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CONTENTS

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Dr. S.M. Mustafa Zaman
Publicity Secretary
Bangladesh Cardiac Society
Room # 362, 2nd Floor
(Middle Block)
National Institute of
Cardiovascular Diseases
Sher-e-Bangla Nagar
Dhaka-1207, Bangladesh
Telephones:
Office: +8801799925522
E-mail: bangladeshheartj@yahoo.com

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Prof. Dr. Amal Kumar Choudhury
Professor, Department of Cardiology
National Institute of Cardiovascular Disease (NICVD), Dhaka

Dr. Khaled Mohsin
Associate Professor, Department of Cardiology
National Heart Foundation Hospital and Research Institute

Dr. Kazi Shariful Islam
Associate Professor , Department of Cardiac Surgery
National Institute of Cardiovascular Disease (NICVD), Dhaka

Dr. Shafiqur Rahman Patwary
Associate Professor, Department of Cardiology
Sir Salimullah Medical College, Dhaka

Dr. Abul Hasan Md. Bashar
Associate Professor, Department of Vascular Surgery
National Institute of Cardiovascular Disease (NICVD), Dhaka

Dr. Gaffar Amin
Assistant Professor, Department of Cardiology
Dhaka Medical College & Hospital, Dhaka

Dr. Bijoy Datta
Assistant Professor, Department of Cardiology
National Institute of Cardiovascular Disease (NICVD), Dhaka

Dr. Asraf Uddin Chowdhury
Assistant Professor, Department of Cardiology
Sheikh SayeraKhatun Medical College , Gopalgonj

Dr. AHM Waliul Islam
Consultant
Department of Cardiology
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INSTRUCTION TO AUTHORS

A. Introduction

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

B. Categories of Articles

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

Original Research:

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

Review Articles:

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

Case Reports:

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

Cardiovascular Images:

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

Letter to the Editor:

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

C. Criteria for Acceptance

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

D. Editorial Process

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

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

E. Cover Letter

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

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

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

F. Manuscript Preparation

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

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

a. Title page information

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

b. Abstract

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

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

c. Keywords

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

d. Abbreviations

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

e. Acknowledgements

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

f. Units

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

g. Figures and graphics

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

h. Tables

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

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

i. References

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

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

Examples of correct forms of references are given below:

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

1. *Standard journal article*
List the first six authors followed by et al.

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

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

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

3. *Both personal authors and organization as author* (List all as they appear in the byline.)
Vallancien G, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1,274 European men suffering from lower urinary tract symptoms. J Urol. 2003;169(6):2257-61.

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

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

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

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

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

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

Books and Other Monographs
1. *Personal author(s)*

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

2. *Editor(s), compiler(s) as author*
Gilstrap LC 3rd, Cunningham FG, VanDorsten JP, editors.Operative obstetrics. 2nd ed. New York: McGraw-Hill; 2002.

3. *Organization(s) as author*
Advanced Life Support Group. Acute medical emergencies: the practical approach. London: BMJ Books; 2001. 454 p.

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

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

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

Other Published Material
Newspaper article

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

Unpublished Material
In press or Forthcoming

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

Electronic Material
1. *Journal article on the Internet*

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

Article published electronically ahead of the print version:

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

Article with document number in place of traditional pagination:

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

Article with a Digital Object Identifier (DOI):

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

2. *Monograph on the Internet*

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

3. *Homepage/Web site*

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

G. Submission Preparation Checklist

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

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

H. Submission

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

Prof. Hl Lutfur Rahman Khan
The Editor, Bangladesh Heart Journal
Professor of Cardiology
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Bangladesh.

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

Email: bangladeshheartj@yahoo.com

Editorial

Congenital and Structural Heart Diseases: We are in Progress

Nurun Nahar Fatema

(Bangladesh Heart Journal 2018; 33(2): 78-79)

Congenital heart disease (CHD) refers to structural or functional heart disease present at birth, even if first discovered later. The incidence of congenital heart disease is the rate that refers to the number of children born with congenital heart disease related to the total number of births over a period of one year. Incidence varies from 6-8/1000 live births in various studies conducted in many centers. In Bangladesh incidence was found up to 25/1000 live births in one year in a study conducted in Combined Military Hospital (CMH), Dhaka. Infant mortality rate of 54/1000 live births in the country is also contributed a lot by the death of infants from congenital heart disease.

There are 8 common lesions which account for about 80% of all congenital lesions. They are in descending order of prevalence are ventricular septal defect (VSD, 30%), patent ductus arteriosus(PDA, 10%), atrial septal defect (ASD, 8%), pulmonary stenosis (PS, 8%), coarctation of aorta (CoA, 5-6%), aortic stenosis (AS, 5-6%), transposition of great arteries (TGA, 4-5%). The remaining 20% or so is made of a variety of rarer or complex lesions.

The important pediatric cardiac milestones are successful PDA ligation in 1938 in USA, coarctation repair in 1945 in USA, ASD closure in 1953 by Gibbon in USA. The life-saving intervention of balloon atrial septostomy was first performed in late 1960 by Rashkind in Philadelphia, USA. In late 1970, introduction of prostaglandin for treatment of ductus dependent pulmonary and systemic circulation provides a measure of securing adequate oxygenation in a number of blue neonates, thus delaying emergency intervention and surgery by keeping them alive. On the other hand, indomethacin is used to close the haemodynamically significant PDA in new born. First percutaneous PDA occlusion was performed in 1967 by Portsmann et al.and first ASD device closure was done by King and Mill in 1976. First balloon valvuloplasty was introduced in 1979. First VSD device closure was performed in 1990s. Amplatzer septal occluder was first described in 1997. Melody valve for percutaneous pulmonary valve implantation was innovated in 2000.

As incidence of CHD is increasing, the magnitude of the problem is becoming enormous in our country. There was not a single pediatric cardiology-trained person in the country until a team started work in CMH, Dhaka after training from Prince Sultan Cardiac Centre (PSCC), Riyadh, KSA in August 1998. That team was composed of paediatric cardiologist, anesthesiologist, surgeon along with echo, cath lab, operation theatre (OT), and intensive care unit (ICU) technicians. PSCC donated cath lab disposables and interventional hardwires to pediatric cardiologist in sufficient amount so that work could be started immediately. So, all possible interventions were started after commencement of thatunit. Following are the list of important milestones of congenital cardiac interventions in Bangladesh.

Pulmonary valve replacement first ever in South Asia with Melody valve 2012, balloon atrial septostomy (reported) in 1999, PDA coil occlusion in1998, pulmonary valvoplasty in infant in 1999, ASD device closure in 2001, muscular VSD device closure in 2005, perimembranous VSD device closure in 2006,PDA device closure in 2003, coronary cameral fistula closure in 2010, PDA stenting in 2006 in pulmonary atresia, VSD coil occlusion in 2007, huge tubular PDA closure in 2007,CoA balloon angioplasty in newborn in 2001,aortic balloon valvuloplasty for critical aortic stenosisin 2001, right ventricular outflow tract (RVOT) stenting in double outlet right ventricle (DORV),VSD,PS in 2007, patent foramen ovale(PFO) closure in 2010.All these interventions were performed in catheterization laboratory of CMH, Dhaka.

Address of Correspondence : Brig. Gen. Nurun Nahar Fatema, Head of Paediatric Cardiology and Paediatrics , Combined Military Hospital Dhaka, Bangladesh. Mobile: +8801819239021, E.mail: colfatema@hotmail.com

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Many interventions were life-saving for newborn and young infant and those were performed routinely with excellent outcomes. Later, other cardiac hospitals started pediatric cardiac programs and delivering services to the large number of children suffering from CHDs.

There was glorious history of congenital cardiac surgeries since late 70's. In Bangladesh, first ever Blalock Taussig (BT) shunt was performed in early 80's in National Institute of Cardiovascular Diseases(NICVD) by Prof. S R Khan. (Information of first ever surgeries are collected from a presentation of Prof. Asit Baran Adhikary in Dhaka Shishu Hospital). Ligation of PDA was performed in 1979(Prof.S R Khan and team), ASD closure in 1981 (Prof. N A Khan and team), VSD closure in 1991(Prof S R Khan and team), successful tetralogy of Fallot (TOF) repair in 1992(S R Khan and team), bidirectional Glenn shunt in 1995 (Prof. SR Khan and team), Mustard operation in 1996 (Prof. SR Khan and team), modified Fontan operation in 1997 (Prof. SR Khan and team), Senning operation in 2001(Asit Baran Adhikary and team), rerouting of total anomalous pulmonary venous connections (TAPVC) in 2002(Asit Baran Adhikary and team), Lecompte procedure in 2006 (Asit Baran Adhikary and team), arterial switch operation in 2007 (Jahangir Kabir and team). Continuous growth in this sector was interrupted for many reasons. Later, congenital heart surgeries started in National Heart Foundation Hospital & Research Institute (NHF&RI) and the center is now playing a major rolein treating congenital cardiac cases. Ibrahim Cardiac Hospital & Research Institute (ICH&RI) is giving support for charity cardiac surgery program for poor people in association with Al Muntada Aid, UK. Many other cardiac centers are also progressing as per capabilities.

Other than interventions and surgeries, many patientsalso require medical interventions among which maintenance of PDA in duct dependent lesion (by IV prostaglandin), closure of PDA (by IVindomethacin, ibuprofen, paracetamol) and treatment of persistent pulmonary hypertension in newborn, various arrhythmias, heart failure, cardiomyopathies are important.

Though many centers are now working with CHDs, still large number of patients are going to neighboring countries specially for neonatal, high risk and complex surgeries. Moreover, most of the children are poor and cannot afford treatment even inside country. Many charity programs are going on to meet the need of these group of patients among which Child Heart Trust(CHTB) is playing a major role by raising fund and involving foreign charity groups like Little Heart, KSA, Al Muntada, UK and Qatar Red Crescent Charity. Some charity groups are helping poor patients as part of technology transfer program to some cardiac hospitals.

Paediatric cardiology and cardiac surgery are the most difficult subspecialty, need long uninterrupted training from good centers, hard work, devotion, honesty and sincerity. Most importantly, these two groups of specialists need support from skilled anesthesiologists and intensivists. There is scarcity of dedicated pediatric cardiac anesthesiologists and intensivists in Bangladesh and these two groups must be strong to bring successful outcomes of any surgery or intervention.

Hopefully, in coming years we will be able to fulfill the need of our patients completely by development of skill from good quality training and initiation of formal course dedicated to pediatric cardiology, pediatric cardiac surgery, pediatric cardiac anesthesia and pediatric cardiac intensive care.

Original Article

Relationship between Hemoglobin A1c Level and Severity of Coronary Artery Disease Among The Hospitalized Patients with Acute Coronary Syndrome.

Syed Dawood Md. Taimur[1],Sahela Nasrin[2],M Maksumul Haq[3], M A Rashid[4],Hemanta I Gomes[5], Farzana Islam[6]

Abstract:

Background : Diabetes mellitus is one of the important risk factors for coronary artery disease. The hemoglobin A1c is used for evaluating glycemic control in diabetic patients. Here, we conducted the study to evaluate the relationship between HbA1c level and severity of coronary artery disease among the hospitalized patients with ACS.

Materials & Methods : This cross sectional study was conducted in the department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka, Bangladesh from September 2015 to December 2015. Total of one hundred patients were studied and they were grouped on the basis of their glycaemic status. One hundred patients with acute coronary syndrome were enrolled in this study. Out of them fifty were diabetic (HbA1c>6.5%) and rest of were nondiabetics (HbA1c<6.5%) (group-A and B).

Results: Out of one hundred patients fifty eight were male and fourty two were female. Mean age of patients in group-A was 58.54±10.22 years and mean age of patients in group-B was 54.52±13.69 years. Mean age of male and female was 57.72±11.48 years and 54.0±13.08 years respectively. Mean HbA1c of patients in group-A was 11.43±1.43% and group-B was 6.34±0.915%. 38% of group-A and 22% of group-B had triple vessel disease, 26% of group-A and 20% of group-B had double vessel disease and 28% of group-A and 18% of group-B had single vessel disease, and 8% of group-A and 40% of group-B had normal coronary arteries. 48% patients of age group 46-50 in group-A had more incidence in coronary artery disease than other age group which was statistically significant (p=0.035). 61-75 years age group in group-B patients had coronary artery disease than other age groups which was statistically not significant(p=0.084). Patients of group-A was significantly relation with coronary artery disease (p>.001) and six times greater coronary artery disease than patients of group-B (OR= 6.15, 95% CI for OR =2.074 -18.289).

