara MK. Comorbidity and repeat

admission to hospital for adverse drug reactions in older adults: retrospective cohort study. BMJ. 2009 Jan 7;338:a2752. doi: 10.1136/bmj.a2752. PubMed PMID: 19129307; PubMed Central PMCID: PMC 2615549.

2. *Monograph on the Internet*

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

3. *Homepage/Web site*

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

**G. Submission Preparation Checklist**

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

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

**H. Submission**

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

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# Stenting Left Main and Bifurcation Disease: From DK crush to Risk Assessment

**Shao-Liang Chen**

**J Inv Clin Cardiol 2023; 5(1): 1-6**

Left main (LM) disease is reported with angiography to be ~10%[1]. LM is segmented by ostial (proximal 3-5 mm), body (mid-segment, 5 mm in length), and distal (distal 5 mm). When LM length is <10mm, from the standpoint of percutaneous coronary intervention (PCI), most interventional cardiologists prefer to cover the whole LM segment using a drug-eluting stent (DES) unless a proximal landing zone is pretty clear. Among isolated distal LM lesions, >80% of disease[2] is extended to left anterior descending artery (LADN). Of overall LM lesions, ~85% involves both LAD and left circumflex (LCX), forming distal LM bifurcation lesions[3].

Criteria for LM treatment[2,4] are: 1) LM diameter stenosis ≥70% by angiography; 2) minimal luminal area (MLA) ≤6.0 mm$^2$ by intravascular ultrasound (IVUS) or optical coherence tomography (OCT); or 3) fractional flow reserve (FFR) ≤0.80. While the impact of LM disease locations on clinical outcome after coronary artery bypass graft (CABG) can be largely ignored, how to stenting distal LM lesions is a key issue. Of a total of 2,775 patients with isolated ostial/midshaft lesions in an unprotected LM disease enrolled in the DELTA multinational registry[5], at a median follow-up period of 1,293 days, there were no significant differences in the propensity score-adjusted analyses for the composite endpoint of all-cause death, myocardial infarction (MI), and cerebrovascular accident between PCI and CABG groups, with an exception of higher rate of target vessel revascularization (TVR) in the PCI arm. For entire cohort of LM disease, in a recent meta-analysis[6] including 29 studies extracted with 21,832 patients (10,424 in PCI vs 11,408 in CABG), the pooled analysis demonstrated remarkable differences in ≥1 year follow-up major adverse cardiac and cerebrovascular event, TVR, and MI, favoring CABG over PCI. Obviously, it is resumed that LM distal lesions is mainly correlated with increased clinical events after PCI. This finding is in line with more recent two large clinical trials[7, 8] with >3-year follow-up. In the EXCEL trial[7], in which 1905 patients with ULMCAD and low or intermediate SYNTAX scores were randomized to PCI with second-generation everolimus-eluting stents vs. CABG, ~80% of patients had disease of the distal LM bifurcation, most commonly treated with a provisional stenting (PS) approach. Although PCI provided comparable 3-year composite rates of death, myocardial infarction (MI) or stroke compared to CABG, repeat revascularization rates after 30 days were higher with PCI. In the NOBLE trial[8], ~80% of patients also had distal LM involvement, again most often treated with PS strategy. In NOBLE, PCI with an earlier generation DES resulted in a higher composite rate of death, MI, stroke or TVR at 5 years than CABG.

The PS approach to true bifurcation lesions consists of a DES to the main branch and balloon angioplasty of the side branch (SB), with stenting of the SB (usually with a T technique) reserved for a suboptimal balloon result. Therefore, whether alternative approaches to the distal LM bifurcation might afford superior results is unknown. In this issue of AsiaIntervention, Dr. Stankovic and coworker[9] have systematically analyzed the similarities and differences between PS and upfront two-stent approaches for LM bifurcation. In this writing there is no more “disclosures” about the comparison of PS versus two-stent treatments. However, the following issues remain to be programmatic.

Is LM bifurcations’ complexity influencing clinical outcome after PCI? 2018 ESC/EACTS guidelines on myocardial revascularization[10] recommended the use of systematic two-stent for true coronary bifurcation lesions if large SB (≥2.75 mm in diameter) with a long ostial SB lesion (>5 mm), anticipated difficulty in accessing an important SB after MV

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stenting, and true distal LM bifurcations. Widespread agreement is lacking. In 2014, the DEFINITION criteria of complex bifurcation lesions[11] were developed from a large bifurcation cohort (n=1550 patients) and subsequently validated in a 3660-patient study. Significant reductions in mortality and in-hospital adverse events were observed in patients with complex bifurcation lesions so defined treated with routine two-stent techniques. Subsequent DEFINITION II trial[12] showed that a planned 2-stent strategy significantly reduced the incidence of 1-year target lesion failure (TLF) compared with provisional stenting, driven by fewer target vessel MI (TVMI) and clinically-driven target lesion revascularization (TLR). In that study, ~80% of two-stent techniques was DK crush, leading to the conclusion that DK crush is the winner. In fact, DKCRUSH V[13], the 2nd randomized trial comparing DK crush with PS for LM distal bifurcation lesions, has reported the significant reduction of 1- to 3-year TLF in patients with complex bifurcations stratified by DEFINITION criteria, supported by a recently published retrospective study[14]. Altogether, DEFINITION criteria consisting of angiographic parameters provide the reliability of separating simple from complex bifurcation lesions and the prediction value for the occurrence of clinical events after LM bifurcation PCI.

What is the internal difference between culotte and double kissing (DK) double crush? Culotte stenting is used to be and continues to be the main techniques of systematic two-stent approach for true coronary bifurcation lesions. In the DKCRUSH III study[15], patients in the Culotte group had significant higher 1-year major adverse cardiac event (MACE, including cardiac death, MI, and TVR), mainly driven by increased TVR, compared with the DK crush. Interestingly, the 1-year MACE rate after culotte for LM bifurcation lesions was similar between DKCRUSH III (16.3%) and EBC Main (17.7%, all-death in this trial)[16] trials. Furthermore, at 3-year follow-up of DKCRUSH III study[17], the difference in MACE between culotte and DK crush group was widened, accompanied with extreme higher rate of stent thrombosis in the culotte arm. As a result, culotte stenting approach should be moved from the list of upfront two-stent techniques for treatment of LM bifurcation lesions.

Why higher rate of periprocedural MI (PMI) after PS approach? Post-stenting MI consists of PMI and spontaneous MI. Spontaneous MI rate is comparable between PS and two-stent[12-16], however, PMI was significantly higher in the PS arm from DKCRUSH V[13] and DEFINITION II[12] trials. In a total of 405 patients with 405 bifurcation lesions who underwent pre-procedure OCT imaging of both the main vessel (MV) and the SB[18], vulnerable plaques were predominantly localized in the MV and were more frequently in the long SB (≥10 mm) lesion group (42.7%) than in the short SB lesion group (24.2%, $p < 0.001$). At 1-year follow-up after provisional stenting, there were 31 (7.7%) TVMIs, with 21 (11.8%) in the long SB lesion group and 10 (4.4%) in the short SB lesion group ($p = 0.009$). Multivariate regression analysis showed that long SB lesion length, vulnerable plaques in the polygon of confluence, and true coronary bifurcation lesions were the three independent factors of TVMI. Obviously, SB lesion length plays an important role in stenting selection and predicting worse events (Table I), consistent with recent meat-analysis[19].

**Table-I**

*Correlation of side branch lesion length with worse event at 1-year after provisional stenting*

| 1-year F/U | Cardiac death | TVMI | TLR | TLF | ST |
|---|---|---|---|---|---|
| SB lesion length < 5-mm | 0.8% | 0 | 2.1% | 2.5% | 0 |
| SB lesion length = 5 mm but <10-mm | 1.3% | 3.3% | 4.5% | 6.6% | 0 |
| SB lesion length ≥10 mm | 2.2% | 6.1% | 8.4% | 13.4% | 2.7% |

*Unpublished data from DKCRUSH II, DKCRUSH V, and DKCRUSH VI studies.*

What is the correlation of PMI with mortality after bifurcation stenting? PMI refers to myonecrosis following PCI using DES. Its rate varies from 1.1% to 55.9% depending on the types and cut-off values of biomarkers and additional EKG criteria or clinical symptoms. The pathophysiology of PMI is multifactorial and includes distal embolization of thrombus or plaques, dissection, spasm, and occlusion of small SBs. In 1,971 patients with true coronary bifurcations who underwent DES implantation in the DEFINITION trial[11], we reported that 1-year mortality was significantly higher in the PMI group (defined as creatinine kinase [CK]-myocardial band [CK-MB]>3 times over the upper normal limit [UNL], 6.4%) group than in the non-PMI group (1.7%). Among 1300 patients with both CK and CK-MB measurements pre- and post-stenting were evaluated from four DKCRUSH studies[20], Sheiban and coworkers reported that 56 (4.3%) patients had PMI. According to SYNTAX, 4th UDMI or ISCHEMIA, SCAI, and EXCEL definitions (Table II), PMI occurred in 21 (1.6%), 56 (4.3%), 29 (2.2%), and 32 (2.5%) patients, respectively. All definitions were significantly correlated with unadjusted mortality at the end of follow-up but not at 30 days or 1-year after stenting. PMI using SYNTAX, SCAI, and EXCEL definitions rather than 4th UDMI definition was strongly associated with adjusted all-cause death. By adjusted analysis, PMI according to 4th UDMI, SCAI, and EXCEL definitions but not SYNTAX definition was positively correlated with cardiac death at a median of 5.58 years of follow-up. CK-MB ≥5 x UNL strongly enhanced the correlation of CK-MB values with mortality (Table III). Accordingly, intravascular imaging-guidance of bifurcation stenting is critical in improving clinical outcomes.