Conclusions: In this way the importance of appropriate glycaemic control has been emphasized in diabetic patients. This study showed the relation between HbA1c levels and the severity of CAD in patient with type-II diabetes mellitus .Our findings demonstrate that elevated HbA1c level was risk factor for severity of coronary artery disease in ACS patients.

Keywords: HbA1c; Coronary artery disease; Acute coronary syndrome.

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1. Assistant Professor & Associate Consultant, Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka.
2. Assistant Professor & Consultant, Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka.
3. HOD & Senior Consultant, Ibrahim Cardiac Hospital & Research Institute, Dhaka
4. CEO & Senior Consultant, Ibrahim Cardiac Hospital & Research Institute, Dhaka
5. Assistant Professor & Associate Consultant, Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka.
6. Medical Officer & Resident, Department of Pediatric Hemato-Oncology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka.

Address of Correspondence: Dr. Syed Dawood Md. Taimur, Assistant Professor & Associate Consultant, Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, 122, Kazi Nazrul Islam Avenue, Dhaka-1000, Bangladesh. Mobile: +8801712801515, Email: sdmtaimur@gmail.com

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Introduction:

Chronic hyperglycemia in type 2 diabetes increases the risk of macrovascular events. Though there is continuing uncertainty about its effect on macrovascular outcomes and death[1], several studies have clearly demonstrated a correlation between type 2 diabetes and acute coronary syndromes (ACS). High prevalence of diabetes and undiagnosed diabetes or pre-diabetic states is seen in patients with stable or unstable coronary artery disease (CAD)[2].

This link can be attributed to hyperglycemia, insulin resistance, and a clustering of the risk factors for atherosclerosis. Potential mechanisms that could explain the relationship between diabetes mellitus and ACS including decreased insulin sensitivity leading to impaired , increased levels of catecholamines leading to increased myocardial damage and infarct size, hyperglycemia-induced osmotic diuresis and volume depletion, enhanced platelet activation, and inflammatory-immune reactions with increased markers of inflammation. Fatty acids-mediated inhibition of glucose oxidation leads to myocardial cell death, injury of cardiomyocyte plasma membrane, calcium overload, and arrhythmias[3]. Several studies have shown prognostic role of hyperglycemia and diabetes in patients with ACS. Hyperglycemia at admission for ACS is associated with less favorable outcome[4-7].

Though acute hyperglycemia may be due to the preexisting diabetes mellitus, it may also occur as a part of stress response to the disease state. Hemoglobin A1c (HbA1c) is less influenced by acute stress. Therefore, HbA1c levels may provide insight into the relation between chronic glucose control and patient outcomes. Thus HbA1c level being a stable indicator of unstressed long-term glycemic control may be a more useful predictor in ACS. HbA1c level is an indicator of average blood glucose concentrations over the preceding 2-3 months, which is a convenient and well known biomarker in clinical practice. It is now recommended as the preferred method for diagnosis and monitoring glycemic control in diabetes mellitus. Studies evaluating the association of HbA1c with ACS have reported discrepant results. Several studies showed higher crude mortality rate in patients with elevated HbA1c following adjustment for many cardiovascular risk factors[8-10].

Patients with elevated HbA1c but without known diabetes likely have diabetes that was neither diagnosed nor treated and other relevant cardiovascular risk factors such as hypertension and dyslipidemia that were also untreated before hospitalization, whereas those with diabetes are more likely to be treated with insulin and control the established risk factors[12]. Thus, the prognostic value of HbA1c level in patients with coronary atherosclerotic disease has not been well characterized and remains controversial. This study was an attempt to know the association between HbA1c and severity of coronary disease in ACS patients.

Materials and Methods:

This cross sectional study was done in the department of cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka, Bangladesh from September,2015 to December, 2015. A total of 100 cases admitted in CCU with complaints of typical chest pain and diagnosed as ACS and divided into diabetic(HbA1c>6.5%) and nondiabetic group(HbA1c<6.5%) (group-A and group-B). Patients belonging to age group of 28-85 years, with diagnosis of diabetes mellitus as per American Diabetes Association (ADA) criteria on treatment were selected. Patients with multi-organ failure, congenital or rheumatic heart disease or recent surgery were excluded from the study. Acute coronary syndrome encompasses a) ST-Segment Elevation Myocardial Infarction (STEMI), a condition for which immediate reperfusion therapy should be considered, b) Non-ST-Segment Elevation Myocardial Infarction (NSTEMI) and c) Unstable angina[19].

After screening, details of patients with regard to symptoms, duration of diabetes mellitus, medical history, and history of smoking were collected. All patients underwent thorough physical examination and the biochemical investigation. They included HbA1c, serum troponin-I, creatinine kinase-MB (CK-MB), electrocardiogram (ECG), and echocardiography. Symptoms of ACS included chest pain, shortness of breath, nausea, vomiting, palpitations, sweating, and anxiety. ACS was established on at least two of the following characteristic symptoms, electrocardiographic changes, typical rise and fall in biochemical parameter like troponin-I was measured[11]. Statistical analysis: Mean ± standard deviation was reported for continuous variables, and percentages (number) were reported for categorical variables. Continuous variables were compared using unpaired Student's *t*-test, and categorical variables were compared using Chi-square tests. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated for each independent variable. All comparisons were two-tailed and $P < 0.05$ was considered statistically significant. The entire analysis was performed with Statistical Package for the Social Sciences (SPSS) version 19.0.

Results:

Out of one hundred patients 58 were male (in group-A had 26 and group-B had 32) and 42 were female(24 were in group-A and 18 were in group-B).Mean age of patients in group-A was 58.54±10.22 years and group-B was 54.52±13.69 years . Mean male age was 57.72±11.48 years and mean female age was 54.0±13.08 years (Table-1). Group-A patients mean HbA1C was 11.43±1.43% and group-B patients mean HbA1C was 6.34±0.915%.

Table-I

Age distribution between the groups

Age group (years)	Group A (n=50)		Group B (n=50)	
	n	%	n	%
28-45	5	10	16	32
46-60	26	52	17	34
61-75	16	32	15	30
76-85	3	6	2	4
Mean ± SD (Years)	58.54±10.22		54.52±13.69	

Eighty percent patients of group-A and eighty six percent patients of group-B had hypertension which was statistically not significant (p=0.616). 86% patients of group-A and 52% patients of group-B had dyslipidaemia which was statistically significant (p=.001).38% patients of group-A and 64% patients of group-B were smoker which was not statistically significant(p=0.148). Positive family history for IHD was statistically significant(p= 0.001)in both group (Table-2).

Table-II

Distribution of risk factors between the groups (n=100)

Risk factor	Group A (n=50)		Group B (n=50)		p- value*
	n	%	N	%	
Smoking Habit					
Smoker or Ex-smoker	19	38	17	34	.148^{NS}
Non-smoker	31	62	33	66	
Hypertension					
Yes	40	80	43	86	.616^{NS}
No	10	20	7	14	
Dyslipidaemia					
Yes	43	86	26	52	.001^{S}
No	7	14	24	48	
Family H/O IHD					
Yes	29	58	23	46	.001^{S}
No	21	42	50	78.1	

NS = Not Significant, S = Significant

Out of one hundred patients, BMI of 53 patients were within 25-29.9. Ten patients BMI range was 30-34.9 and thirty seven patients BMI range was 18.4-24.9.(Fig-1)

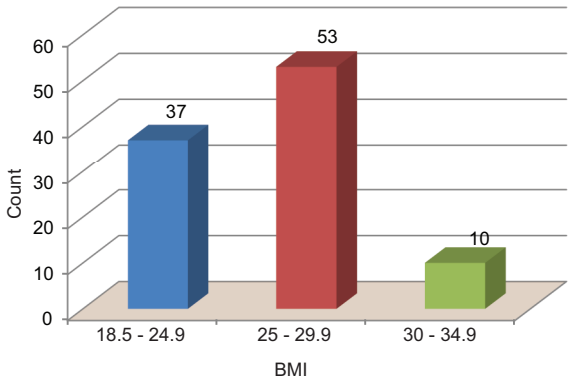

Fig-1: *Different BMI range in all age group of patients.*

Coronary angiogram was done which revealed thirty percent patients had triple vessel disease, twenty three patients had double vessel disease and twenty one patients had single vessel disease, and rest (24 patients) of had normal coronary arteries. 38% of group-A and 22% of group-B had triple vessel disease,26% of group-A and 20% of group-B had double vessel disease and 28% of group-A and 18% of group-B had single vessel disease, and 8% of group-A and 40% of group-B had normal coronary arteries.(Table:-3).

Table-III

Distribution of coronary artery disease between two groups

CAD	Group-A	Group-B	p-value
SVD	14 (28%)	9(18%)	
DVD	13(26%)	10(20%)	0.776 ^{NS}
TVD	19(38%)	11(22%)	
Normal	4(8%)	20(40%)	

NS=Not significant

Within group-A, 46-60 years age group had more incidence(48%) in coronary artery disease than other age group which was statistically significant (p=0.035). In group-B, 61-75 years age group had more frequency(26%) of coronary artery disease than other age groups which was statistically not significant(p=0.084). (Table-4)

Table-IV

Frequency of coronary artery disease in different age group

Age Group	Group-A		p-value	Group-B		p-value
Years	Frequency	Percentage (%)		Frequency	Percentage (%)	
28-45	3	6		7	14	
46-60	24	48	0.035 ^{S}	9	18	0.084 ^{NS}
61-75	16	32		13	26	
76-85	3	6		1	2	

NS = Not Significant, S = Significant

Out of 100 patients, 46 patients of group A and 29 patients of group B had coronary artery disease which is statistically significant (p<0.05). Adjusted OR for association of elevated HbA1c with acute coronary syndrome when analyzed by regression analysis adjusting for confounders like dyslipidemia, family history of ischemic heart disease. HbA1c showed strong relation with adjusted odds ratio =6.159(CI: 2.074-18.289). (Table-V)

Table-V

Correlation between HbA1C and coronary artery disease

	Coronary artery disease		Total Pt	OR	95% CI	P value
	Yes	No				
Group A	46	4	50			
Group B	29	21	50	6.159	2.074-18.289	0.00[s]

S = Significant

Discussion:

Many studies have clearly demonstrated the strong association of HbA1c with macrovascular complications in type II diabetes mellitus. There are few studies in the western literature reporting the association of HbA1c with macrovascular complications like ACS. The present study was done to know the relationship of HbA1c level with ACS in our population. The cohort study done by Selvin and colleagues [14] on 7435 patients with type 2 diabetes mellitus has shown that 1% increase HbA1c was associated with 18% increase in the risk of coronary heart disease.

In our study, difference between mean HbA1c among group-A and group-B was statistically significant. Subgroup analysis based on status of dyslipidaemia and positive family history of IHD also showed similar results. The prospective population study done by Khaw KT.et al., [15] on 10232 subjects has shown that after adjustment for systolic blood pressure, cholesterol level, body mass index, waist-hip ratio, smoking, and previous myocardial infarction or stroke, there was a 21% increase in cardiovascular events for every 1% increase in HbA1c level above 5%.

According to age patients were categorized into different age group , group-A & B, within 46-60 years age group of group-A(*P* = 0.035) had higher frequency of coronary artery disease ,on the other hand 61-75 age group of group-B(p=0.084) had also higher frequency of coronary artery disease which was statistically not significant on the other hand higher incidence of coronary artery disease found in Razu HN et al.[11] and Mehmet FO et al.[13]. There are several biologically possible mechanisms that might account for the finding that chronic hyperglycemia is associated with ACS. Hyperglycemic periods play a major role in the activation of oxidative stress and overproduction of mitochondrial superoxide, which trigger various metabolic pathways of glucose-mediated vascular damage[16,17]. Glucose can react with various proteins to form advanced glycation end products, which may contribute to long term complications in diabetes, plaque formation, and atherosclerosis[18]. These effects are gradual and likely to be cumulative, occurring during decades of exposure to chronically elevated blood glucose levels. Elevated HbA1c level is likely the result of long-term insulin resistance.

Metabolic disturbances associated with insulin resistance including hyperglycemia, dyslipidemia, hypercoagulability, and inflammation might play a major role in the adverse impact of elevated HbA1c on cardiovascular system. Limitations of the present study was small group of patients so that we did not collect the long term follow up details and mortality associated with ACS in relation to HbA1c level. The number of patients in each group was also small to calculate cardiovascular risk with increase in percentage of HbA1c.

Conclusion:

HbA1c level was strongly related with risk of coronary artery disease specially those patients who were diagnosed as ACS. Occurrence of ACS was significantly more in patients with diabetic with poorly controlled blood sugar level when compared with nondiabetic patients. Our finding supports the notion that diabetic patients with higher HbA1c level should be closely followed due to their higher risks of cardiovascular outcomes. glycemic control may help to reduce cardiovascular events in type 2 diabetic patients.

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Original Article

A Snapshot on Myxoma Operation of 62 Patients at National Institute of Cardiovascular Diseases(NICVD), Dhaka, Bangladesh

KS Islam

Abstract:

Background: Intra-cardiac mass, particularly myxoma operation is common at NICVD .Its frequency is about 1-2% among all operations done here. The main aim of this study was to analyze the different aspects of this tumour and its surgery on 62 patients operated over last three years (2015-2017).

Methods: It is a retrospective study .The data were collected over a period of 03 years (2015-2017) For this I studied the ward admission register, OT and ICU registers ,ICU flow charts, talked with the respective unit doctors to collect my data. Then the data were analyzed manually and by computer.

Results: Age range of the patients were from 7.5 years to 65years with a mean±SD (36.94±13.99). Male and female patient ratio were M:F=1:1.81.Myxoma were more common in the 4th and 5th decade of life in this study population .Preoperative time delay for operation after hospital admission was 9±2.12days.All the operations were done as an elective procedure rather urgent or emergency procedure. Post operative mortality was around 12.90% among these patients. The causes of high mortality following myxoma operation were Low Output Syndrome, Congestive Heart Failure , Cerebral stroke and septicaemia.

Conclusion: Myxoma operation is common in NICVD. Most of our patients were dealt as a routine procedure. Their features and surgical procedure were similar with a little difference among the neighbouring countries. Our post operative outcome was a little bit worse (12.90% mortality) over the mentioned period. We need to find out the causes and to take care of these patients to reduce mortality in future.

Keywords: Intra-cardiac mass , Myxoma, NICVD, Bangladesh

(Bangladesh Heart Journal 2018; 33(2): 85-89)

Introduction:

Cardiac tumors are either primary (Â0.1%.) or secondary(1%). Those arising in the heart are primary They may be benign or malignant. Myxomas are the most common benign primary cardiac tumors. It is found in all age groups, in both sexes and most often occur in women in the4th to 5th decade of life. Generally they are sporadic. Myxomas are found as an autosomal dominant syndrome in around 7% cases in association with Carney complex which comprises, myxomas, spotty pigmentation of the skin and endocrine hyperactivity. Here both sexes are affected equally and at any age, arise as single or multiple lesions in all chambers of the heart.They tend to recur after surgical excision in this complex.[1]

Cardiac tumors present with variable clinical features like obstructive, embolic or systemic features. Intramyocardial tumors can trigger cardiac arrhythmia and may cause sudden death[1].

Generally they are polypoid, pedunculated having a smooth surface and sometimes covered with athrombus. Size varies from 1 to 15 cm but average about 5 cm in diameter.

Department of cardiac surgery, National Institute of Cardiovascular Diseases(NICVD), Dhaka, Bangladesh

Address of Correspondence: Dr. Kazi Shariful Islam MS (CV&TS), Associate Professor, Cardiothoracic Surgery, NICVD, Dhaka, Mobile: 01943221899, E-mail:kazishariful_islam@yahoo.com

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Weight of approximately 70 gm. Histologically , polygonal or spindle shaped cells are found in a matrix of acid mucopolysaccharide .The cells may form capillary-like channels. These communicate with arteries and veins located at the base of the myxoma.[1]

They are commonly found in the atria. Approximately15% to 20% arise in the right atrium and 75% arise in the left atrium, and Most left atrial myxomas are located onthe border of the fossa ovalis, but they can originate from any where on the atrial wall. Rest of myxomas are located in the ventricles.[1]

Because most myxomas arise in the left atrium, systemic embolization is common, occurring in 30% to 50% of cases[1]

National Institute of Cardiovascular Diseases (NICVD) is the oldest and tertiary care cardiovascular centre in this country.Peoples from all over the country and all social strata particularly poor and middle class of people come here for the treatment of cardiovascular diseases. Over last three years (2015-2017) around 1000 cardiac surgery cases of different varieties were done. Among allthe operations done over this period, myxomas were around 2%.[2]

Only few studies were done on this issue in our country. This study will give us some insight about myxoma operation at our centre. It will also help us a little bit to understand the reasons for increased mortality over last three years. Thus it will aware us further to reduce the mortality of our myxoma patients as well as stimulate others to carry out further research in this field.

Materials & methods:

This is a retrospective study carried out in the department of cardiac surgery at NICVD,Dhaka Bangladesh during the period of 2015 to 2017.All the patients admitted either through OPD or directly referred to all the cardiac surgery units from cardiology units of NICVD for operation over that period were studied on the different aspects.2D ,Echo wasthe tool to diagnose Myxoma preoperatively. Some units also did second Echo just before operation to reconfirm the presence of Myxoma. Those patients refused operation after admission were excluded from the study. All the operating units followed standard protocol of CPB establishment. Regarding removal of myxoma from the cardiac chambers, they followed almost similar protocol. After

excisions of most of the myxomas, monopolar low power(10-15w) electrocautery was usually used here at the base of stalk. All informations or data were collected from admission file, the male and female ward registers, OT and ICU registers and OT notes. I also personally talked with the doctors of the concerned surgical units, perfusionist of NICVD Dhaka to collect further informations. All the data were collected in a sheet of Microsoft Excel of a computer and then analysed manually by the calculator and Microsoft Excel of the computer to find out the results of my study. Numerical values were expressed as range, mean±SD and percentage Results: Age range of the patients were 7.5 years to 65 years with a mean±SD (36.94±13.99) years. Myxomas were more common in 4th to 5th decade(>50% patients were in this range) of life. Myxomas were more common in female patients with M:F=1:1.81 .

Table-I

Age range with relative occurrence of myxomas in different decades.

Age range(7.5-65 Years) Mean±Stdev(36.94±13.99) years	Number of patients	Percentage(%)
0-10	04	6.45
11-20	03	4.83
21-30	11	17.74
31-40	16	25.80
41-50	16	25.80
51-60	10	16.12
61-70	02	3.22
Total=	62	

Myxomas are most commonly located in left atrium then right atrium .There was single occurrence of -biatrial and right ventricular myxoma in my study population.

Table-II

Origin site of operated myxoma in the cardiac chamber Morphology of the myxomas:

Name of the cardiac chambers	Total no	Percentage (%)
Left atrium	54	87.09
Right atrium	06	9.67
Biatrial	01	1.61
Right ventricle	01	1.61
Left ventricle	00	00
Multicentric	00	00

Most of the LA and RA myxomas were ovoid to slightly irregular shape towards their apices. Majority of them were pedunculated 59 (95%) with attachment to the interatrial septum only few were sessile 3 (5%).

Majority of the stalks were attached to the IAS close to the limbus fossa ovalis

Their colours were grey to dark brown with some reddish spots almost in all cases.

Histopathology of the intracardiac masses were myxoma in almost all cases.

Most LA myxoma 91% (appx) were removed by standard LA tomy approach through interatrial groove. Around 5.55% of LA myxomas were removed by right atriotomy with trans-septal approach and around 4%) (appx) LA myxoma were removed by biatrial/bicameral approach.RA ,RV and biatrial myxomas were approached through RA tomy approach

LA-Left Atrium, RA-Right Atrium, TV-Tricuspid valve, ASD-Atrial Septal Defect

About 84% myxomas were removed en-mass and 16% in piecemeal. After excision of the tumour the base of the stalk was cauterized in 90% cases. The other procedures were suturing of the surrounding endocardium in around 5% cases .direct suturing and pp closure in only few cases.

Patient waited for 9±2.12 days before operation after admission in the hospitals. After operation ICU stay was 6.5±3.53 days .Total hospital stay was 20.61±10.60 days.

The mortality was 12.90%. Low output syndrome was most common cause of mortality followed by CVA and CHF and infective endocarditis

Table-III

Approaches during surgical removal of atrial myxoma

Name of the approaches	Number of patients	Percentage(%)
For LA myxoma:		
LA tomy	49	90.74
RAtomy with trans-septal approach	3	5.55
Biatrial-/bicameral approach	2	3.70
For RA Myxoma- RA Tomy approach	6	100.00
For RV Myxoma-RA tomy with trans TV approach	1	100.00
For Biatrialmyxoma-RA tomy with creation of an ASD and removal of myxoma	1	100.00

Table-IV

Procedure of removal of intra-cardiac masses

Method of myxomaremoval from LA	No ofcases	Percentage(%)
En-mass	52	83.87
In picemeal	10	16.12
Dealing with the stalk of the tumour‘		
Excision and EC of the base of the stalk	56	90.32
Excision with suturing of endocardium of IAS	03	4.83
Excision with rim of IAS with suture closure of ASD(Iatrogenic)	02	3.22
Excision with rim of IAS + ppclosureof ASD	1	1.61

EC=Electro-cautaryofthebase , IAS=Inter atrial septum PP=pericardial patch closure,ASD=Atrial septal defect.

Table -V

Preoperative,ICU and hospital stay of operated patients

Stays in the hospital	(Min—Max) Days	Mean±SD (Days)
Preoperative stay	5-12	9±2.12
ICU Stay	1-12	6.5±3.53
Hospital Stay	1-81	20.61±10.60

ICU=Intensive care unit

Table-VI

Outcome variables

Variables	No of patients	Percentage(%)
LOS	3	4.83
Post operative CVA	2	3.17
Others (CHF)	2	3.17
Postop high fever(?septicemia)	1	1.61
Mortality	8	12.90%

LOS=Low output syndrome, CVS=Cerebrovascular accident ,CHF=Congestive Heart Failure,IE=Infective endocarditis,

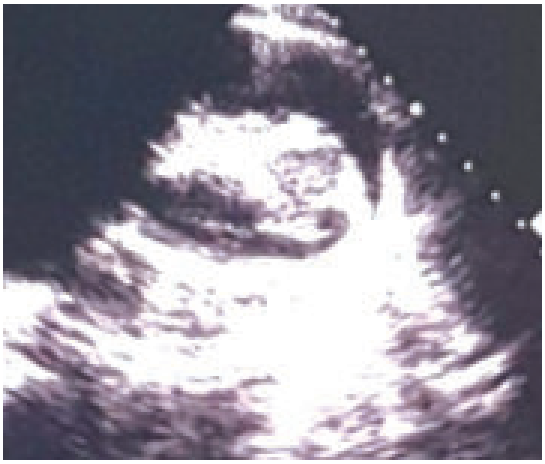

Fig.-1: *RV Myxoma in 2D echo*

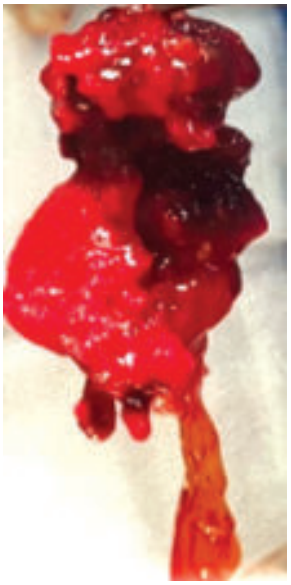

Fig.-2: *Part of excised RV mass*

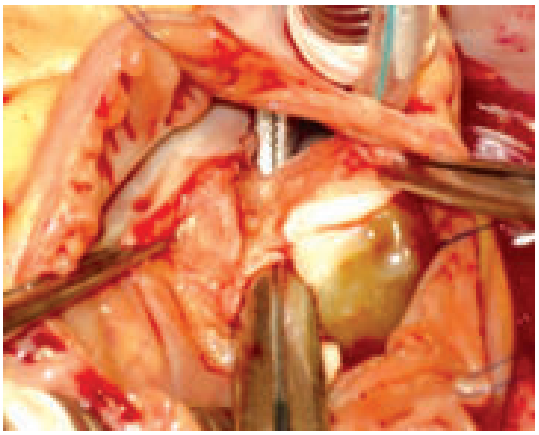

Fig.-3: *Left atrial myxoma seen after incising interatrial septum*[4]

Discussion:

In my study I found 62 cases of myxoma among about 3000 cardiac operations done at our centre over last three years and the incidence was around 2% .Age rage was 7.5 to 65 years. Male female ratio was 1:1.81. Most of the myxomas were located in the left atrium (87%) with high incidence in 4th and 5thdecade . Most of our patients were done as routine case with a post operative mortality 12.90%.