**Table-II**

*Components of definitions for peri-procedural myocardial infarction*

| | Component of definitions |
|---|---|
| SYNTAX definition | New Q waves in ≥2 leads |
| | - peak CK-MB/peak total CK >10% |
| | - CK-MB > 5 x UNL |
| 4th UDMI definition | CK-MB > 5 x 99% percentile UNL |
| | - new ischemic ECG changes |
| | - new Q waves |
| | - flow-limiting complications |
| | - new loss of viable myocardium |
| | - new wall motion abnormally |
| ISCHEMIA definition | CK-MB > 5 x UNL |
| | - ST-segment elevation or depression |
| | - new Q waves |
| | - persistent LBBB |
| | - new TIMI flow 0/1 in major vessel or SB |
| | - NHLBI ≥type C dissection |
| | CK-MB > 10 x UNL |
| SCAI definition | CK-MB ≥5 x UNL |
| | - new Q waves |
| | - persistent LBBB |
| | CK-MB > 10 x UNL |
| EXCEL definition | CK-MB > 5 x UNL |
| | - new Q waves |
| | - persistent LBBB |
| | - occlusion, new thrombosis or TIMI <3 |
| | - new loss of viable myocardium |
| | - new regional wall motion abnormally |
| | CK-MB > 10 x UNL |

**Table-III**

*Association of CK-MB values with all-cause or cardiac death at the end of follow-up*

| CK-MB increases (x ULN) | All-cause death | | | Cardiac death | | |
|---|---|---|---|---|---|---|
| | Event, N (%) | HR | 95% CI | Event, N (%) | HR | 95% CI |
| CK-MB < 1 x UNL | 46/577 (8.0) | - | - | 25/577 (4.3) | - | - |
| 1 x UNL ≤CK-MB < 3 x UNL | 61/598 (10.2) | 0.63 | 0.36-1.10 | 38/598 (6.4) | 0.59 | 0.30-1.13 |
| 3 x UNL ≤CK-MB < 5 xUNL | 8/68 (11.8) | 1.08 | 0.49-2.35 | 4/68 (5.9) | 0.81 | 0.28-2.32 |
| CK-MB ≥5 x UNL | 11/57 (19.3) | 2.07 | 1.02-4.18 | 9/57 (15.8) | 2.79 | 1.28-6.06 |

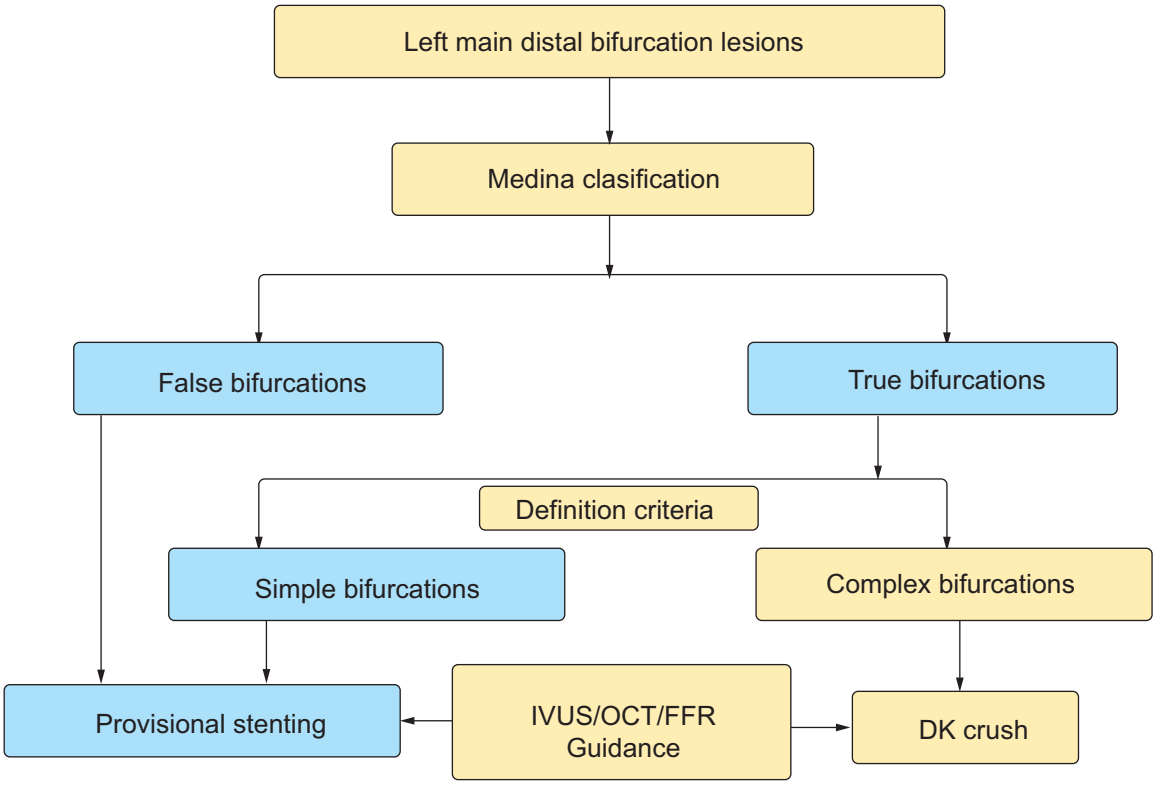


**Fig.-1:** *Algorithm of stenting left main distal bifurcation lesions*

In conclusion, stenting LM bifurcation lesions is technically demanding. Careful assessments according to angiography, intravascular images, and or FFR are key points of device and approach selection (Figure 1). Quality of stenting procedures determines the short- and long-term clinical outcomes. DK crush is associated with less frequent worse clinical events, particularly for complex bifurcation lesions defined by DEFINITION criteria.

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# Association of Diabetic Retinopathy with Angiographic Severity of Coronary Artery Disease in Patients with Non-ST Elevation Myocardial Infarction

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**Abstract:**

**Background:** Bed side ophthalmoscopic examination is a simple measure of diagnosis of diabetic retinopathy and has been shown to be a predictor of poor outcome in various cardiovascular conditions including coronary artery disease (CAD)**.** Retinopathy lesions are fairly common findings in clinic settings and may predict risk of coronary heart disease (CHD). The present study was intended to find the relationship between diabetic retinopathy with the severity of coronary artery disease in patients with NSTEMI.

**Methods:** This cross-sectional observational study was conducted in the Department of cardiology, National Institute of Cardiovascular Diseases and Hospital (NICVD), Dhaka, Bangladesh, from March 2019 to August 2020. A total of one hundred twenty DM with NSTEMI patients undergoing coronary angiogram and also fundoscopic examination with fundal photography during the index hospitalization were included in this study. Study subjects were divided into two groups on the basis of diabetic retinopathy (Group-I: NSTEMI with diabetic retinopathy ; Group- II: NSTEMI without diabetic retinopathy). Severity of coronary artery disease was determined by Gensini score and correlation between diabetic retinopathy and Gensini score was assessed.

**Results:** Gensini score was significantly higher in patients with diabetic retinopathy than that in patients without diabetic retinopathy (62.2±27.7 vs. 43.3±25.3,p<0.001).Gensini score increased with increasing severity of diabetic retinopathy(P <0.001).The risk of having severe CAD in patient with diabetic retinopathy was 13.03 (95% CI =2.410-70.419) (P<0.003).A significant correlation between diabetic retinopathy and Gensini score was noted (p value <0.001)

**Conclusion:** It may be concluded that Presence and severity of diabetic retinopathy is associated with angiographically severe coronary artery disease in patient with non-ST elevation myocardial infarction (NSTEMI) and it may be considered as an independent predictor of severity of CAD.As is a bed side assessment ,so before performing coronary angiography ,it appears to be additive for risk stratification.

**Keywords:** •Coronary artery disease •Diabetic retinopathy •Left ventricular ejection fraction •Gensini score •TVD •RCA •LCX

**J Inv Clin Cardiol 2023; 5(1):** 7-13

## Introduction:

Cardiovascular diseases (CVDs) are the most common cause of premature death in the world. CVDs account for 50% of all non-communicable disease (NCD) deaths in the world each year and represent a significant threat to human welfare and sustainable development[1]. The exact prevalence of CAD in Bangladesh is not known. Only a limited number of small-scale epidemiological studies are available. More recent data indicates that CAD prevalence is 1.85% to 3.4% in rural population and 19.6% in an urban population[1].

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Diabetes Mellitus is a heterogeneous primary disorder of carbohydrate metabolism with absolute insulin deficiency (Type 1) or relative insulin deficiency (type 2), resistance or both leading to hyperglycemia[3]. The South Asian region shares a major proportion of this worldwide burden of diabetes. The prevalence of diabetes ranges from 0.9% in Bangladesh to 21.2% in India[4].

Diabetic retinopathy (DR) is an early and frequent marker of other vascular complications of diabetes and its relation with coronary ischemia is known[5]. Unstable angina patient with DR have more significant left ventricular dysfunction than without DR[6]. In individuals with type-2 diabetes, the presence of retinopathy signifies an increased CHD risk, independent of glycemic levels, symptomatology[7], other cardiovascular risk factors and is also associated with an increased risk of mortality and cardiovascular events[8].

While its adverse impact on vision is well known[9], the importance of retinopathy signs beyond visual impairment is less well recognized. Both non proliferative diabetic retinopathy(NPDR) and proliferative diabetic retinopathy(PDR) have now been linked with major clinical diseases like stroke, coronary heart disease, heart failure and nephropathy[10], as well as newer subclinical measure of cardiovascular disease such as coronary artery calcification and cardiac remodeling[11]. The presence of retinopathy signs has also been associated with higher degree of coronary artery calcification[12] and more diffuse/severe coronary artery stenosis on angiograms[13].

Atherosclerosis Risk in Communities study( ARIC) showed that the presence of retinopathy was associated with two-fold higher risk of incident of coronary heart disease (and myocardial infarction), three-fold higher risk of fatal coronary heart disease, and four-fold higher risk of congestive heart failure, independent of diabetes duration ,glycemic control ,smoking ,lipid profile ,and other risk factor.There is a graded ,dose dependent association of increasing diabetic retinopathy severity with increasing coronary heart disease risk[14]. The World Health Organization Multinational Study of Vascular Disease in Diabetes(WHO-MSVDD)[15] and other studies reported associations of not only NPDR but also PDR with ischemic heart disease[16].

In addition to population studies ,there are clinical studies that suggest the presence of retinopathy can be used as an indicator of silent myocardial ischemia and help guide investigative approaches in diabetic patients with suspected heart disease [17].

Coronary microcirculation dysfunction associated with diabetes, although explored extensively in recent years,[18] still represents a poorly understood phenomenon in the clinical setting. Endothelial dysfunction, with its unfavorable consequences in various vascular beds, has been widely recognized to be a result of pathophysiological processes in diabetes, with less information available in the context of the coronary microvasculature[18].

The Gensini score system is a technique developed by Gensini et al[19], for the assessment of the severity of coronary artery disease (CAD). This scoring system is based on the artery morphology, coronary anatomy, and severity of stenosis in lesions[20].

Clinicians are in constant search of a non-invasive, practical and precise tool to predict severity of coronary disease. If the association between diabetic retinopathy and the Gensini score is found, it can readily be used as a tool to predict severe CAD. The purpose of this study is to search for whether increased diabetic retinopathy is associated with increased angiographic severity in non-ST elevation myocardial infarction patients.

**Methods:**

**Study patients:**

One hundred twenty cases were selected from consecutive type 2 DM patients with NSTEMI who underwent coronary angiography between March 2019 to August 2020 and cases were divided into two groups on the basic of presence or absence of diabetic retinopathy. Type 2 DM was diagnosed according to the recommendations of the American Diabetes Association. Exclusion criteria were known duration of DM of less than 1 year, type 1 diabetes mellitus, uncontrolled hypertension (systolic blood pressure > 180 mmHg and/or diastolic blood pressure> 100 mmHg), previous coronary bypass surgery, and known nondiabetic retinal disease

**Coronary angiography:**

Selective coronary cineangiography was performed from the radial or femoral approach using Judkin's technique. Multiple views were obtained in all patients,

with the left anterior descending and left circumflex coronary arteries visualized in at least four views and the right coronary artery in at least two views by using cine-angiographic equipment. Coronary angiograms were scored according to followings-Severity score: This was the Gensini score, which has been described previously. Briefly, the coronary arterial tree was divided into segments with multiplying factors according to the functional importance of any given segment (5 for the left main trunk to 0.5 for the most distal segments) and the percent reduction in lumen diameter of each narrowing was assigned a score (0, 1, 2, 4, 8, 16, 32 according to the degree of stenosis). The sum of the scores of all segments gives the Gensini score, which places emphasis on the severity of the disease.