In an article as mentioned by Mandal et al Bangladesh, incidence of myxoma over a period of 17 years was 1.08%.[3] In a centre of Kolkata, India, this incidence of myxoma was 0.6% without any post operative mortality or recurrence .[4] In Rawalpindi, Pakistan. Cardiac myxomas constituted 0.40% of the total cardiac operations. They most commonly occurred in the fourth decade.In an article it is mentioned that they did routine operation on sixty-five patients whereas 28 patients with severe symptoms or embolic risk underwent emergency surgery.[5]High incidence of my study is due to short study period(03 years)with high number of myxoma in the background of relatively small number (3000) of total cardiac operations over three years in comparison to them. Most of our cases were operated as routine procedure.

KyoSeon Lee et al , Korea wrote in their article that total 93 cases were performed over 30 years. Of the 93 patients Male:Female=1:2.1 ,our M:F was1:1.81.their .mean age of patients was 54.7±16.6 years whereas our patients mean age was 36.94±13.99 years.it means our patients are affected and operated at a relatively younger age.Intheir study the origin site of the tumor was the left atrium (LA) in 92.5%, right atrium in 4.3%, left ventricle in 2.2%, multiple myxomas in both atria and the right ventricle in one patient (1.1%).In our study we found origin site in LA in 87.09%, in RA 9.67%, Biatrial 1.61 and in RV 1.61%.We did not find any LV and multicentric myxoma during our study period.[5]

In an article it is mentioned that the mean intensive care unit (ICU) stay of 2.3±0.8 days and mean hospital stay of 7.9±1.8 days.[10].Mean ICU stay of our patient was 6.5±3.53 days and hospital stay 20.61±10.60 days.Apparently in our hospital, both ICU stay and hospital stay were more than them. Moreover preoperative mean waiting time of our patients was9±2.12days.this aspect is not mentioned in their article.has been reported, resulting in a shorter length of hospital stay, and it is considered a safe and feasible method for atrial myxoma excision.There are few surgical techniques for myxoma resection other than midsternotomy like minimally invasive technique,endoscopic resection of the tumour and minithoracotomy with robot assisted surgery.[6,7,8].However we performed all our cases through midsternotomy we removed myxomas as follows :By LA tomy 90.74%,RA tomy in 5.55% biatrial in3.70%.They performed via a biatrial approach in 74.2%, atrial septotomy through right atriotomy in 17.2%, and left atriotomyonly in 8.6%.Our per operative aortic cross clamp time

was39.33±10,28min and cardiopulmonary bypass timew as 48±12.46min. Their mean cardiopulmonary bypass time was 80.7 ±39.0 min , and mean aortic cross-clamping time was 51.3 ± 27.5 min.Our surgical practice is a little bit different from them and our operation time is relatively shorter than their operative time.[5,6]

They found a pedunculated (tumour attached with a pedicle) mass in 67.6%, while the other 32.3% had a sessile mass (tumour attached with a broad base In our series myxomas were 85.48% cases pedunculated and 14.51% sessile.In their study simple myxoma resection including the endocardium and attached stalk without any need to repair was performed in 17 patients, direct closure of the defect area was performed in 47, and patch closure with autopericardium or prosthetic material was performed in 29 cases.[6] In an article it is mentioned that pretumorous cells around the stalk should be destroyed by laser photocoagulation which obviates the need for a wide surgical resection.[8]Since we did not have such facility in our institute we did low power electrocautary (10-15 w) using diathermy machine in 90.32% cases .We did suturing of the base of the stalk in 4.83%, excision and direct suturing of iatrogenic ASD in3.22% ,excision and pericardial patch closure of ASD in1.61% cases.

In a journal it has been mentioned that the level of restoration of normal quality of life within 30 days after atrial myxoma surgery is excellent with the robotically assisted than conventional approach.[11]

Regarding postoperative mortality of myxoma operation, different literature shows different results ranging from 0 to 8%.[4,5,12].Our postoperative mortality for myxoma patient was 12.90%.Obviously it is high.We need to find out the more causes and to take necessary steps soon to reduce this mortality.

Conclusion: We are dealing most of our cases as a routine procedure. Our preoperative waiting time is more due to multiple reasons. ICU and hospital stays are alsomore than others .We must reduce preoperative waiting time and operate the patients as either routine or emergency considering the clinical scenario of the patient .We should pay meticulous attention during and after operation to reduce mortality. We should improve our documentation and we should continue research on it .In future for better outcome we should adopt advanced technology when they will be available at our centre.

Acknowledgement:
I acknowledge the contribution of all the doctors of cardiac surgery units including my one, OT & ICU nurses and technicians of NICVD, Dhaka Bangladesh as they performed the operations and took care of their patients during and after surgery.

Conflict of interest : I do not have any conflict of interest with any one to disclose.

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Original Article

Echocardiographic Evaluation of left Ventricular Function Following Late Percutaneous Coronary Intervention after Acute Anterior Myocardial Infarction with Left Ventricular Systolic Dysfunction

Md. Tufazzal Hossen[1], Sayed Ali Ahsan[2], Md. Abu Salim[3], Khurshed Ahmed[3], Md. Mukhlesur Rahman[3], Dipal K Adhikary[3], Ariful Islam Joarder[4], Md. Fakhrul Islam Khaled[4], Madhusudan Paul[1], Abu Bakar Md. Jamil[1] Md. Zainal Abedin[5], Md. Fazlul Karim[6]

Abstract:

Background: The effect of late percutaneous coronary intervention on left ventricular function is incompletely understood. Objectives: To evaluate the effect of late Percutaneous Coronary Intervention on LV systolic function following coronary stenting after acute anterior myocardial infarction. Methods: A total of 60 patients, > 24 hours to 6 weeks after anterior AMI who attended in UCC, BSMMU between July 2014 to June 2015 were included in this study. They underwent coronary stenting. After coronary stenting all patients were in TIMI flow-3. Serial echocardiographic assessment of LV function before and after late intervention with modified Simpson's rule in apical 4 chamber view as well as comparison between baseline result with that of after intervention were done. The patients were on standard medical therapy in post intervention period. Result: Mean age was 54.3±8.91 years with minimum 30 years and maximum 75 years. Most of the patients were male (67%). LVESV was 60.0±14.4 ml before PCI and 58.3±15.3 ml at discharge (p value 0.091) & 44.1±17.6 ml after 3 months (p value <0.001). LVEF was 40.2±3.1% before PCI, 40.2±3.3% at discharge (p value 0.509) & 47.6±5.9% after 3 months (p value <0.001). There was no significant improvement of LV function from baseline till discharge but significant improvement occurred after 3months. Conclusion: Using echocardiographic techniques, our results showed that left ventricular volume decreased and the left ventricular ejection fraction increased significantly after three months of late intervention.

Key Words: Late Percutaneous Coronary Intervention (PCI), LVESV (left ventricular end systolic volume), LVEF (left ventricular ejection fraction)

(Bangladesh Heart Journal 2018; 33(2): 90-93)

Introduction:

Percutaneous coronary intervention (PCI) and stent placement has revolutionized the management of ischemic heart disease in terms of symptomatic improvement. However, it remains a question whether PCI and stenting do improve the left ventricular function and if it does, whether the improvement is to the same degree in all groups of

1. Resident (Phase-B), Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh
2. Professor, Bangabandhu Sheikh Mujib Medical University, Dhaka ,Bangladesh
3. Associate Professor, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh
4. Assistant Professor, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh
5. MD Final Part Student: Bangabandhu Sheikh Mujib Medical University, Dhaka , Bangladesh.
6. Assistant Professor, Sheikh Hasina Medical College, Jamalpur, Bangladesh.

Address of Correspondence: Md. Tufazzal Hossen, Resident Phase-B, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Mobile No. +8801915055310, E-mail: tulaluddin70@gmail.com

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patients like those with primary coronary intervention, late coronary intervention, the patients with chronic coronary occlusion, the patients with total occluded artery and additionally what role is played by the interval between PCI and myocardial infarction and also the progression of change of left ventricular function with time following PCI.

Myocardial revascularization using PCI is widely used and improves clinical outcome particularly in post infarction patients with markedly reduced LVEF.[1] Momtahen et al.[2] suggest that PCI is associated with a significant improvement in global and regional left ventricular(LV) function and favorable clinical outcome as shown by functional improvement in NYHA class and angina severity. This improvement of LV contractility was significant 3 months post-PCI whereas it did not show further significant improvement thereafter. The LVEF improvement was nonetheless more pronounced in patients with baseline LVEF ≤40%.

Although the short term and long term beneficial effects of primary PCI are established but the benefit of late PCI is not out of controversy. Late percutaneous coronary intervention (PCI) after Acute Myocardial Infarction (AMI) is increasingly used as treatment strategy. It is a necessity to provide a research based information regarding the opportunity of improvement of left ventricular function following late percutaneous coronary intervention

This study evaluated the effect of late percutaneous coronary intervention on left ventricular systolic function by echocardiography after anterior AMI.

Methods:

This prospective observational study was done in University Cardiac Centre (UCC), Bangabandhu Sheikh Mujib Medical University (BSMMU) from July 2014 to June 2015. Sample size was calculated using the formula for determining sample size to show difference between two means. It was 60. Patients presenting between 24 hours to six weeks of acute anterior STEMI (ST-elevation myocardial infarction) in UCC from July 2014 to June 2015 were considered for the study. Among them, patients having ischemic symptoms or positive evidence of inducible ischemia in ETT, significant lesion at LAD (left anterior descending artery) and left ventricular (LV) mild to moderate systolic dysfunction were enrolled. Patients who underwent primary PCI, presented more than 6 weeks after acute myocardial infarction, with valvular heart disease, with unsuccessful PCI, having severe LV systolic dysfunction (EF <30%) or normal LV systolic function and coronary involvement other than LAD were excluded from the study.

Patients' demographic profiles were recorded. All patients underwent 2-dimensional echocardiography before PCI. Follow up echocardiogram was done at discharge and after 3 months of PCI to assess LV systolic function. For 2-dimensional echocardiography, a vivid 7 system with phase array probe (3.5 MHz) was used. Estimates of LV end systolic volume (ESV) and ejection fraction (EF) were obtained from the average of three consecutive cardiac cycles taken from apical four chamber view using the modified Simpson's rule. Measurements were performed off-line by two independent echocardiographers who were blind to each other. Mean values from two independent reporters were taken as final value. Data was collected in a pre-designed form.

Statistical Analysis:

Statistical analyses were carried out by using SPSS (the Statistical Package for Social Sciences version 22 for Windows). Categorical variables were expressed in percentage. Continuous variables were expressed in mean±SD (Standard deviation). Baseline echocardiographic findings were compared with those at discharge and 3 month with paired Student t test. P value <0.05 was considered as statistically significant.

Results:

This study evaluated effect of late percutaneous coronary intervention on left ventricular systolic function after acute AMI in terms of LVESV and EF at baseline and after 3 months.

Demographic profile of the study population:

Figure-1 shows the age distribution of the study patients, most of the patients belonged to 51-60 years age group. Mean age 54.3±8.91 years, minimum age 30 and maximum 75 years.

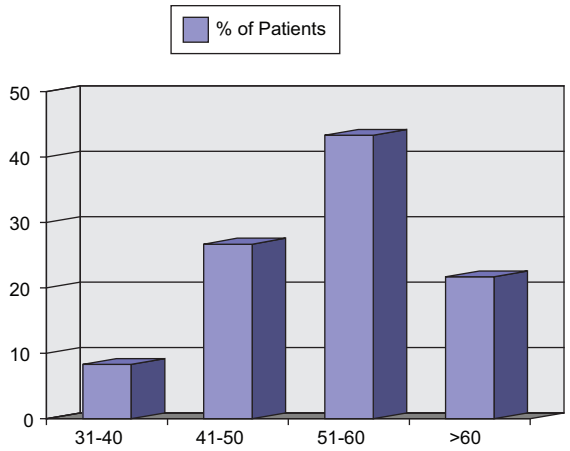

Fig.-1: *Age distribution of the study patients (n=60)*

Figure-2 showed the sex distribution of study patients. Males were predominant with male to female ratio being 2:1.