**Detection of Diabetic Retinopathy (DR):**

Fundoscopic examination was done one day after admission. Initially tropicamide 1.0% eye drop was given in both eyes. After full dilatation of both pupils fundoscopic examination was done by Keeller ophthalmoscope for detection of diabetic retinopathy. Then next day fundal photo was done and assessed by an ophthalmologist. Patients were divided into two groups on the basis of presence or absence of diabetic retinopathy. Those with presence of retinopathy categorized as group I and absence of retinopathy as group II.We also evaluated risk factors for CAD, which included patient age, sex, duration of diabetes, hypertension, hypercholesterolemia, smoking status, history of a cerebrovascular accident or peripheral arterial disease, and family history of CAD. Subjects who had quit smoking for over 5 years were considered non smokers.The therapeutic modality for DM (oral antidiabetics or insulin) and history of previous myocardial infarction (MI) were also recorded. Biochemical parameters including blood fasting glucose, serum total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), creatinine were noted.

**Statistics:**

Results are expressed as the mean ± SD. For univariate analysis, the significance of differences between the two groups for continuous variables was assessed with the unpaired Student's t test, while the chi-square test was used for nominal variables. Comparisons of the severity of CAD with severity of

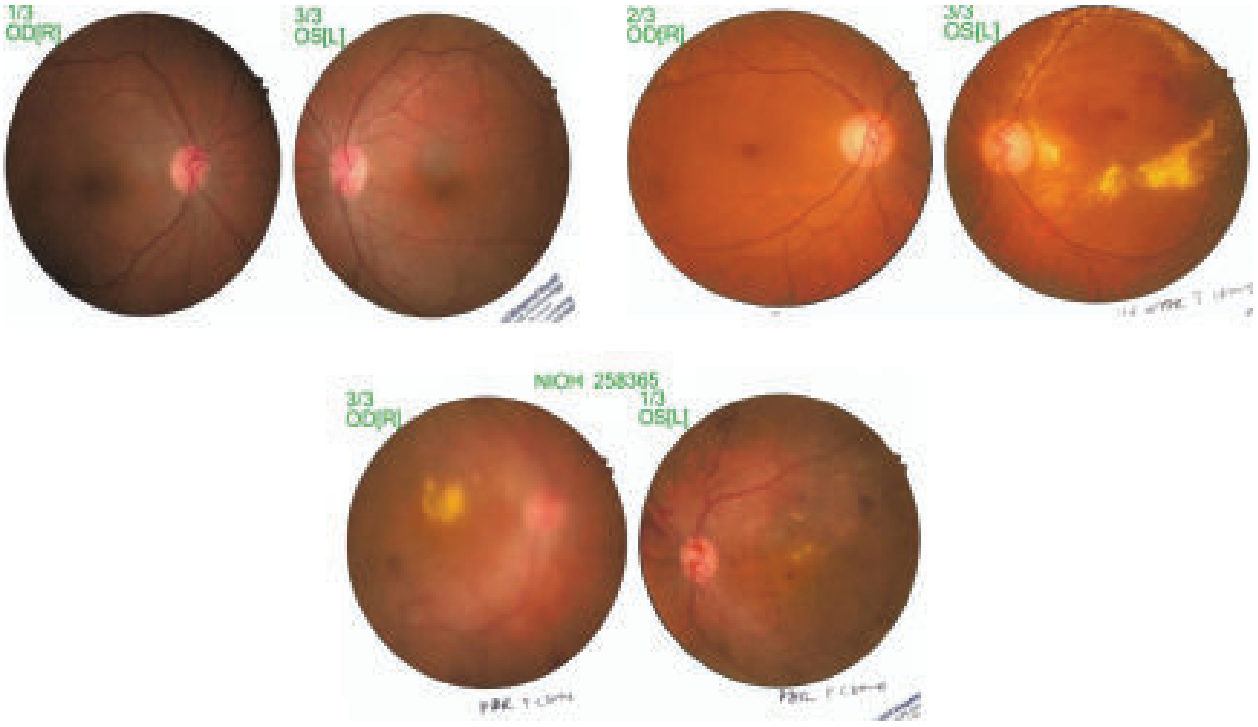


**Fig.-1:** *No diabetic retinopathy, Mild and moderate non proliferative diabetic retinopathy and Proliferative diabetic retinopathy*

diabetic retinopathy were performed with 2-way ANOVA using appropriate covariates (with parameters having significant differences in univariate analysis). The correlations between the CAD severity with other parameters were analyzed using simple correlation analysis. Multivariate associations of the CAD severity and extent scores were determined by performing multiple stepwise linear regression analysis with parameters having significant correlations in the simple correlation analysis. Statistical analyses were carried out by using SPSS 16.0 (Statistical Package for the Social Sciences by SPSS Inc., Chicago, IL, USA)

**Results:**

Diabetic retinopathy: According to the results of the fundus examinations, 60 (50%) of 120 patients had

DR (non proliferative mild in 16, moderate to severe in 22, and proliferative in 22) whereas the remaining

60 (50%) had no retinopathy.

Comparison of clinical characteristics and risk factors: The patients with DR had a significantly longer duration of known diabetes (P < 0.001), a higher ratio of being on insulin therapy (P = 0.03) in table 4.3, higher serum creatinine levels (P< 0.001 and P < 0.001, respectively).

**Table-I**

*Risk factors of the study patients (N=120).*

| Risk Factors | | Group I (n=60) | | Group II(n=60) | | p value |
|---|---|---|---|---|---|---|
| | | Number | % | Number | % | |
| Smoking | Yes | 26 | 43.3 | 31 | 51.7 | 0.36NS |
| Hypertension | Yes | 35 | 58.3 | 38 | 63.3 | 0.57NS |
| Dyslipidemia | yes | 19 | 31.7 | 14 | 23.3 | 0.31NS |
| Family H/O of premature CAD | | 13 | 21.7 | 6 | 10.0 | 0.08NS |
| Previous H/O of PAD | | 4 | 6.7 | 4 | 6.7 | 1.00NS |
| Previous H/O of CVD | | 4 | 6.7 | 3 | 5.0 | 0.69NS |
| Mean duration of DM (yrs) | | 11.8±5.7 | | 5.7±1.7 | | <0.001S |
| No. of patients taking insulin | | 22 | 36.7 | 8 | 13.3 | 0.003S |

Here,Group I=Patients with diabetic retinopathy, Group II= Patients without diabetic retinopathy p value reached from chi-square test for qualitative variables and t-test for quantitative variables. S= Significant (p >0.05)

**Table-II**

*Distribution of the study patients by biochemical status (N=120).*

| Biochemical parameters | Group I(n=60) Mean±SD | Group II (n=60) Mean±SD | p value |
|---|---|---|---|
| RBS (mmol/L) | 16.1±8.6 | 14.1±9.5 | $0.37^{NS}$ |
| Serum Creatinine (mg/dl) | 1.31±0.22 | 1.04±0.20 | $<0.001^{S}$ |
| Troponin I ng/dl | 23.39±13.92 | 21.17±12.96 | $0.37^{NS}$ |
| Total Cholesterol (mg/dl) | 204.5±18.8 | 204.3±19.7 | $0.93^{NS}$ |
| LDL Cholesterol (mg/dl) | 130.6±16.6 | 129.0±21.7 | $0.65^{NS}$ |
| HDL Cholesterol (mg/dl) | 40.1±4.7 | 39.7±5.3 | $0.74^{NS}$ |
| TG (mg/dl) | 168.8±21.4 | 172.1±29.8 | $0.49^{NS}$ |

Here, Group I=Patients with diabetic retinopathy, Group II= Patients without diabetic retinopathy RBS=Random blood sugar, LDL= Low density lipoprotein, HDL= High density lipoprotein TG= Triglycerides p value reached from unpaired t test, ns = Not significant (p>0.05), S= Significant (p<0.05)

**Table-III**

*Distribution of the study patients by Gensini score (N=120)*

| Gensini Score | Group I (n=60) | | Group II (n=60) | | p value |
|---|---|---|---|---|---|
| | Number | % | Number | % | |
| Severe CAD (>36 points) | 54 | 90.0 | 31 | 51.7 | <0.001S |
| Not severe CAD (≤36 points) | 6 | 10.0 | 29 | 48.3 | <0.001S |
| Mean ± SD | 62.2±27.7 | | 43.3±25.3 | | <0.001S |

Here,Group I=Patients with diabetic retinopathy, Group II= Patients without diabetic retinopathy S=Significant (p<0.05) CAD= Coronary artery disease
p value reached from chi square test of categorical approach and unpaired t-test of quantitative approach.

**Table-IV**

*Association between Diabetic Retinopathy and Gensini score (N=120).*

| No. of vessel involved | Gensini Score | | P value |
|---|---|---|---|
| | Mean | SD | |
| No DR (n = 60) | 43.35 | 25.30 | |
| Mild DR (n=16) | 48.69 | 19.77 | <0.001s |
| Moderate DR (n=22) | 58.27 | 23.87 | |
| Severe DR (n=22) | 75.98 | 30.86 | |

S = Significant (p<0.05),p value reached from ANOVA test.
The table shows that the patients in the DR (+) group had significantly higher Gensini scores than patients in the DR(-ve) group.

**Prediction of CAD severity:**

**Table-V**

*Univariate logistic regression for determinants of severity of coronary artery disease as assessed by Gensini score.*

| Variables of interest | Regression coefficient(β) | p value | OR | 95% CI |
|---|---|---|---|---|
| Duration of Diabetes mellitus | 0.135 | 0.02S | 1.14 | 1.025 – 1.279 |
| Insulin taking | 0.164 | 0.03S | 1.17 | 1.05 – 2.976 |
| Serum creatinine | 1.982 | 0.005S | 4.46 | 1.435 – 28.620 |
| DR (+) | 2.131 | <0.001S | 8.41 | 3.148 – 22.517 |
| Smoking | - 0.321 | 0.286 | 0.725 | 0.414 – 1.271 |
| HTN | 0.006 | 0.292 | 1.006 | 0.567- 1.784 |
| Dyslipidemia | - 0.120 | 0.703 | 0.887 | 0.478 – 1.645 |

Dependent variable: Gensini Score>36 points; Independent variables: duration of diabetes mellitus, insulin taking, serum creatinine, DR (+) ,S = Significant, NS = Not significant

The above table depicts the univariate logistic regression analysis of odds ratios for characteristics of the subjects likely to develop coronary artery disease. The variable diabetic retinopathy, duration of diabeticmellitus, creatinine were found to be significantly associated with CAD severity with their odd ratios being 8.41,1.14 and 4.6 respectively.