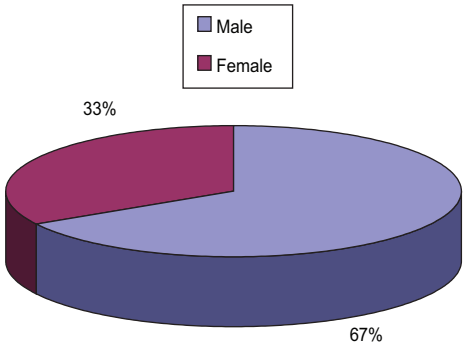

Fig.-2: *Sex distribution of the study patients (n=60)*

Table-1 shows the risk factors of the study patients.

Table-I

Distribution of the study patients by risk factors (n=38)

Risk factors	Number	Percentage (%)
Diabetes mellitus	17	28.3
Hypertension	24	40.0
Dyslipidemia	11	18.3
Smoking	8	13.3

n= Number of patient (60)

Table-2 shows the comparison of echocardiographic parameters before PCI and at discharge of the studied patients. LVESV& EF did not show significant change from baseline to at discharge.

Table-II

Comparison of echocardiographic parameters before PCI and at discharge

Echo-cardiographic variables	Before PCI (n=60)	At discharge (n=60)	P value
LVESV(ml)	60.0±14.4	58.3±15.3	.091^{ns}
EF(%)	40.2±3.1	40.2±3.3	.509^{ns}

Paired t-test were performed to compare the echocardiographic variables before PCI and at discharge.
SD= Standard déviations
ns= Non Significant
n= Number of patient (60)
LVESV= Left Ventricular End Systolic Volume , EF= Ejection Fraction

Table-3 shows the comparison of echocardiographic parameters before PCI and after 3 months of PCI. LVESV significantly reduced and LVEF significantly improved.

Table-III

Comparison of echocardiographic parameters before PCI and three months post PCI

Echo-cardiographic variables	Before PCI (n=60)	After 3 months (n=59)	P value
LVESV(ml)	60.0±14.4	44.1±17.6	<0.001s
EF(%)	40.2±3.1	47.6±5.9	<0.001s

Data were expressed as mean±SD
Paired t-test were performed to compare echocardiographic variables before PCI and after 3 months.
SD= Standard Deviation
S= Significant.
n= Number of patient (60)
n= Number of patient (59)
LVESV= Left Ventricular End Systolic Volume , EF= Ejection Fraction

Discussion:

Percutaneous coronary intervention (PCI) is the treatment of choice in patients presenting with acute myocardial infarction.

In this study, we assessed 60 anterior MI patients by echocardiography before and after PCI. Significant improvement in LVEF was found after 3 months of intervention.

Silva et al.[3] have shown that late recanalization, 12 hours to 14 days post anterior MI improved LVEF and myocardial contractility. Buszman et al.[4] revealed that LVEF was increased 6±7.2 % after PCI. Improvement of LVEF was seen in other study by Ioannidis et al[5]., LVEF improved from 40±17% to 54±15% in Remmelink et al[6]. and from 48.8±11.6% to 52.5±11.5% in Agirbasli et al.[7] study. Banerjee et al.[8] in another study reported that late PCI on persistent total occlusion 3-28 days after MI did not observe any change in LVEF compared with optimal medical therapy. On the other hand, Carluccio et al.[9] demonstrated that PCI improved LVEF (from 32% to 43%; *P*=0.0004).

In this study in Bangladeshi people, out of 60 patients one patient died. In this study, mean LVESV before PCI was 60.0±42.7ml but at discharge was 58.3±15.3ml, which is not statistically significant (p value =0.091). Horie et al.[10] documented mean LVESV at baseline 34.6±10.6 ml decreased to 31.1±11.2 ml after one month (p > 0.05).

In this study, mean EF before PCI was 40.2±3.1% but at discharge was 40.2±3.3%, which is not statistically significant (p value =0.509). Horie et al.[10] documented mean EF at baseline 48.5±8.65% increased to 53.9±8.96% after one month (p = 0.01). Nozari et al.[11] conducted a study where earliest interval of MI and PCI was 3 weeks. Mean EF increased before PCI to at discharge from 40.52±6.36% to 41.83±7.14% (p =0.143).

In this study, mean LVESV before PCI was 60.0±14.4ml but at 03 month of PCI was 44.1±17.6ml, which is statistically significant (p value <0.001). Baks et al.[12] demonstrated mean end systolic volume index decreased significantly from 34±13 ml/m^2 to 31±13 ml/m^2 (p = 0.02) after 5 months of PCI.

In this study, mean EF before PCI was 40.2±3.1% but at 03 month of PCI was 47.6±5.9%, which is statistically significant (p value <0.001). Baks et al.[12] demonstrated overall mean ejection fraction remained unchanged from 61±9% to 62±11% (p=0.54) after 5 months of PCI. Nozari et al.[11] conducted a study where earliest interval of MI and PCI was 3 weeks. Mean EF increased before PCI to at 3 months from 40.52±6.36% to 44.0±7.89% which was highly significant (p <0.001).

Mean value of LVESV was higher and mean value of LVEF was lower in this study in comparison to Horie at al.[10] probably because of anterior MI, LV dysfunction and only LAD involvement were the selection criteria.

Conclusion:

It is concluded that Late percutaneous coronary intervention in AMI (anterior) improves left ventricular systolic function. Further multicentric study on large sample size and for long duration is needed.

Limitations

The study has some limitations:

1. This study was performed only for a short period.
2. This study was done in highly selective group of patients comprising small cohort in one hospital only, which may not reflect the true picture of Bangladeshi patients.
3. Multicentric study was not done.

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Original Article

Impact of Admission Blood Glucose Added on GRACE Risk Score for All-Cause In-Hospital Mortality in Patients with Acute Coronary Syndrome

Md. Mesbahul Islam[1], Mohsin Ahmed[2], Abdul Wadud Chowdhury[3], Mohammad Ali[4], Khandakar Abu Rubayat[5]

Abstract:

Background: Abnormal glucose metabolism is a predictor of worse outcome after acute coronary syndrome (ACS). However, this parameter is not included in risk prediction scores, including GRACE risk score. We sought to evaluate whether the inclusion of blood glucose at admission in a model with GRACE risk score improves risk stratification. Objectives: To assess whether inclusion of admission blood glucose in a model with GRACE risk score improves risk stratification of ACS patients admitted in a tertiary hospital of Bangladesh. Methods: This cross sectional comparative study was carried out in the department of cardiology, Dhaka Medical College Hospital (DMCH), Dhaka between May 2016 to April 2017. Data were collected from ACS patients admitted at CCU, DMCH who fulfilled inclusion and exclusion criteria. GRACE score was calculated for each patient. The predictive value of death by GRACE score was compared with the predictive value of combined GRACE score + admission blood sugar. Comparison between these results in two groups were done by unpaired t-test, analysis was conducted SPSS-22.0 for windows software. The significance of the results was determined in 95.0% confidence interval and a value of p <0.05 was considered to be statistically significant. Results: A total of 249 cases of ACS patients were selected. Most of the patients belonged to 5th and 6th decades 25.3% vs 37.3% and the mean age was 55.7±11.7 years. Most of the patients were male. High GRACE risk score (≥155) and elevated admission blood sugar (≥11) was found significantly higher in-hospital death whereas only high GRACE risk score (≥155) and normal admission blood sugar (<11) was found non significant regarding in-hospital death. Test of validity showed sensitivity of GRACE risk score regarding in-hospital death was 85.29%, specificity 57.7%, accuracy 61.4%, positive and negative predictive values were 24.2% and 96.1% respectively. The sensitivity of GRACE risk score + admission blood sugar regarding in-hospital death was 85.29%, specificity 62.33%, accuracy 65.46%, positive and negative predictive values were 26.36% and 96.4% respectively. Receiver-operator characteristic (ROC) were constructed using GRACE score and GRACE score + admission blood sugar of the patients with in-hospital death, which showed the sensitivity and specificity of GRACE score for predicting in-hospital death were found to be 79.4% and 58.1%, respectively. Whereas after adding admission blood sugar value to GRACE score both the sensitivity and specificity increased to 82.4% and 58.6% respectively in this new model. Logistic regression analysis of in-hospital mortality with independent risk factors showed GRACE score (≥155) + admission blood sugar (≥11.0 mmol/l) was more significantly associated with in-hospital mortality (P =0.001, OR = 6.675, 95% CI 2.366-13.610). Conclusion: In patients with the whole spectrum of acute coronary syndrome admission blood glucose can add prognostic information to the established risk factors with the GRACE risk score.

Keywords: Admission blood glucose, GRACE risk score, acute coronary syndrome.

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1. Registrar, Department of Cardiology, Ibn Sina Specialized Hospital, Dhanmondi, Dhaka, Bangladesh.
2. Associate Professor, Dept. of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh.
3. Professor, Dept. of Cardiology, Dhaka Medical College, Dhaka, Bangladesh.
4. Specialist, Department of Cardiology, Bangladesh specialized Hospital, Shyamoli, Dhaka, Bangladesh.
5. MD, final Part, Dhaka Medical College, Dhaka, Bangladesh.

Address of Correspondence: Dr. Md. Mesbahul Islam, Registrar, Department of Cardiology, Ibn Sina Specialized Hospital, Dhanmondi, Dhaka, Bangladesh. Cell no. +8801710834077, Email: dmesbah37@gmail.com

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Introduction:

Coronary artery disease (CAD) is an increasingly important medical and public health problem and is the leading cause of mortality in Bangladesh as well. Like other South Asians, Bangladeshis are unduly prone to develop CAD, which is often premature in onset, follows a rapidly progressive course and angiographically more severe.[1] Some of the independent predictors of early death from STEMI include age, Killip class, time to reperfusion, cardiac arrest, tachycardia, hypotension, anterior infarct location, prior infarction, diabetes mellitus, smoking status, renal function and biomarker findings.[2] The presence of diabetes doubled the age adjusted risk for cardiovascular disease in men and tripled it in women in the Framingham Heart Study and it remained an independent risk factor even after adjusting for age, hypertension, smoking, hyperlipidemia, and left ventricular hypertrophy.[3] Acute hyperglycemia is common in patients with STEMI even in the absence of a history of type 2 DM. Hyperglycemia is encountered in up to 50% of all STEMI patients, whereas previously diagnosed DM is present in only 20 % to 25 % of STEMI patients.[4] In the clinical practice, admission plasma glucose is used as a measure of Acute Hyperglycemia and Hemoglobin Alc (HbA1c) for Chronic Hyperglycemia. Acute Hyperglycemia was defined as plasma glucose >198 mg/dl (11 mmol/L) at admission, regardless of diabetic status.[5]

Elevated plasma glucose and glycated hemoglobin levels on admission are independent prognostic factors of both in-hospital and long term outcome regardless of diabetic status.[6] For every 18 mg/dL increase in glucose level, there is a 4 % increase in mortality in non-diabetic subjects.[7] Admission glucose has been identified as a major independent predictor of both in-hospital Congestive Heart Failure and Mortality in STEMI.[8] Hyperglycemia at presentation, while often reflecting undiagnosed and persisting abnormalities of glucose handling, may also represent a transient stress response mediated through the autonomic nervous system with release of catecholamines and adrenal corticosteroids.[9] This catecholamines response occurs early, is restricted to the first five days and is proportional to the size of infarction, being associated with faster heart rate, poorer Killip class and lower ejection fraction on discharge.[10] Hyperglycemia is associated with large infarction and depressed left ventricular function, heart failure on admission and elevated Brain Natriuretic Peptide.[11] On the other hand whatever the cause of hyperglycemia in acute myocardial infarction, it has got a detrimental effect on myocardium itself. Effects of hyperglycemia include the promotion of oxidative stress, impairment of endothelial function, promotion of coagulation, non-enzymatic glycation of platelet glycoproteins with abrupt changes in agreeability, amplification of inflammation, suppression of immunity and direct toxicity to myocytes and promotion of apoptosis. Acute hyperglycemia has been shown to impair ischemic preconditioning, attenuate the protective effect of pre-infarction angina on microvascular function and reduce the effectiveness of collateral blood supply into ischemic zones.[12]

An ACS may take the form of an ST-elevation myocardial infarction (STEMI), a non-ST-elevation myocardial infarction (NSTEMI), or unstable angina. The Global Registry of Acute Coronary Events (GRACE) risk score is a validated and established score for risk stratification of patients with acute coronary syndromes, obtained from a multicentre registry.[13] Though elevated plasma glucose is an independent prognostic factor is not included in this risk scoring system. We sought to evaluate whether inclusion of admission blood glucose in a model with GRACE risk score improves risk stratification.

Methodology:

The cross-sectional comparative study was carried out in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka from May 2016 to April 2017. All the patients of Acute Coronary Syndrome (STEMI, NSEMI, UA) after exclusion and inclusion criteria were taken as sampling population. Patients/attendance was briefed about the study and consent was taken. Brief history was taken included with symptoms and risk factors. Relevant physical examination, 12 lead ECG was done on admission and routinely thereafter. Blood glucose level e”11.0 mmol/l or 198 mg/dl was considered as admission hyperglycemia. Baseline investigations including-cardiac biomarkers, serum creatinine, lipid profile and echocardiography were done for each patient. All the above informations were recorded in a data collection form consisting of relevant questionnaire. GRACE score for each patient was calculated by using the online GRACE risk calculator by eight variables taken into account: patient age, heart rate, systolic blood pressure, serum creatinine, Killip heart failure class, the existence or not of cardiac arrest at admission, any deviations of the ST segment and cardiac enzyme levels. Then occurrence of in-hospital death and complications (acute LVF, cardiogenic shock, ventricular tachycardia, ventricular fibrillation, asystole and AV block) were recorded. The predictive value of death by GRACE score was compared with the predictive value of combined GRACE score + admission blood sugar. Comparison between these results in two groups were done by chi square test, analysis was conducted SPSS-23.0 for Windows software. The significance of the results was determined in 95.0% confidence interval and a value of $p < 0.05$ was considered to be statistically significant.

Results:

Most of the patients belonged to 5th and 6th decades 25.3% vs 37.3% and the mean age was 55.7±11.7 years. Most of the patients were male. Male female ratio was 2.8:1. (Table I). High GRACE risk score (≥155) and elevated admission blood sugar (≥11) was found significantly higher in-hospital death whereas only high GRACE risk score (≥155) and normal admission blood sugar (<11) was found non significant regarding in-hospital death (Table II). Test of validity showed the sensitivity of GRACE risk score regarding in-hospital death was 85.29%, specificity 57.7%, accuracy 61.4%, positive and negative predictive values were 24.2% and 96.1% respectively. The sensitivity of GRACE risk score plus admission blood sugar regarding in-hospital death was 85.29%, specificity 62.33%, accuracy 65.46%, positive and negative predictive values were 26.36% and 96.4% respectively (Table III). Receiver-operator characteristic (ROC) curves were constructed using GRACE score and GRACE score + admission blood sugar of the patients with in-hospital death, which showed the sensitivity and specificity of GRACE score for predicting in-hospital death were found to be 79.4% and 58.1%, respectively. Whereas after adding admission blood sugar value to GRACE score the sensitivity increased to 82.4% and specificity increased to 58.6% at the same cut off value (Table IV). Logistic regression analysis of in-hospital mortality with independent risk factors showed GRACE score (≥155) + admission blood sugar (≥11.0 mmol/l) was more significantly associated with in-hospital mortality (OR = 6.675, 95% CI 2.366-13.610). (Table V).

Table-I

Demographic characteristics of the study patients (n=249)

Age (years)	Number of patients	Percentage
≥30	6	2.4
31-40	24	9.6
41-50	63	25.3
51-60	93	37.3
61-70	42	16.9
>70	21	8.4
Mean±SD	55.7±11.7	
Sex		
Male	184	73.9
Female	65	26.1
Smoking	134	53.8
Diabetes mellitus	43	17.3
Hypertension	131	52.6
Dyslipidemia	34	13.7
Family H/o CAD	64	25.7

Table-II

Association of in-hospital death with GRACE risk score plus admission blood sugar (n=249)

GRACE risk score plus admission blood sugar	In-hospital death				p value
	Yes(n=34)		No(n=215)		
	n	%	n	%	
GRACE risk score ≥155 and admission blood sugar ≥11 mmol/L	29	85.3	81	37.7	0.001[s]
GRACE risk score ≥155 and admission blood sugar <11 mmol/L	11	32.4	50	23.3	0.252[ns]

s=significant, ns= not significant
P value reached from Chi square test

Table III

Sensitivity, specificity of predicted in-hospital death between GRACE risk score and GRACE risk score plus admission blood sugar.

	Sensitivity	Specificity	Accuracy	PPV	NPV
GRACE risk score (≥155)	85.29	57.7	61.4	24.2	96.1
GRACE risk score (≥155) and admission blood sugar (≥11 mmol/L)	85.29	62.33	65.46	26.36	96.4

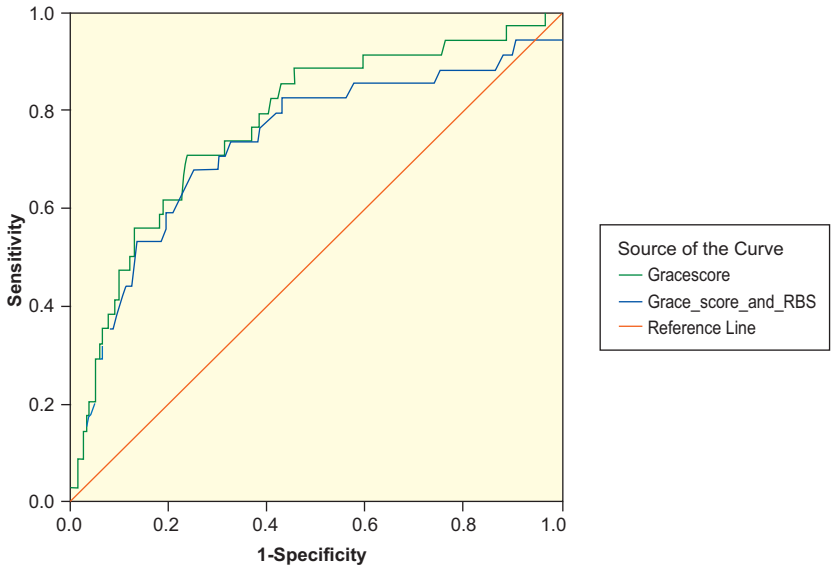

Fig.-1: *Receiver-operator characteristic curves of GRACE score and GRACE score + admission blood sugar.*

Table-IV

Receiver-operator characteristic (ROC) curve of GRACE score and GRACE score + admission blood sugar for prediction of in-hospital death

	Cut off value	Sensitivity	Specificity	Area under the ROC curve	95% Confidence interval (CI)	
					Lower bound	Upper bound
GRACE score	≥155	79.4	58.1	0.731	0.627	0.836
GRACE score + admission blood sugar	≥155	82.4	58.6	0.769	0.680	0.859

Table-V

Multi variable logistic regression analysis (n=249)

	Adjusted OR	95% CI		P Value
		Lower	Upper	
Age (>50 years)	0.333	0.114	0.975	0.045[s]
GRACE score (≥155)	5.840	1.407	24.235	0.015[s]
Admission blood sugar (≥11.0 mmol/l)	1.794	0.633	5.083	0.271[ns]
GRACE score (≥155) + admission blood sugar (≥11.0 mmol/l)	6.675	2.366	13.610	0.001[s]
Diabetes mellitus	0.171	0.020	1.471	0.108[ns]
Smoking	1.768	0.676	4.624	0.246[ns]
Dyslipidaemia	0.417	0.077	2.253	0.310[ns]
Heart failure (Killip class II-IV)	1.842	0.528	6.434	0.338[ns]
Ejection fraction (<30%)	0.133	0.001	1.210	0.998[ns]

s=significant, ns= not significant

Multivariable logistic regression analysis was performed

Discussion:

This cross sectional comparative observational study was conducted in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka, over a period of one year from May 2016 to April 2017. The main objective was to assess whether inclusion of admission blood glucose in a model with GRACE risk score improves risk stratification of ACS patients. For this purpose 249 patients with acute coronary syndrome were included according to exclusion and inclusion criteria. In our study most of the patients belongs to 5th and 6th decades 25.3% vs 37.3% and the mean age was 55.7±11.7 years. Nearly similar results were observed by Mudespacher et al.[14] and Timoteo et al.[15]. Most of the patients were male. Male female ratio was 2.8:1. High GRACE risk score (e"155) and elevated admission blood sugar (e"11) was found significantly higher in-hospital death whereas only high GRACE risk score (e"155) and normal admission blood sugar (<11) was found non significant regarding in-hospital death.

Several possible mechanisms may explain this observation. First, hyperglycemia is a reflection of relative insulin deficiency, which is associated with increased lipolysis and excess circulating free fatty acids; this effect may be exaggerated in cases of acute stress such as myocardial infarction.[16,17] Free fatty acids, although normally the substrate of choice for healthy myocardium, are toxic to ischemic myocardium and may lead to damaged cardiac-cell membranes, calcium overload, and arrythmias.[18] Insulin deficiency may also limit the ability of cardiac muscle to take up glucose for anaerobic metabolism. Second, acute hyperglycemia may precipitate an osmotic diuresis. The resulting volume depletion may interfere with the Frank-Starling mechanism, an important compensatory mechanism for the failing left ventricle in which increased end-diastolic volume leads to increased stroke volume.[19,20] Third, stress hyperglycemia may be a marker of more extensive cardiac damage in acute myocardial infarction.[21] More extensive cardiac damage may lead to a greater rise in stress hormones (promoting glycogenolysis and hyperglycemia) and may also increase the risk of congestive heart failure and mortality. Thus, stress hyperglycemia could simply be an epiphenomenon reflecting the most severe cardiac damage. Fourth, patients who develop stress hyperglycemia are likely to be dysglycemic when not stressed. Patients with dysglycemia are at a higher risk of cardiovascular disease than patients who have normal blood glucose,[22] and may have a worse prognosis after acute myocardial infarction because of more extensive underlying coronary artery disease.

In this study we found that the sensitivity of GRACE risk score regarding in-hospital death was 85.29%, specificity was 57.7%, accuracy was 61.4%, positive and negative predictive values were 24.2% and 96.1% respectively. The sensitivity of GRACE risk score + admission blood sugar regarding in-hospital death was 85.29%, specificity was 62.33%, accuracy was 65.46%, positive and negative predictive values were 26.36% and 96.4% respectively. Similar results were also observed by Timoteo et al.[15].

A report from the GRACE registry showed that short-term and six-month mortality was increased significantly with higher admission glucose levels in patients across the whole spectrum of acute coronary syndromes.[23] This association is probably mainly driven by an increased risk of early death, consistent with the paradigm that admission glucose level is a marker of stress rather than a reflection of a general glucometabolic state.[24]

Receiver-operator characteristic (ROC) were constructed using GRACE score and GRACE score + admission blood sugar of the patients with in-hospital death, which showed the sensitivity and specificity of GRACE score for predicting in-hospital death were found to be 79.4% and 58.1%, respectively. Whereas after adding admission blood sugar value to GRACE score both the sensitivity and specificity increased to 82.4% and 58.6% respectively in this new model.

Logistic regression analysis of in-hospital mortality with independent risk factors showed GRACE score (e"155) + admission blood sugar (e"11.0 mmol/l) was more significantly associated with in-hospital mortality (P =0.001, OR = 6.675, 95% CI 2.366-13.610). So, the new model better identifies those who do not have events than those who do. Thus the new model (with the addition of admission blood glucose to GRACE score) is better at identifying 'truly low-risk' patients and is as good as in identifying patients who develop events. This might not be ideal when we are evaluating a risk score to identify high-risk patients. However, recent cardiovascular disease guidelines are encouraging a practice shift toward greater focus on identification of 'truly low-risk' patients instead of focusing on identification of high-risk patients. This allows a better selection of patients avoiding unnecessary interventions that might increase costs as well as the risk of procedure-related adverse events.

Conclusion:

Admission hyperglycemia is associated with increased mortality of patients with acute coronary syndrome. Therefore inclusion of admission blood glucose can add prognostic information to the established risk factors with the GRACE risk score.

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Original Article

Correlation between Inflammatory Marker and Glycemic Control in Patients with Ischemic Heart Disease

Mohsin Ahmed[1], Md Mesbahul Islam[2] , Mohammad Arifur Rahman[3], Khandaker Abu Rubaiyat[4] , C. M Khudrate-E-Khuda[5], Kazi Abul Fazal Ferdous[6] , Bikash Chandra Das[7], Sanoat Kalam Linda [8]

Abstract:

Background: Recent evidence suggests that inflammatory markers and poor glycemic control are significantly associated with the development of cardiovascular complications. The purpose of this study was to determine the association between inflammatory marker (CRP) and glycemic status (HbA1c) in ischemic heart disease patients.