**Table-VI**

*Multivariate logistic regression for determinants of severity of coronary artery disease as assessed by Gensini score.*

| Variables of interest | Regression coefficient (β) | p value | OR | 95% CI |
|---|---|---|---|---|
| Duration of Diabetes mellitus | -0.125 | 0.21NS | 0.88 | 0.725 – 1.075 |
| Insulin intake | -0.480 | 0.47NS | 0.62 | 0.164 – 2.331 |
| Serum creatinine | 0.833 | 0.48NS | 2.29 | 0.219 – 24.144 |
| Presence of DR | 2.567 | 0.003S | 13.03 | 2.410 – 70.419 |

Dependent variable: Gensini score>36 points; Independent variables: duration of diabetes mellitus, insulin intake, serum creatinine, Presence of DR ,S = Significant, NS = Not significant

The above table demonstrates the multivariate logistic regression analysis of odds ratio (OR) for characteristics of the subjects likely to cause of high Gensini Score assessed as coronary artery disease severity. The variables revealed to be significantly associated with high Gensini score by multivariate analysis were entered into the model directly. The table depicts that DR (+) was found to be the significant predictor of high Gensini Score with OR being 13.03.

**Discussion:**

This study evaluated the association of diabetic retinopathy with angiographic severity of coronary artery disease in patients with Non-ST Elevation Myocardial Infarction. The mean age of the patients in group I and group II were 57.0±6.9 years vs. 56.9±6.8 years respectively. This is almost similar to the study done by Norgaz et al[6]. In this study ,male patients were 73.3% vs. 66.7% in group I and group II respectively.In the study by Norgaz et al., male patient in both the groups were 45% and 55% respectively[6]. The present study showed a smaller number of women than men in both the groups. Only 26.7% and 33.3% respectively.This gender disparity is multi factorial: less predisposition to CAD in reproductive age,overall, less health care seeking attitude of female patients and less attention by male counterparts of the family.Regarding CAD risk factors in this study, smoking, hypertension, dyslipidemia,family history of premature CAD, previous history of PAD,previous history of CAD and obesity, presented in the above table did not differ significantly between two groups. Only duration of diabetes and number of patient taking insulin were found significantly higher in group I than group II. This is almost similar to study done by Saleem et al., 2017[21].

In the present study,Gensini score of NSTEMI patients ,who underwent CAG, differed between no diabetic retinopathy ,mild diabetic retinopathy to severe diabetic retinopathy. The mean Gensini score in group I 62.2 ± 27.7 versus 43.3 ±25.3, P< 0.001.Norgaiz et al. studied the relationship between diabetic retinopathy and CAD severity by Gensini score (6). In Saleem et al 2017, it was shown that the patients with diabetic retinopathy had significantly higher vessel (2.62 ± 0.60 versus 1.9 ± 1.03, P< 0.001) and severity (103 ± 37.17.0 versus 38.55 ±22.20, P< 0.001) score than patients with no evidence of diabetic retinopathy.They demonstrated that higher the diabetic retinopathy also have severe coronary artery disease.Patient without diabetic retinopathy have mean Gensini score 43.35 ± 25.30,mild diabetic retinopathy 48.69 ± 19.77 ,moderate retinopathy score 58.27 ± 23.87 and severe diabetic retinopathy

75.98 ± 30.87 and the difference of diabetic retinopathy between the subgroups were significant. Apart from this Rong J et.al 2013., investigate association between diabetic retinopathy and CAD severity by Gensini score .They found that the prevalence of coronary atherosclerosis, is significantly higher in the patients with T2DM with DR compared with those without DR[22]. So, the overall findings relating to the association between diabetic retinopathy and CAD severity correlates with the findings of present study.In this study significant positive correlation was also found between diabetic retinopathy and Gensini score.

In the present study, univariate logistic regression analysis of the variables likely to cause severe CAD (Gensini score>36) was done.The univariate regression analysis revealed that the odds ratios of diabetic retinopathy ,duration of diabetic, number of patient

taking insulin, s.creatinine were statistically significant and independently associated with severe CAD with Gensini score >36. However, when these parameters were analyzed in multivariate logistic regression analysis, only diabetic retinopathy found to be independent determinants of severe CAD. After comparing the findings of present study with other studies,it can be summarized that there is significant correlation between diabetic retinopathy and CAD severity.

**Conclusion:**

It may be concluded that Presence and severity of diabetic retinopathy is associated with angiographically severe coronary artery disease in patient with non-ST elevation myocardial infarction (NSTEMI) and it may be considered as an independent predictor of severity of CAD.As is a bed side assessment, so before performing coronary angiography, it appears to be additive for risk stratification.

**Conflict of interest- None**

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ORIGINAL ARTICLE

# Evaluation of Left Ventricular Diastolic Dysfunction in diabetic patients with preserved Ejection fraction and its association with myocardial performance index

**Bishal Shrestha[1], Rabi Malla[2] , Arun Maskey[2], Sujeeb Rajbhandari[2] , Rabindra Simkhada[2] , Arjun Budhathoki [1], Chitra Raj sharma [1], Manoj Koirala[1] , Divya Karmacharya[2], Eloma Shrestha[2], Sunita Sharma [3]**

## Abstract

**Background:** Diastolic dysfunction causes impairment of ventricular filling when the ventricle becomes stiff, relaxes slowly or incompletely. Diabetes mellitus increases the risk of diastolic dysfunction which can develop heart failure even in the absence of other co-morbidities. Tei index is an echocardiographic tool to evaluate global function of ventricle.

**Objective:** To determine the prevalence of Left Ventricular diastolic dysfunction (LVDD) in patients with type 2 diabetes mellitus; and its association with LV myocardial performance index (Tei Index).

**Method:** 100 patients with type 2 diabetes mellitus with normal left ventricular ejection fraction without overt cardiac disease were included. LVDD was determined as per using 2016 American Society of Echocardiology (ASE) guideline. LV Tei index was calculated by tissue doppler imaging method.

**Results:** Mean age of the patients was 58.1 ± 12.6 years with 54 percent male. Mean HbA1C was 7.45± 0.99. 23 (23%) patients met the criteria of LVDD and 46 (46%) patients did not have LVDD. Whereas 31 (31%) of patients were categorized as indeterminate. Tei index was significantly higher in patients with LVDD (0.56±0.05 and 0.43±0.06 ($p < 0.05$).

**Conclusion:** Prevalence of LV diastolic dysfunction in type 2 diabetes mellitus was 23% in our study. There was a significant association between LVDD and Tei index. Rising trend of prevalence of diastolic dysfunction with increasing age of patients was observed.

**Key Words:** *2016 ASE/EACVI guideline*, *Diastolic dysfunction, diabetes mellitus, MPI, Tei index, Tissue doppler imaging*

**J Inv Clin Cardiol 2023; 5(1): 14-18**

## Introduction:

Cardiac dysfunction is the major cause of morbidity and mortality in diabetes worldwide[1].Diabetes is a major risk factor not only for CAD, but also for left ventricular dysfunction and heart failure[2]. In the Framingham Heart Study, it was shown that HF was twice as common among men and five times as common among women with diabetes as among those without diabetes[3]. Diabetic cardiomyopathy is characterized by the development of diastolic dysfunction at the early stage, followed by systolic dysfunction in the absence of coronary artery disease, hypertension, or significant valvular heart disease[4]. Type 2 diabetes seems to be more strongly associated with the development of HFpEF than with HFrEF. In line with these findings, left ventricular diastolic dysfunction (LVDD), the preclinical stage of HFpEF, is also more prevalent among type 2 diabetes patients than in those without diabetes[5].

Although type 2 diabetes is a known risk factor of LVDD and HFpEF, the use of echocardiography is in general not considered in existing type 2 diabetes primary care disease management programs.[6] Given the large impact of both diabetes and HFpEF for

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patients and community, it is important to know the exact prevalence of LVDD in patients with type 2 diabetes as this can be helpful to target prevention and intervention strategies for both LVDD and early stages of HFpEF.

American society of echocardiography/European association for cardiovascular imaging ASE/EACVI jointly updated its complex 2009 guideline for detection of LVDD in 2016[7]. The primary goal of this update is to simplify the approach and thus increase the utility of the guidelines in daily clinical practice.

Myocardial Performance Index (MPI/Tei Index), which includes both systolic and diastolic time intervals to assess the global cardiac dysfunction was used by Tei and his co-workers in 1995[8]. The value is independent of Heart Rate and Blood Pressure. It has been evaluated in many cardiac conditions like low heart failure with reduced ejection fraction, Pulmonary Arterial Hypertension, Amyloidosis, Myocardial infarction, congenital heart disease[9]. The cut off value is different among normal and various abnormal cardiac conditions and Higher value of Tei index has been shown to correlate with poor prognosis in symptomatic HF[10]. It has also been used as a surrogate marker for diabetic cardiac dysfunctions in different studies with encouraging results. Tei index can be calculated from pulse wave doppler method and tissue doppler imaging method. Tissue Doppler Imaging (TDI) enables measurement of both relaxation and contraction time simultaneously in single cardiac cycle[11].

Current study aims to determine the prevalence of LVDD as per updated 2016 ASE/EACVI guideline in our diabetic patients having preserved EF with no evidence of overt cardiac disease and find out the association of LVDD with Tei index.

**Methodology:**

This is hospital based, cross sectional, prospective study conducted in Bir Hospital, Kathmandu, Nepal from 2022 February to 2022 August (6 months) with IRB clearance from the institute (NAMS). Diabetic patients with sinus rhythm, normal EF, with no gross structural heart disease (more than mild valvular disease, HCM, DCM, RCM, pericardial disease), without COPD, CKD, no ECG evidence of infarction or bundle branch block were included in the study. Informed consent was taken; Echocardiography was done, recent documents were reviewed and data were collected and recorded as per proforma by the principal investigator.

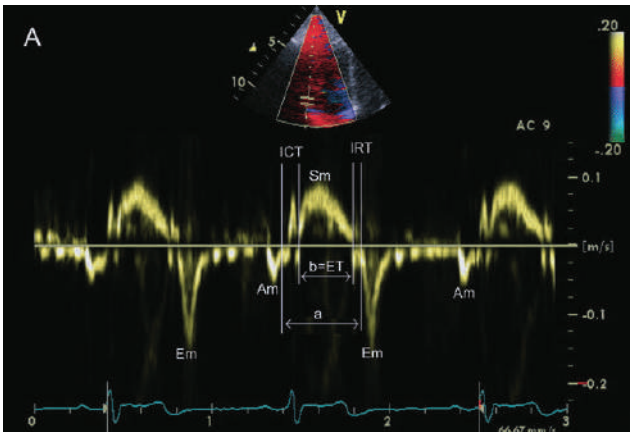
Following four variables are obtained on echocardiography (phillips affinity) as per ASE/EACVI 2016 guideline to determine whether the patient has LVDD. their abnormal cutoff values are:

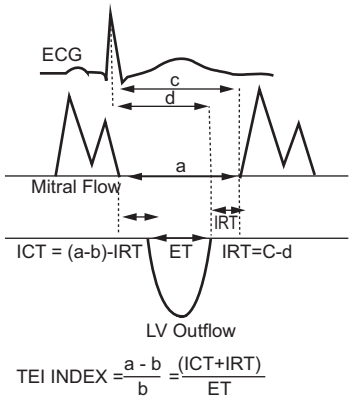
1) annular e' velocity (septal e' < 7 cm/sec, lateral e'< 10 cm/sec),
2) average E/e' ratio > 14,
3) LA maximum volume index > 34 mL/m2 calculated by biplane method and indexed with BSA,
4) Peak TR velocity > 2.8 m/sec.

LV diastolic function is considered normal if upto1 parameter is abnormal. LV diastolic dysfunction is present if at least 3 parameters are abnormal. The study is indeterminate if 2 values are abnormal.

Tei index was calculated from tissue doppler imaging method. The sample volume was placed on septal mitral annulus to get a good TDI signal as shown in figure 1 and schematic graphic representation. The interval "a" is the interval between cessation to onset of diastolic myocardial velocities. The interval "b": the ejection time (ET) is duration of systolic myocardial velocity (SMV). Tei index is calculated from following formula

MPI = (a-b)/b = (IVRT + IVCT)/ET.



**Data Analysis**

All data were entered into Microsoft Excel and the statistical analysis was done using the SPSS version 26 software (SPSS INC, Chicago, III). Categorical variables were analyzed as number and percentage, continuous variable with normal distribution is presented as mean ± SD. After processing of all available information, statistical analysis of their significance was done.

Age, duration of diabetes, and echo parameters were compared between different groups by performing unpaired *t*-test for normalized data. Categorical variables were compared by chi-square test. Pearson correlation test was used to correlate between MPI and LVDD association. 95% confidence interval was accepted for our study.

**Results**

Total 100 diabetic patients who met the inclusion and exclusion criteria were evaluated. Mean age of the patients was 58.1 ± 12:6 years with 54 percent being male. Mean duration of diabetes was 5.68 ± 5.7 years. and mean HbA1C was 7.45 ± 0.99 (available in 61 patients). (Table 1)

**Table-I**

*Demographic and Baseline characteristics of study population*

| Characteristic. | Mean± SD or. Number (%) |
|---|---|
| Age(years) | 58.14±12.58 years |
| Male (%) | 46 (44.2%) |
| Duration of Diabetes (years) | 5.68±5.7 years |
| HBA1C | 7.45±0.992 |

Using the criteria of 2016 American society of echocardiography for diagnosis of LVDD, 23 (23%) patients met the criteria of LVDD and 46 (46%) patients did not have LVDD, whereas 31 (31%) of patients were categorized as indeterminate (figure 1). LVDD was more prevalent with advanced age. There was no significant difference in LVDD vs no LVDD as per sex, duration of diabetes and recent HBA1C level. (Figure 2, 3).

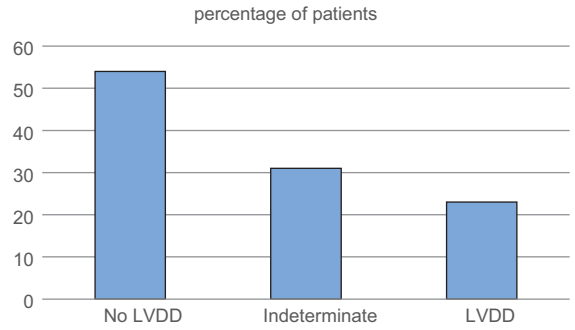



**Fig.-1:** *Prevalence of diastolic dysfunction*

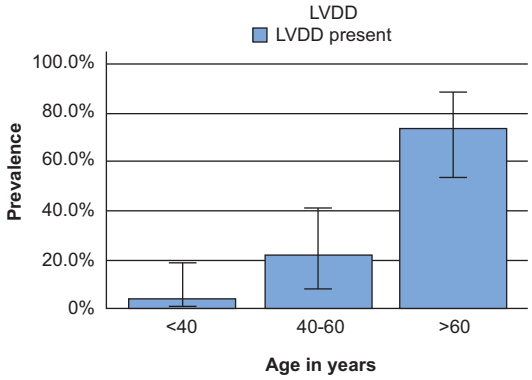



**Fig.-2:** *Association between age distribution and diastolic dysfunction*

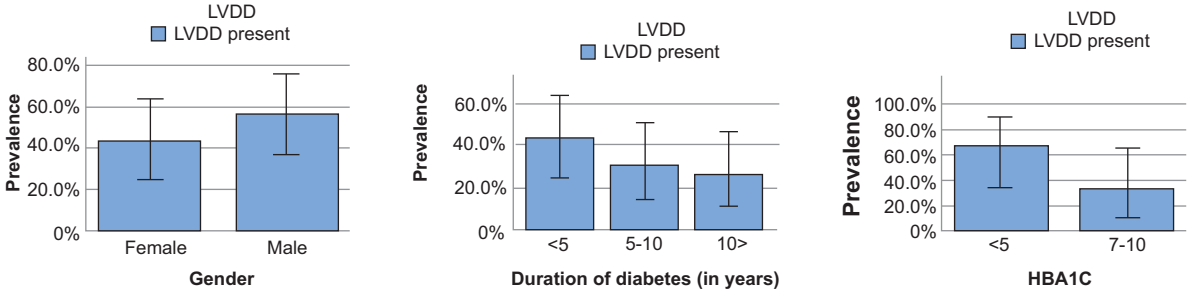



**Fig.-3:** *Association between sex, duration of diabetes recent HbA1C level and diastolic dysfunction.*

Tei index as calculated from the tissue Doppler imaging was 0.56± 0.05 in patients with diastolic dysfunction and 0.43±0.06 in patients with no diastolic dysfunction with significant positive correlation coefficient of 0.695 (p = 0.01). (Table 2, figure 4)

**Table-II**

*Echocardiography parameters noted in patients Tei index mean±SD*

| | Tei index mean ± SD |
|---|---|
| LVDD | 0.56 ±0.05 |
| No LVDD | 0.43±0.058 |
| Indeterminate | 0.5±0.053 |

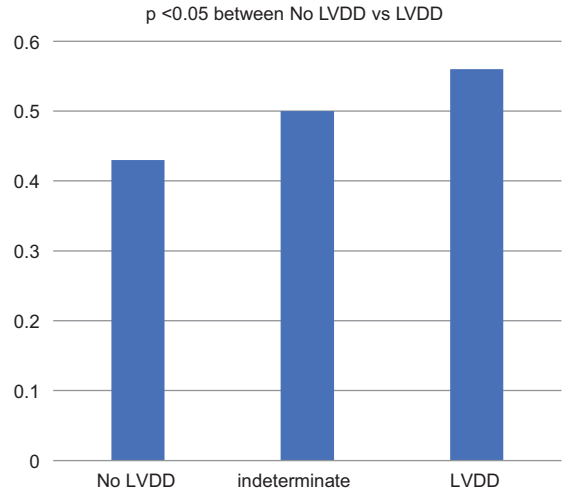



**Fig.-4:** comparison of tei index in between the groups

**Discussion:**

Prevalence of LVDD is higher diabetic patients as compared to normal individual[12]. Though inconsistently, studies have shown that older age, higher BMI, longer duration of diabetes, poor glycemic control are associated with increased risk of developing LVDD in diabetes even in the absence of hypertension and coronary artery disease[13,14]. The prevalence of LVDD in asymptomatic diabetic patients varies widely across different studies, ranging from 14% to 71% mainly because of heterogeneity of diagnostic criteria used, populations studied and associated comorbidities[15,16]. A systematic review and meta-analysis done in 2018 found the prevalence to be 46%[17].

Compared to older 2009 criteria; the updated 2016 ASE criteria detects more advanced and may be more clinically significant LVDD and is less specific to detect milder form of LVDD. Further studies are warranted to investigate the prognostic impact of these criteria.[18] Validating their accuracy is difficult. One of such attempts was done with multicentric study, where this echocardiographic assessment criteria of LVDD was compared with invasively measured LVEDP; which showed 87% accuracy to diagnose elevated filling pressure of PCWP >12 mmHg[19].

In a study conducted recently in 200 diabetic and 281 non diabetic, prevalence of LVDD was 17.5% among diabetic and 4.5% among nondiabetic respectively using 2016 ASE criteria to diagnose diastolic dysfunction.[12] These findings demonstrate similar findings of LVDD prevalence as with ourstudy which is significantly lower than other studies which has used older criteria to diagnose LVDD. More number (31%) of study patients in our study have indeterminate study in regard to LVDD assessment which is an inherent issue while using this criterion as also seen in a study by Van de *et al.* which has shown indeterminate study upto 30%[20].

As systolic and diastolic dysfunction frequently coexist, combined measurement of left ventricular chamber performance such as Tei index was thought to be more reflective. however, it does not determine the cause. The cutoff values have been different in different studies and different conditions. For example, in a study, 'cut off-points' of >0·47 identified patients with mild to moderate heart failure with a sensitivity of 86% and a specificity of 82%[21] In another study Tei index >0.63 was shown to be good predictor of both LVDP >12mmHg and LVDD[22].

One study done in India, which enrolled 100 patients with diabetes without hypertension and overt heart disease. used older criteria (E/A ratio, Valsalva, E/E') to diagnose and categorize LVDD and calculated MPI by PW doppler. Researchers found that 65% had LVDD, Mean Tei index values were significantly higher with increasing diastolic dysfunction (0.24, 0.45, 0.6 and 0.68 among normal, grade 1, grade2 and grade 3 LVDD)[23]. Lower prevalence of LVDD in our study is because of use of updated guideline to diagnose LVDD. The value of Tei index in patients without LVDD in our study is higher, probably because many of lower grade LVDD in their study population might fall in normal to indeterminate group in our study; and also, the method of measurement of Tei index is different.

**Conclusion:**

With updated 2016 guideline 23% of patients had LVDD in patients with diabetes with preserved ejection fraction and without obvious cardiac disease in our study. Increasing age was significantly associated with higher incidence whereas male sex, duration of diabetes and HbA1C level were not significantly different. Tei index was significantly higher in patients with LVDD than those without LVDD.

Limitations of the study : with updated guideline to assess LVDD, significant proportion of patients have indeterminate study and we did not further characterize those subset. furthermore, we have not graded LVDD. We have not taken normal subjects as control group. We have not ruled out subclinical LV systolic dysfunction with abnormal GLS.