Method: This cross sectional study was performed on 668 patients of ischemic heart disease in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka, who underwent Coronary angiogram from January 2017 to December 2017. CRP value were divided into normal (<6 mg/L), borderline (6-10 mg/L) and high (>10 mg/L) and HbA1c was divided <6.5% and ≥6.5%. After performed Coronary angiography the extent of disease was divided into insignificant CAD of (<50% stenosis), significant CAD considered as >50% stenosis and single vessel, double vessel, triple vessel CAD and normal coronaries. The relationship between CRP with HbA1c was analyzed by Chi square test. ANOVA test was used to analyze the continuous variables, shown with mean and standard deviation. Pearson's correlation coefficient was used to test the relationship between CRP and HbA1c in CAD patients. p value <0.05 was considered as statistically significant.

Result: Most (65.0%) of the patients belonged to age 41-60 years. The mean age was found 51.4±10.7 years. Majority (82.3%) of patients were male. Among risk factors, highest (40.0%) patients had hypertension followed by 209 (31.3%) diabetes mellitus and 204 (30.5%) smoker. Positive correlation was found (r=0.220, p= 0.001) between HbA1c with CRP in CAD patients. High CRP was found 138(38.4%) in <6.5% HbA1c and 187(60.5%) in ≥6.5 percent HbA1c. The difference was statistically significant (p<0.05). Multi variable logistic regression was found high HbA1c, high CRP and diabetes mellitus were statistically significant (p<0.05) in severe CAD (Double and triple vessel) patient.

Conclusion: Positive correlation was found between serum levels of CRP and HbA1c in CAD patients. Thus, aiming at good glycemic control and estimation of serum CRP levels will possibly be of help in planning early intervention, thereby preventing further complications which in turn may help preserve cardiac functions in ischemic heart disease patients.

Keywords: Coronary artery disease, C-reactive protein, HbA1c

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1. Associate Professor, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh
2. Registrar, Department of Cardiology, Ibn Sina Hospital, Dhaka, Bangladesh
3. Junior Consultant, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh
4. MD, Final Part, Department of Cardiology , Dhaka Medical College, Dhaka, Bangladesh
5. Junior Consultant, Department of Cardiology, Dhaka Medical College, Dhaka, Bangladesh
6. Medical Officer, Department of Cardiology , Dhaka Medical College, Dhaka, Bangladesh
7. Registrar, Department of Cardiology, Dhaka Medical College, Dhaka, Bangladesh
8. FCPS Part-II, Department of Medicine, Bangabandhu Sheikh Mujib Medical Unerversity, Dhaka, Bangladesh

Address of Correspondence: Dr. Mohsin Ahmed, Associate Professor, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh. Cell: +8801613393186, E-mail: mohsinsohel07@gmail.com

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Introduction:

The autoimmune response associated with overproduction of T helper-1 (Th1) cytokines which activate macrophage production of proinflammatory mediators interleukin-6 (IL-6) and TNF-±.[1] IL-6 is produced also by a variety of cells such as adipocytes, which produce 30% of the circulating IL-6, fibroblasts and endothelial cells.[2] It mediates damage to micro- and macro-vascular tissues, altered insulin secretion either directly or through stimulation of free fatty acid production and altered glucose homeostasis.[3] C-reactive protein is an acute-phase protein and a marker of non-specific inflammation synthesized in the liver. The biosynthesis of CRP is largely regulated by IL-6.[4] Plasma markers of inflammation, such as CRP and IL-6 are positively associated with risk of vascular disease in non diabetic individuals.[5] Recently, inflammation has been considered, at least in part, to lead to the development and progression of atherosclerosis.[6]

C-reactive protein (CRP), a marker of systemic inflammation, is emerging as an independent risk factor for cardiovascular disease.[7–9] High CRP levels have been linked to an increased risk of thrombotic events including myocardial infarction.[9–11] Elevated CRP levels have also been linked to an increased risk of later development of diabetes.[12,13] Furthermore, CRP levels are higher in people with diabetes compared with those without diabetes.[14–16] Less is known about whether CRP in people with diabetes is related to level of glycemic control. Wu et al.[17] found that CRP is associated with HbA1c levels.

Elevated glycohemoglobin A1 (HbA1c) is an established predictor for developing atherosclerosis.[18,19] Eeg-Olofsson et al.[20] studied a total of 7,454 patients from the Swedish National Diabetes Register over a period of 5 years (aged 20–65 years, diabetes duration 1-35 years) and found a progressively increasing risk of coronary heart disease and cardiovascular diseases with higher HbA1c levels independent of traditional risk factors. HbA1c is a better marker for determining risks of CAD and mortality than fasting blood glucose and even non-diabetic patients with elevated HbA1c levels are also at increased risk for CVD and mortality.[21] Both enhanced inflammation and hyperglycemia contribute to the development and progression of atherosclerosis and are frequently found in patients with clinically advanced disease. Given the interrelation between inflammation, hyperglycemia, and atherosclerotic disease.

There was a statistically significant positive correlation of serum hsCRP levels with HbA1c indicating the role of poor glycemic control. Studies have shown similar association between hyperglycemia and inflammation.[22] It is known that glycation triggers the inflammatory process, leading to a rise in hsCRP levels. Thus, hsCRP can predict the onset of glycation-induced inflammatory process secondary to poor glycemic control.[23]

To provide further insight into the role of inflammation in the development of cardiovascular disease, we sought to elucidate the link between level of glycemic control and inflammation. The purpose of the study was to investigate the correlation between CRP and HbA1c in the patients with ischemic heart disease.

Methodology:

This cross sectional study was performed on 668 patients of ischemic heart disease (CSA, UA, NSTEMI and STEMI) in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka, who were underwent Coronary angiography from January 2017 to December 2017. Demographic variables, such as age and sex, west and hip circumference and angiography results were recorded. After explaining the aims of the study and obtaining the patient's approval for participation blood samples were sent. CRP value were divided into normal (<6 mg/L), borderline (6-10 mg/L) and high (>10 mg/L)[19] and HbA1c was divided <6.5% and e"6.5%. After performed Coronary angiography the extent of disease was divided into insignificant CAD of (<50% stenosis), significant CAD considered as >50% stenosis[20] and single vessel, double vessel, triple vessel CAD and normal coronaries. The relationship between CRP with HbA1c was recorded by Chi square test. Statistical Package for the Social Sciences (SPSS) version 23.0 for windows was used to analyze the data. Categorical variables were expressed as proportions (percentages) and numerical data was expressed as means (standard deviations) and ranges. ANOVA test was used to analyze the continuous variables, shown with mean and standard deviation. Pearson's correlation coefficient was used to test the relationship between CRP and HbA1c in CAD patients. p value <0.05 was considered as statistically significant.

Results:

This cross sectional study was performed on 668 patients of ischemic heart disease (CSA, UA, NSTEMI and STEMI) in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka, who were underwent Coronary angiography from January 2017 to December 2017.

Most (65.0%) of the patients belonged to age 41-60 years. The mean age was found 51.4±10.7 years with range from 25-85 years. Majority (82.3%) patients were male and 390 (58.4%) patients were illiterate (Table-1). In risk factors, highest 267 (40.0%) patients had hypertension followed by 209 (31.3%) diabetes mellitus, 204 (30.5%) smoker, 189

(28.3%) H/O ischemic heart disease and 151 (22.6%) dyslipidemia (Table-2). Positive correlation (r=0.220, p= 0.001) of HbA1c with CRP (Figure 1). High CRP was found 138(38.4%) in <6.5 percent HbA1c and 187(60.5%) in e"6.5 percent HbA1c. The difference was statistically significant (p<0.05) (Table III). Multi variable logistic regression was found high HbA1c, high CRP and diabetes mellitus were statistically significant (p<0.05) in severe CAD (Double and triple vessel) patients (Table IV).

Table-I

Demographic characteristics of the study subjects (n=668)

Demographic characteristics	Frequency	Percentage
Age (in years)		
≤40	123	18.4
41-60	434	65.0
>60	111	16.6
Mean±SDRange (min-max)	51.4±10.7(25–85)	
Sex		
Male	550	82.3
Female	118	17.7
Educational status		
Illiterate	390	58.4
Primary	110	16.5
Secondary	111	16.6
Higher	37	5.5
Graduate and above	20	3.0

Table-II

Distribution of the study subjects by clinical risk factors (n=668)

Risk factors	Frequency	Percentage
Diabetes mellitus	209	31.3
Hypertension	267	40.0
Dyslipidemia	151	22.6
Obesity	28	4.2
Smoking	204	30.5
Tobacco	97	14.5
Alcohol	2	0.3
Family history of CAD	31	4.6
H/O ischemic heart disease	189	28.3
Previous PTCA	11	1.6
Previous CABG	10	1.5

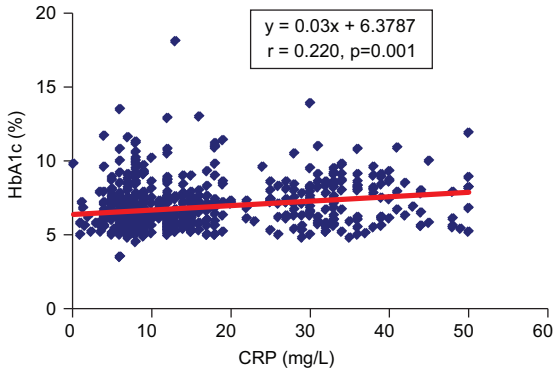

Fig.-1: *Scatter diagram showing the positive correlation (r=0.220, p= 0.001) of HbA1c with CRP (n=668).*

Table III

Association between HbA1c with CRP of the study population

CRP	HbA1c		p value
	<6.5n (%)	≥6.5n (%)	
Normal (<6 mg/L)	33 (9.2)	23 (7.4)	0.001[s]
Borderline (6-10 mg/L)	188 (52.4)	99 (32.0)	
High (>10 mg/L)	138 (38.4)	187 (60.5)	

Data were analyzed by Chi-square test, s= significant

Table-IV

Multi variable logistic regression analysis for severe CAD

	Adjusted OR	95% CI		P Value
		Lower	Upper	
HbA1c (e"6.5)	0.261	0.025	0.882	0.023[s]
CRP (>10 mg/L)	30.222	8.874	99.389	0.001[s]
Diabetes mellitus	0.103	0.011	0.953	0.045[s]
Hypertension	1.059	0.268	4.181	0.935[ns]
Dyslipidemia	0.698	0.146	3.346	0.653[ns]
Smoking	0.547	0.143	2.092	0.378[ns]
Constant	0.007	-	-	0.001[s]

s= significant, ns= not significant

DISCUSSION:

Recently, inflammation has been implicated in the development and progression of atherosclerosis. From the pathological viewpoint, all stages i.e. initiation, growth and complications of the atherosclerotic plaque, may be considered as inflammatory responses to vascular endothelial injury. Being the major cause of mortality and morbidity in patients with T1DM[24] it is very important to study and monitor markers of inflammation to define patients at higher risk of vascular complications.

Glycemic control, BMI, LDL cholesterol, HDL cholesterol, triglycerides, and systolic blood pressure were defined as the determinants of inflammatory activity in type 1 diabetes.[25,26]

In this present study it was observed that most (65.0%) of the patients belonged to age 41-60 years. The mean age was found 51.4±10.7 years with range from 25-85 years. Majority (82.3%) patients were male and 390 (58.4%) patients were illiterate. Similar report Muhammad et al.[27] found mean age of the study population was 51.5±9.5 years and most (65.7%) of the patient were male.

In this study, among the risk factors, highest 267 (40.0%) patients had hypertension followed by 209 (31.3%) diabetes mellitus, 204 (30.5%) smoker, 189 (28.3%) H/O ischemic heart disease and 151 (22.6%) dyslipidemia. This findings were also consistent with others studies like Razban et al.[28]; Muhammad et al.[27] and Seyedian et al.[29].

From this study, we cannot infer, whether poor glycemic control leads to inflammation or whether inflammation leads to higher glucose levels (or whether a third factor influences both). Prospective studies are needed to evaluate that question. However, either direction of causality would have important implications. If poor glycemic control leads to inflammation, then better glycemic control should lower inflammation and therefore lower the risk of cardiovascular complications.