**Conflicts of interest:** No conflict

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# Evaluation of Left Atrial Size in Patients with Hypertension with Left Ventricular Hypertrophy in a Tertiary Care Hospital of Nepal

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## Abstract

**Background:** The hypertensive heart disease is characterized by left ventricular hypertrophy (LVH), atrial remodeling and AF. The impact of hypertension on the left atrium is little known. LVH is a link between hypertension and left atrium enlargement (LAE). LAE raises the suspicion of increased BMI, DM, dyslipidemia in hypertension. Thus, the aim of this study is to evaluate the LA size hypertensive patients without metabolic disorder and to evaluate whether there is a relationship between LVH and LAE.

**Methods:** This is a prospective, observational study conducted in SGNHC, Kathmandu, Nepal. Our Study included 91 hypertensive patients fulfilling inclusion criteria from August 2021 to January 2022.

**Results:** 91 patients were enrolled for the study, out of which 50 (54.9%) were males and 41 (45.1%) were females. Mean age of patients was 47.77±12.1 years. 23(25%), 28(31%) and 40 (44%) patients fulfilled SV3+RaVL, SV1+RV5, RaVL criteria respectively of LVH. 10(11%),12 (13%) and 25(27.5%) satisfied the criteria of LVMI, IVSD, RWT respectively. The mean values of LA diameters, area, volume and volume Index were calculated. There were a significant associations between left ventricular internal diameters and age with LAVI (p-value <0.05). There was a mild positive association between SBP more than 140mm of Hg and LAVI, although not significant statistically. Similarly, there was a significant association between increased BMI above 25 and LAVI more than 34.

**Conclusion:** LAE is associated with LV diameter, age, BMI and SBP.

**Key words:** LA volume index (LAVI), Left Ventricular internal diameter (LVID), Left ventricular Hypertrophy ( LVH)

**J Inv Clin Cardiol 2023; 5(1):** 19-23

## Introduction:

Worldwide, 1.28 billion persons between the ages of 30 and 79 are projected to have hypertension, with the majority (two-thirds) residing in low- and middle-income nations. According to estimates, 46% of persons with hypertension are asymptomatic. It is diagnosed and treated in 42% of cases only[1].

The spectrum of hypertensive heart disease is characterized by left ventricular hypertrophy (LVH), atrial remodeling and atrial fibrillation. Although the impact of hypertension on the left ventricle has been thoroughly researched, little is known about the impact on the left atrium. Left ventricular (LV) hypertrophy and coronary heart disease (CHD) mortality have been linked, according to research[2].

Increased LV mass as detected by echocardiography is a strong independent predictor of cardiovascular morbidity in hypertension. Age, body weight, the duration of atrial fibrillation, LV mass, annular calcification, the severity of coronary artery disease, and hypertension have been related to left atrium (LA) size in previous studies[3].

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LA volume is a marker of left ventricle (LV) diastolic dysfunction severity and duration. Increased LA volume is mainly the result of impaired LV filling. In patients with hypertension, the latter is a consequence of LV hypertrophy and remodeling. It has been suggested that LV hypertrophy is a link between hypertension and left atrium enlargement. Furthermore, LA enlargement caused by hypertension is often detected earlier than LV hypertrophy or dilatation in the course of hypertensive heart diseases[4].

In addition, the presence of left atrial enlargement is associated with increased body mass indexes, smoking, diabetes mellitus, dyslipidemia in hypertensive patients[5].

Thus the aim of the present study is to evaluate the LA size in diagnosed hypertensive patients without the history of metabolic disorder and to evaluate whether there is a relationship between LV hypertrophy and LA size among patients meeting the inclusion criteria.

**Study Methodology**

This study is a hospital based, cross-sectional, prospective study conducted at Shahid Gangalal National Heart Centre (SGNHC), Kathmandu, Nepal from August 2021 to January 2022 (6 months). Informed consent was taken prior to enrolment in the study. After approval from Institutional review board (IRB) of National Academy of Medical Sciences (NAMS), Bir hospital, 91 patients who fulfilled inclusion criteria were enrolled. We performed office blood pressure measurements twice on the non-dominant arm after 10 minutes of rest using Omron M5-I device (Omron, Kyoto, Japan) and results was averaged. Systolic blood pressure (SBP), diastolic blood pressure (DBP ), pulse pressure (PP SBP –DBP ) and MAP was determined.

A 12-lead ECG and simultaneous rhythm strip was recorded at 25mm/s with a gain setting of 10mm/mV.The ECG (Sokolow- Lyon, Cornell voltage and RaVL criteria) was applied to calculate the size of LV. Those patient fulfilling ECG LVH criteria were further enrolled for echocardiography. Echocardiographic assessment was performed by using Vivid 7 ultrasound system (General Electric Healthcare, Milwaukee, WI) equipped with a harmonic 1.7 to 3.4 MHz variable frequency phased-array transducer. LV mass and LVMI was calculated by using Devereux formula. LA volume was measured using the modified Simpson's method using apical 2- and 4-chamber views at end systole of the LV. Left Atrial Volume Index was calculated as LA volume/body surface area.

The data was collected by questionnaire at OPD, ER and ward by the principal investigator. The patient's detailed history, physical examinations, and required investigation was recorded in structured proforma.

**Inclusion Criteria**

All hypertensive patients with age > 18 years were included in the study.

**Exclusion Criteria**

1. The patient with coronary artery disease (CAD)
2. The patient under treatment with congestive heart failure
3. The patient with cardiomyopathy or arrhythmia
4. Valvular Heart disease
5. Known case of diabetes mellitus

**Statistical Methods**

All data were collected and checked manually. Data were entered into an electronic spread sheet (Microsoft Excel, Redmond) and the statistical analysis was done by using the SPSS version 26 software. All categorical variables were expressed in frequency and percentage. For numerical data, normality was checked by using visual inspection of Histograms, Q-Q plots and Shapiro Wilk test. All normal numerical data were presented in a mean±SD. For association, Karl Pearson correlation coefficient was computed and interpreted. Chi-square test was performed for categorical data. Processing of all available information and statistical analysis of their significance was done. For the purpose of this study a 95% confidence interval was accepted.

**Results:**

Among 91 patients, 50 (54.9%) were males and 41 (45.1%) were females. Mean age of patients was 47.77±12.1 years. Majority of them were asymptomatic 41 (45.1%) that was followed by presenting symptoms of headache 31(34.1%), palpitation 10 (11%), Shortness of breath 5(5.5%), chest pain 3(3.3%), pedal edema 1(1.1%) respectively. Majority of patients were taking CCB+ ARB i.e. 53 (58.2%) that was followed by amlodipine 24 (26.4%). The mean systolic, diastolic BP and MAP was found to be 154.03, 95.48 and 114.83 mm of Hg respectively

(Table 1). The mean BMI of patients was found to be 25.5. 51(56%) had BMI 25 and above. 23(25%) patients fulfilled SV3+RaVL criteria of LVH. 28(31%) satisfied SV1+RV5 criteria. 40 (44%) patients fulfilled RaVL criteria. 10(11%) satisfied increased LVMI criteria.12 (13%) satisfied IVSD more than 0.9(female) and more than 1(male). RWT 0.42 and above were 25(27.5%). The mean EF of the patients was found to be 65.55%. The mean values of LA longitudinal diameter (in cm), LA transverse diameter (in cm), LA surface area (in cm2), LA volume Biplane (in ml) and LA Volume Index (ml/m2) were 4.4, 3.2, 13.8, 36.2 and 20.9 respectively (Table 2). There was no any significant association between ECG LVH criteria and LA volume index. There was significant association between left ventricular internal diameter in systole and diastole with left atrial volume index (p-value <0.05) but no association was seen between IVSD, LVMI and RWT with left atrial volume index (Table3). There was a significant association between age and LAVI with p-value 0.008 (Table 4). There was a mild positive association between SBP more than 140mm of Hg and LAVI, although not significant. Similarly, there was a significant association between BMI 25 and above and LAVI more than 34 with p-value 0.046.

**Table-I**

*Blood Pressure Measurement (n=91)*

| | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|
| Systolic Blood Pressure (in mm of Hg) | 110.00 | 197.00 | 154 | 17.6 |
| Diastolic Blood Pressure (in mm of Hg) | 70.00 | 117.00 | 95.5 | 9.7 |
| Mean Arterial Pressure (in mm of Hg) | 83.00 | 137.00 | 114.8 | 11.4 |

**Table-II**

*Descriptive statistics of left ventricular size (n=91)*

| | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|
| LA longitudinal diameter (in cm) | 3.00 | 6.00 | 4.4 | .6 |
| LA transverse diameter (in cm) | 2.100 | 4.300 | 3.2 | .46 |
| LA surface area (in cm2) | 7.80 | 21.00 | 13.7 | 3 |
| LA volume Biplane (in ml) | 16.00 | 80.00 | 36.2 | 13.5 |
| LA Volume Index (ml/m2) | 10.00 | 48.00 | 20.9 | 8 |

**Table-III**

*Correlation between Left Ventricular statistics and LA Volume index (n=91).*

| Left ventricular statistics | | LA Volume Index (ml/m2) |
|---|---|---|
| Interventricular Septal Diameter End Diastole (in cm) | Pearson Correlation | -.038 |
| | p-value | .719 |
| Left ventricular internal diameter end systole(in cm) | Pearson Correlation | .244* |
| | p-value | .020 |
| Left ventricular internal diameter end diastole ( in cm) | Pearson Correlation | .231* |
| | p-value | .028 |
| Relative Wall Thickness | Pearson Correlation | -.175 |
| | p-value | .096 |

*. Correlation is significant at the 0.05 level (2-tailed).

**Table-IV**

*Correlation between Age of the patients and LA Volume index (n=91).*

| | | LA Volume Index (ml/m2) |
|---|---|---|
| Age of the patients (in years) | Pearson Correlation | .278** |
| | p-value | .008 |

** Correlation is significant at the 0.01 level (2-tailed).

Chi-Square Test

| | | LA Volume index above 34 | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|
| | | 35.00 | 42.00 | | | |
| BMI 25 and above | 27.60 | 0 | 3 | 3 | 4.0 | 0.046 |
| | 29.00 | 1 | 0 | 1 | | |
| Total | 1 | 3 | 4 | | | |

**Discussion:**

Left atrial enlargement is risk factor for atrial fibrillation, embolism and death. Left atrial size is regarded as reflection of the average effect of left ventricular filling pressure against the LA over time and it has been proposed as marker of diastolic burden[14].

In a study conducted in Nepal concluded that the Left Atrial Size correlates significantly with the Left Ventricular Mass Index. In addition, the presence of left atrial enlargement was predominantly seen among patients with increased BMI, DM, dyslipidemia[5]. LA diameter was assessed as an indicator of LA enlargement in this study. However, associations among increasing age and BMI with increased LA volume index is similar findings in our study as well.

A study in Japan to find out the factors affecting LVMI in hypertensive patients concluded that Left ventricular volume and mass are independent factors affecting LAVI. The incidence of PAF is associated with LA size. LA size may be useful surrogate marker for monitoring the effectiveness of medical therapy and occurrence of AF. In this study, LA volume index is not associated with BP,BMI and age but left atrial volume index has positive correlation with left ventricular volume, LVMI[6].In our study, we have excluded the patients with AF, there was a mild correlation of BMI and SBP more than 140 with left atrial volume index. Similar to the findings of the study in Japan, LAVI is associated with left ventricular systolic and diastolic diameter. In contrast to this, LVMI is not associated with LAVI in our study. Most of the patients in our study were taking ACEI/ARB as antihypertensive that have tendency to reduce LAVI as per the same study findings.

According to Rojek et. al., LV mass and function are the main determinants of LAVI. However, in persons with lower LV mass, LAVI depends on the steady component of blood pressure, but not pulsatile one. Increased LAVI reflects early changes in response to systemic blood pressure elevation[4]. To the contrary, in our study, LVMI is not found significantly associated with LAVI. Also, there is a mild association between SBP more than 140 mm of Hg but not statistically significant.

According to Meel R et. al., there is no significant differences in the maximum and minimum LAVI among different age categories[8]. This study was conducted among normal population but our study is conducted among hypertensive patients so hypertension itself could be the risk factor for abnormal LAVI.

According to recent study published in European Heart Journal 2021 showed that LAV is significantly increased in patients with high BMI[9]. This study was conducted among normal population without having hypertension. In our study, there is a significant association between BMI more than 25 and LA volume index more than 34ml/m2. Such association may be either due to hypertension itself or may be due to confounding effect of increased BMI.

According to Seko Y. et. al., 2021 showed that patients with both ECG-based and echo-based LVH cases in only 4.1% of cases applying LVMI more than 95 gm/m2 in female and more than 115 gm/m2 in

male[10]. Similar to our study where only 10 (11% patients) satisfied increased LVMI criteria for male and female.

In a study published in ESC heart failure Journal in 2021 showed that in patients with heart failure with preserved Ejection fraction , only age atrial fibrillation NT-proBNP and LVMI were associated with LAVI[11]. But in our study LAVI is not associated with increased LVMI. The study designs of both studies are different as in this study patient with heart failure were enrolled but that were excluded in our study.

A recent study showed that LA volume index was superior to LA diameter index. LA volume index had independent prognostic implications in terms of coronary heart disease prediction in hypertension patients with preserved left ventricular ejection fraction[12]. Even though the study design is different from our study, LAVI was taken as the major indicator of LA enlargement in our study.

**Limitations**

Our study is single centered hospital based study with sample size of 91. Most of the enrolled patients in our study were newly diagnosed hypertension and had no any hypertensive complications. Two dimensional echocardiography modality was used in our study.

**Conclusion:**

LA enlargement in hypertension is significantly associated with age and increased LV diameter. There is a mild association with SBP More than 140 mm of Hg and BMI 25 and above though not statistically significant. There was no significant association between LVMI and LAVI.

**Conflict of Interest:** None

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# Successful Primary Percutaneous Coronary Intervention in a Young Patient in Peripheral Hospital And Its Out Come

**Tariqul Islam Khan[1], Gobinda Kanti Paul[2], Mohsin Ahmed[3], Md. Arifur Rahman Sazal[4], Aminur Razzaque[5], Shariful Islam Ratan[6]**

**Abstract:**

Among all coronary artery disease (CAD) ST-T elevated MI is the most emergency condition to treat within time. but in our country all peripheral hospital has no enough scope for the recommended treatment, that is primary percutaneous coronary intervention (PCI) due to lake of infrastructure and trained cardiologist. Most of our patients does not reach to hospital within the golden time. In most of these patients are treated with pharmacological revascularization by Streptokinase or Tenecteplase. Few patients are saved who are with no other risk factors and can reach in hospital within the time frame of myocardial infarction cascade. For this consequence most of our patients suffers from the sequalae of myocardial infarction, that may be arrhythmias, cardiomyopathy to death. We performed primary PCI in peripheral hospital in a young patient successfully and the outcome is satisfactory. We want to continue this service in our hospital for preventing mortality and morbidity of our cardiac patients.

Key words: young patient with STEMI, Primary percutaneous transluminal coronary angioplasty. Peripheral hospital.

**J Inv Clin Cardiol 2023; 5(1):** 24-28

## Introduction:

Cardiovascular diseases (CVDs) are the leading cause of death globally, taking an estimated 17.9 million lives each year. CVDs are a group of disorders of the heart and blood vessels and include coronary heart disease, cerebrovascular disease, rheumatic heart disease and other conditions. More than four out of five CVD deaths are due to heart attacks and strokes, and one third of these deaths occur prematurely in people under 70 years of age[1]. In our country most of the people are not aware about cardiac emergency and in periphery all the hospital are not designed for UpToDate treatment like percutaneous coronary intervention (PCI) not even giving pharmacological treatment. At same time specialized hospital are not with in the reaching area so that acute case of cardiac patients can reach in time. Recently we have started cardiac cathlab service in Mymensingh Medical College Hospital for the first time in Mymensingh Division. We are trying to give service for more than 2.5 core people of Mymensingh division and surrounding districts.

## Case report:

A 32 years old farmer presented to the emergency department with 4 hours duration of severe central chest pain with sweating. He was shifted to Department of Cardiology for cardiac management. After electrocardiogram it revealed ST- T elevation in chest leads V1 to V6 with RBBB, that is anteroseptal myocardial infarction (STEMI). He was in cardiogenic shock and we started Inotrope immediately. He was normotensive and moderate smoker, non-diabetic.

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After initial management we discussed with the patient party regarding different options and our limitations. Usually, we do thrombolysis in such case with Streptokinase, and also with Tenecteplase. But it (STEMI) was class I level of evidence A indication for primary PCI [2]. We explained that we can go for primary percutaneous transluminal coronary angioplasty also. Considering every expect patient party agreed for PPCI in our hospital. Loading- Aspirin- 300mg, Ticagrelor- 180mg given. We took written informed consent of patient's party. After initial triage he was taken in our cardiac cathlab for primary angioplasty. We started coronary angiogram from the right coronary system showed right dominant good-sized vessels and free from significant disease. In left coronary system, left anterior descending artery (LAD) was 100% occluded from proximal part. Left main (LM), left circumflex (LCX)artery was good sized and significant disease free. we took extra backup (EBU) 6fr PCI guide catheter and engaged in left coronary artery properly within short time. As per resent guideline recommendation we did not try to aspirate the thrombus by suction catheter [3]. Then we crossed the lesion with Run through floppy guide wire .But desired flow was not established. So, we took a compliant balloon for predilatation, size was 2.5×12. Now a DES 3/18mm (Endeavor Resolute) deployed with 16 atm in proximal LAD. Intra- coronary vasodilators was given and TMI III flow was established. As there was thrombus, we also given Eptifibatide(integril) 10mg bolus, then 6ml/ hr for next 12 hour. Patient was symptom free just within 30 minutes of procedure. Post PCI ECG shows

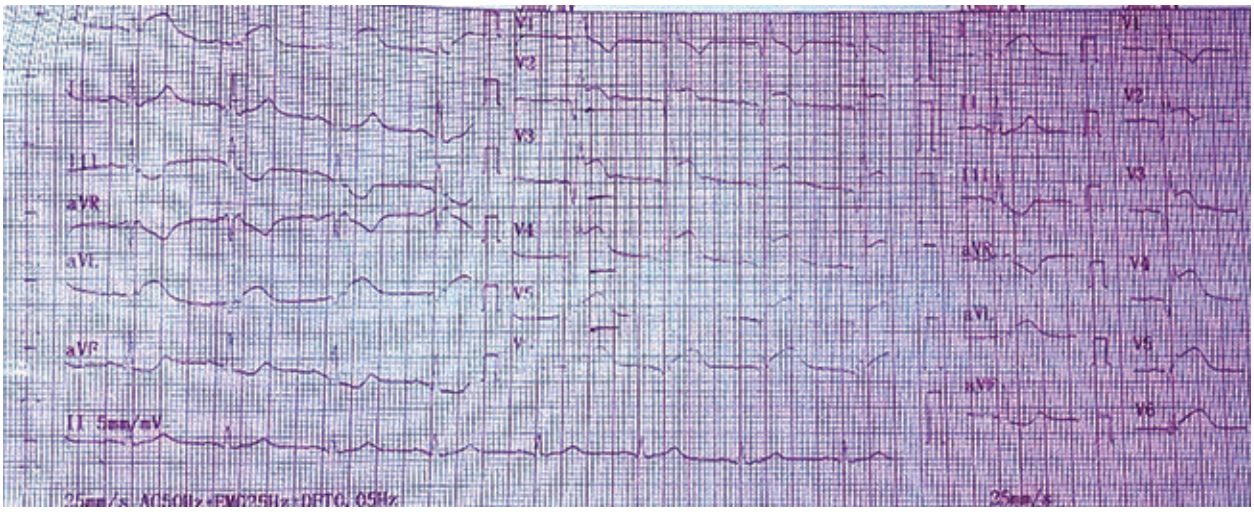



**Fig.-1:** *ECG on admission, showing ST-T elevation in V1- V6 and RBBB*

**Recommendations for Revascularization of the Infarct Artery in Patients With STEMI**
**Referenced studies that support the recommendations are summarized in Online Data Supplement 7.**

| COR | LOE | RECOMMENDATIONS |
|---|---|---|
| 1 | A | 1. In patients with STEMI and ischemic symptoms for <12 hours, PCI should be performed to improve survival (1-5). |
| 1 | B-R | 2. In patients with STEMI and cardiogenic shock or hemodynamic instability, PCI or CABG (when PCI is not feasible) is indicated to improve survival, irrespective of the time delay from MI onset (6,7). |
| 1 | B-NR | 3. In patients with STEMI who have mechanical complications (e.g., ventricular septal rupture, mitral valve insufficiency because of papillary muscle infarction or rupture, or free wall rupture), CABG is recommended at the time of surgery, with the goal of improving survival (8,9). |
| 1 | C- LD | 4. In patients with STEMI and evidence of failed reperfusion after fibrinolytic therapy, rescue PCI of the infarct artery should be performed to improve clinical outcomes (10-13). |
| 2a | B-R | 5. In patients with STEMI who are treated with fibrinolytic therapy, angiography within 3 to 24 hours with the intent to perform PCI is reasonable to improve clinical outcomes (14-20). |

**Fig.-2:** *Recommendation for Revascularization in STMEI patient 2*

the ST segment was in isoelectric line. Post PCI echocardiogram shows there are hypokinetic and dyskinetic wall motion abnormality by Global longitudinal Stain (GLS) in anterolateral area of left ventricle. Patient was discharged with duel anti platelet, statin, ACE inhibitor. After 2 months follow-up patient was quite well and echocardiogram shows less wall motion abnormality, in GLS we found most of the injured myocardium salvaged. as we are in peripheral hospital, it was a challenge for us and also for the patient. Despite all limitations we are successful as per protocol which is a great blessing for the cardiac patients of Mymensingh division.

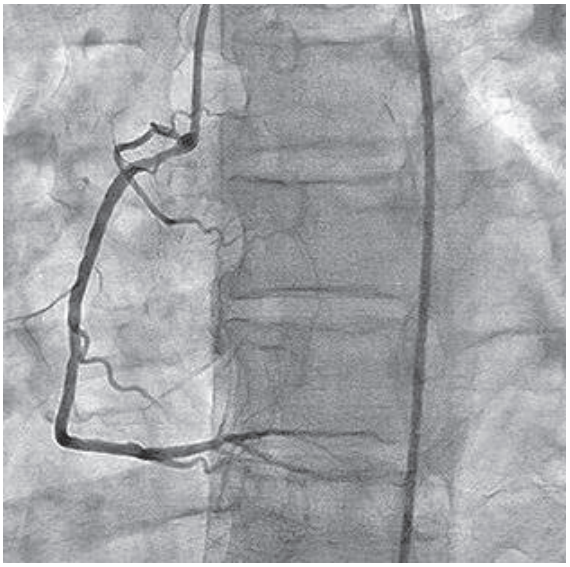


**Fig.-3:** *Normal Right coronaries*

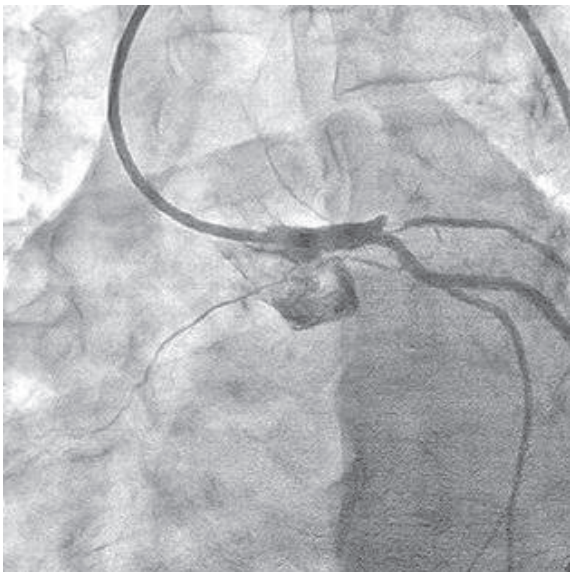


**Fig.-4:** *100% occluded LAD*

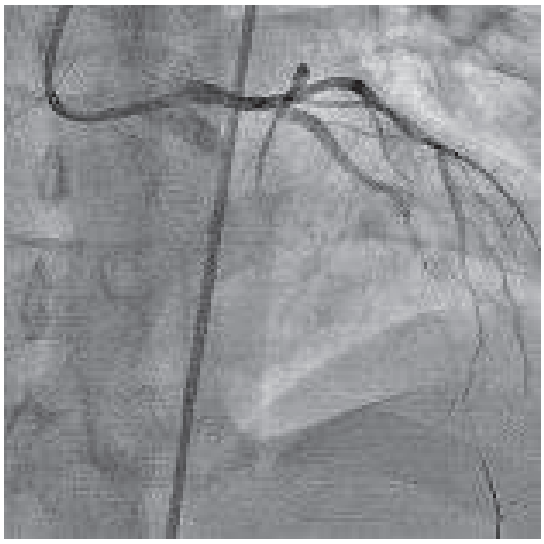


**Fig.-5:** *floppy guide wire crossing lesion*

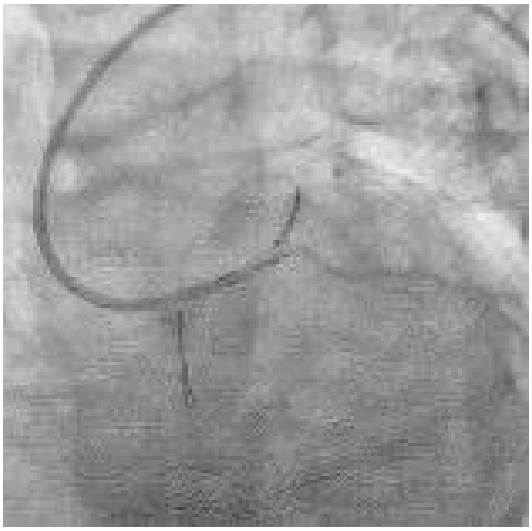


**Fig.-6:** *Predilatation with compliant balloon*

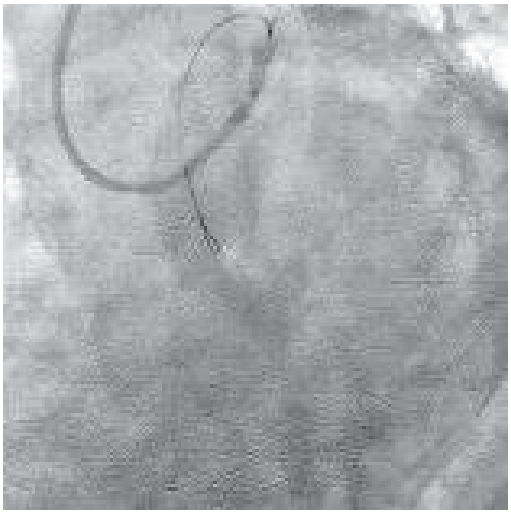


**Fig.-7:** *Deployed DES*

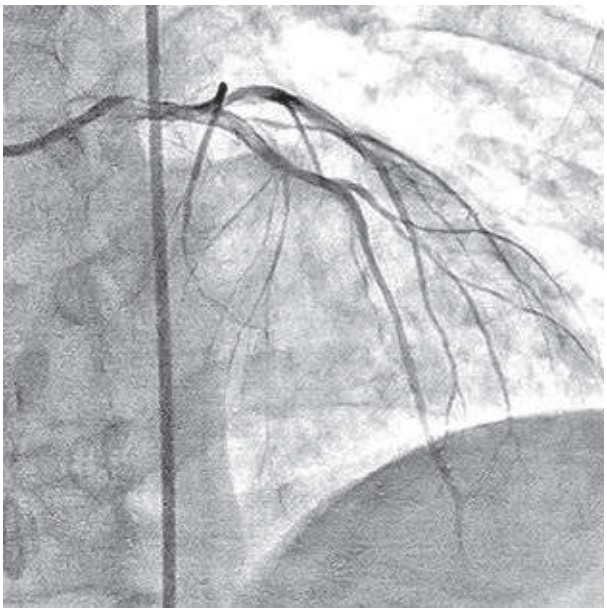
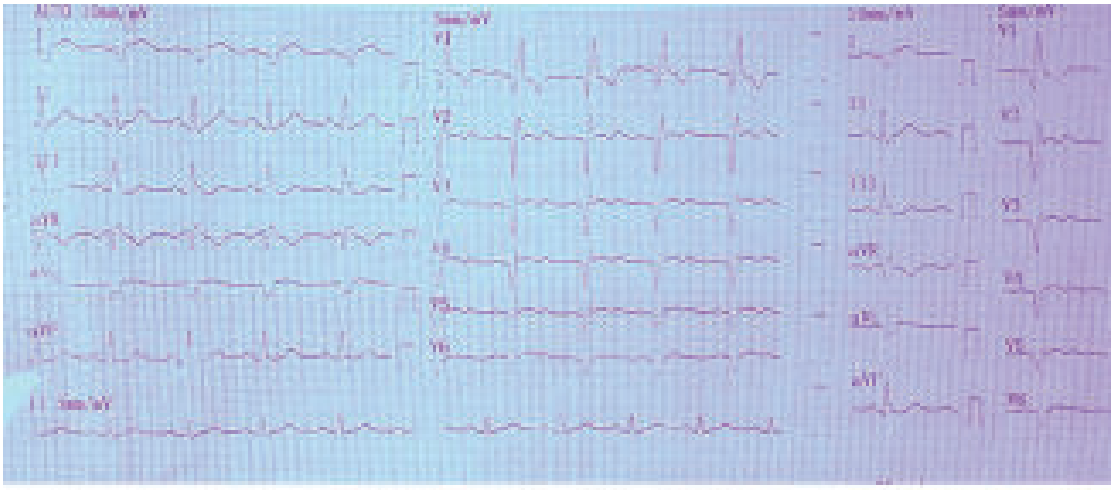
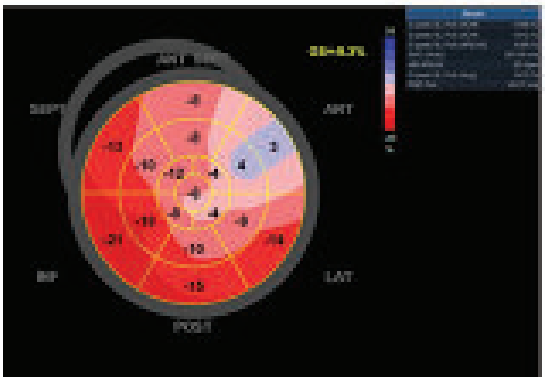


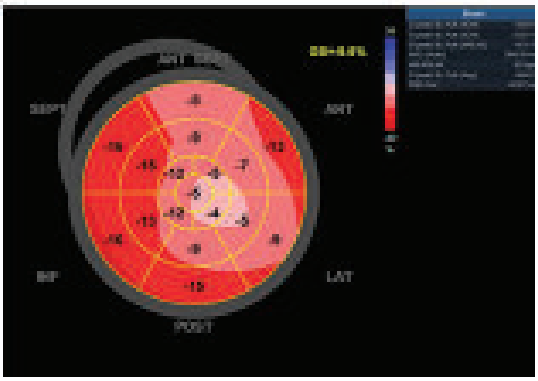
**Fig.-8:** *TIMI III flow established in LAD*



*(a)*



*(b)*



*(c)*

**Fig.-9:** *(a) Post PCI ECG , ST-T in isoelectric line, (b) Post PCI Echo, GLS showing large injured myocardium, (c) Follow-up Echo, GLS showing salvaged myocardium*

**Discussion:**

Coronary artery disease (CAD) is one of the most important causes of mortality and morbidity in worldwide population. ST-segment elevation myocardial infarction (STEMI) and patients with equivalent findings (true posterior MI, hyper-acute T-wave changes, anterior ST depression with ST elevation in lead aVR, and new left bundle branch block with Sgarbossa concordance criteria or hemodynamic instability) account for 30–50% of myocardial infarctions (MI) and are associated with substantial short- and long-term morbidity and mortality[4,5].

Gold standard treatment of STEMI patient is primary PCI according to recent guideline. But in our country it is nit possible due to many limitation like lake of awareness, distance of specialized hospital with manpower. In patients undergoing primary PCI for treatment of STEMI, complete reperfusion with development of TIMI 3 flow is achieved in over 90% of patients compared to 50–60% of patients treated with fibrinolytic therapy. Patients who achieve less than TIMI 3 flow with PCI are frequently late presenters, have large thrombus burden, and have poorer outcomes[6]. As a peripheral hospital, in Mymensingh medical college hospital we have taken al the challenge in mind and did primary PCI. Our target is to give the patients best possible treatment which is their human right. We are trying to decrease the rate of mortality and morbidity of cardiac patient of Mymensingh division.

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