In this study positive correlation was found (r=0.220, p= 0.001) between HbA1c with CRP in CAD patients. Fawaz et al.[30] found their study a positive correlation of inflammatory marker (CRP) and HbA1c which supports other studies.[2,31] This can be explained by the fact that HbA1c reflects the biological activities of hyperglycemia and advanced glycation end products, all of which can induce inflammation.[32] Hyperglycaemia has an indirect influence on atherosclerosis through lipid changes. It increases potentially atherogenic forms of small VLDL and small dense LDL which are susceptible to glycation and oxidation. However, chronic hyperglycaemia may be a separate risk factor for accelerated macroangiopathy.[33] Roopakala et al.[34] reported that positive correlation (r=0.347, p= 0.008) of HbA1c with CRP in diabetic nephropathy.

Positive correlation coefficient between hemoglobin A1c and CRP levels in studied patients (r = 0.371, p=0.05).[35] Study done by Tutuncu et al.[36] on comparison of hs- CRP levels in new Diabetes groups observed a positive correlation between hs-CRP levels and age, BMI, waist, hip, SBP, DBP, pulse, FPG, HbA1c, TG, non-HDL cholesterol; and there was a negative correlation with HDL-cholesterol and eGFR. Wu et al.[17] reported that high levels of hs-CRP were correlated with high levels of HbA1c and FPG in men and with only FPG in women.

In this study, high CRP was found higher (60.5%) at ≥6.5 percent HbA1c level. The difference was statistically significant (p<0.05). King et al.[37] reported that elevated HbA(1c) levels (e”9.0%) had a significantly higher percent of elevated CRP than people with low (<7%) HbA(1c) levels (P <0.001).

Festa et al.[38] found links between CRP and insulin resistance. Other studies have related hyperglycemia to inflammation by demonstrating simultaneous inflammation, endothelial dysfunction, and insulin resistance at the physiologic level.[39,40] One of the several mechanisms proposed is oxidative stress on the endothelium, which promotes inflammation and is enhanced by hyperglycemia.[41-43] The current study demonstrates that higher HbA1c is significantly associated with elevation of CRP. These results imply a significant relation between inflammation and glycemic control in people with established CAD.

Multi variable logistic regression was found high HbA1c had 0.261(95% CI 0.025 to 0.882), high CRP had 30.222 (95% CI 8.847 to 99.389) and diabetes mellitus had 0.103 (95% CI 0.011 to 0.953) times increase in odds having severe CAD (Double and triple vessel). Which were statistically significant (p<0.05).

Therefore, detection of inflammatory marker and close observation of their glycemic control is essential to prevent cardiovascular complications. Early and effective prevention of cardiovascular disease will improve lifestyle with the emphasis on disease prevention.

Conclusion:

There is a positive correlation between serum levels of CRP and HbA1c in CAD patients. Thus, aiming at good glycemic control and estimation of serum CRP levels will possibly be of help in planning early intervention, thereby preventing further complications which in turn may help preserve cardiac functions in ischemic heart disease patients.

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Original Article

Safety of Longer Size Stent in Treating De-Novo Long Coronary Lesion: Outcome at 1.5 Years Follow-Up, A Single Center Experiences

AHM Waliul Islam[1], Shams Munwar[2], Azfar H. Bhuiyan[3], AQM Reza[2], Sahabuddin Talukder[2], Tamzeed Ahmed[2], Nighat Islam[4], Atique bin Siddique[4], Intekhab Yousuf[5], Zia Ur Rahman[5], M S Alam[3]

Abstract:

Background: Percutaneous coronary intervention (PCI) of long lesions by long single stent or overlapping multiple stent might have higher incidences of ISR due to increased metal burden as well as coronary intervention increase cost of hospital stay. Therefore, our primary aim of our study was to evaluate the long-term safety of treating long lesion by a single longer size stent and its follow-up by coronary angiogram and or clinical evaluation at our OPD.

Methods: patient who had gone through PCI from the year 2014 to mid Oct 2017 at our center, had longer lesion and were treated by more than 38mmstent were selected and analyzed. Total 255(Male 213: Female 42) patients were enrolled in this study, underwent elective PCI and follow up CAG at on average 1.5 yrs. Total 267 stents were deployed in 255 patients, in some of the patient had double vessel disease to treat. Mean age for both male: female was(55 :56) yrs. Associated Coronary artery disease (CAD) risk factors were Dyslipidemia, Hypertension, Diabetes Mellitus, Positive FH for CAD and Smoking (all male), CKD, Hypothyroidism.

Results: Among the study group; 192 (75.3 %) were hypertensive; 189(74.1%) were Dyslipidemic, 126(49.4%) patients were Diabetic, positive FH 74(29.4 %), CKD 8 (3.1%), Hypothyroidism 2 (0.8%) and 104(40.8%) were all male smoker. Common stented territory was, LAD 126(49.4%), RCA 115(45.1%), and LCX 24(9.4%).Among the total patient population, Single vessel stented were 236 (92.5%) and DVD 19 (7.5%). Total 267 stents were deployed, among them 48mm were in total 159 (59.6%); among 40 mm were stented in 61(22.8%) and 38 mm in 47(17.6%) vessels. At an average follow-up period of 1.5 years, all stented territory remain patent without any residual stenosis.

Conclusion: We conclude that treating de-novo coronary long lesion by a single longer size stent is safe without any residual stenosis at an average follow-up period of 1.5 yrs. Thus, to reduce chances of recurrent ISR, hospital re-admission and reduce hospital cost as well.

Key Wards: CAG, PTCA, PCI, DES, Long lesion, Stents

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Introduction:

Treating long segment coronary lesion is always a challenge for interventionist to deal with. Percutaneous Coronary Intervention (PCI) by implanting a stent inside a coronary artery, has been shown to decrease the morbidity of acute closure of the vessel.[1] Clinical and angiographic restenosis rates in selected lesions are reduced with coronary stenting as

1. Associate Consultant, Dept. of Invasive and Interventional Cardiology, Apollo Hospitals Dhaka.
2. Senior Consultant, Dept. of Invasive and Interventional Cardiology, Apollo Hospitals Dhaka.
3. Specialist, Dept. of Invasive and Interventional Cardiology, Apollo Hospitals Dhaka.
4. Senior Registrar, Dept. of Invasive and Interventional Cardiology, Apollo Hospitals Dhaka.
5. Registrar, Dept. of Invasive and Interventional Cardiology, Apollo Hospitals Dhaka.
Dept. of Invasive and Interventional Cardiology, Apollo Hospitals Dhaka

Address of Correspondence: Prof. Dr. AHM Waliul Islam, Interventional Cardiologist at Apollo Hospitals Dhaka. Cell: +8801713228884, E-mail: drwali62@hotmail.com

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compared with angioplasty. [2-3]Multiple or long coronary stents are now being implanted in long lesion or in tandem lesions.

Longer lesion usually need a longer segment to be covered by stents, and thus may require more than one stent. Both greater stented length and higher number of stents may exacerbate the risk of restenosis and mask direct relation lesion length and lumen narrowing after coronary stenting [4-5]Previously, treating long lesion by multiple overlapping stents has shown significant stent restenosis [6-8]

Treatment of long and diffuse coronary lesion have been associated with increased risk of restenosis after PCI. A higher angiographic restenosis of 58% reported after plain balloon angioplasty.[9]Although, the advent of bare metal stents was a breakthrough, was not successful in treating long coronary lesion. Implantation of multiple stents in treating long lesion resulted in diffuse in-stent restenosis. [10] With the advent of drug eluting stents in treating long segment coronary lesion, there has been dramatic reduction of ISR and repeat revascularization as compared to BMS.[11-12]

With the advent of modality of treating coronary stents of different DES, treating of a single long de-novo coronary lesion by using a single stent in our Bangladeshi patient population yet to known clearly. Therefore, we have carried out this non randomized prospective cohort of patient who had PCI with a stent> 38mm in length. Our primary aim of the study was to evaluate the long-term safety of treating long lesion by a single longer size stent and its follow-up by coronary angiogram and or clinical evaluation at our OPD.

Methods:

Patients who underwent PCI from the year 2014 to May 2018 at our center, had longer lesion and treated by a long stent of more than 38mmstent were selected and analyzed. Patient had baseline pre-PCI coronary angiogram either at our center or elsewhere. Based on QCA images and stented segment, lesions were divided into three group according to the length of the stented segment: Stented segment length 38mm, stented segment length 40mm and stented segment length 48mm. Total 255(Male 213: Female 42) patients were enrolled in this study, underwent elective PCI and follow up CAG at on average 1.5 years. Total 267 stents were deployed in 255 patients, in some of the patient had double vessel disease to treat. Lesions prepared by a low profile balloon, followed by stenting of the lesion. Further, post-dilatation was done by 3.0-3.5mm non-compliant balloon with 16-20ATM for better optimization of stent. Mean age for both male: female was (55 :56) yrs. Associated CAD risk factors were Dyslipidemia, High Blood pressure, Diabetes Mellitus, Positive FH for CAD and Smoking (all male), CKD, Hypothyroidism.

Long lesion: In the present study patient who were treated with stent from 38 mm onward were defined as long lesion. The procedure was considered successful with residual stenosis of <25% was left after stent placement. Death of any cause, myocardial infarction and Target lesion revascularization either by repeat percutaneous coronary intervention (PCI) or Coronary Artery Bypass Grafting (CABG) were considered as major adverse cardiac events. The diagnosis of MI was established in presence of chest pain, ECG changes of Q in 1 or more leads with raised CK-MB or Trop I. The follow-up protocol included phone contact or medical visit at the OPD or coronary angiogram. All patients were given informed consent for intervention and control CAG.

Drug Therapy

All the patients received Aspirin 300 mg and Clopidegrol as a loading dose 300 mg prior to CAG and PCI with or without Ticarel or Prasureland continued for 9-12 months and received atorvastatin along with standard medical management for CAD. During the procedure, an intravenous heparin bolus (100IU/Kg) and GP IIb/IIIa receptor blocker Integrillin were administered as required. The use of GP IIb/IIIa Receptor blocker was recommended as per protocol.

Stents:

Among the stent used; Sirolimus Eluting stent (Biotronik), Everolimus Eluting stent (Boston Scientific and Abbott vascular) and Taxus (Boston Scientific), Resolute Integrity (Medtronic)

Data: Data were presented as mean ± SD with percentage.

Results:

Table 1. shows demographic profile of Studied population. Female were older than male (Male 55: Female 56) yrs. Male are having more cardiovascular risk factors than female, as smokers were all male (Male 2.8: Female 2.5). Female were more obese than male(BMI male 26: Female 28). Table 2. Shows the contrast used and serum creatinine level in studied patient before and after the procedure. Average contrast uses in both sexes are 75ml and s. creatinine level were remaining almost identical in both pre and post procedure. Female has poorly controlled diabetes (Male vs Female:8.9 vs 10.7mmol/L). Table.3. Shows the territory wise the different size stent used in both male and female. Interestingly, it has been shown that in both sexes average vessel diameter in all three territories was less than 3mm in diameter. Figure 1. Shows the percentage distribution of coronary stents according to territory.Figure 2. shows the stented territory, LAD 46%, RCA 45%, LCX 9%. Figure 3. Shows the distribution stents according its length in mm. Figure 4. Shows the of CAD risk factors. 189(74.1%) were Dyslipidemia, 192 (75.3 %) were hypertensive; 126 (49.4%) patients were Diabetic, positive FH 74 (29.4 %), CKD 8 (3.1%), Hypothyroidism 2 (0.8%) and 104 (40.8 %) were all male smoker.Figure 5. shows the stenting of LAD with along 2.75 x 48 mm stent. Figure 6. Shows the stent patency after 1.5 yrs.

Table-I
Profile of patient

	Male	Female
Number	39	9
Age (yrs)	55.2±10.0	56.4±9.4
BMI(kg/m^2)	25.9 ± 2.3	27.9±3.5
SBP(mmHg)	131±18	133±18
DBP(mmHg)	79.0±9.1	79±12
No Risk Factor	2.8±1.0	2.5±0.8

Data were presented as Mean ± SD

Table-II
Contrast used and S. Creatinine level

	Male	Female
Contrast in ml	76.1±12.4	75.8±10.7
S.Creatinine(pre)	1.25±0.4	1.18±0.34
S. Creatinine (post)	1.24±0.3	1.1±0.21
RBS(mmol/L)	8.9±4.1	10.7±2.8
HbA1C	6.95±4.1	7.4±6.6

Data were presented as Mean ± SD

Table-III
Average size of Stent used with inflation pressure

		Length (mm)			Diameter(mm)			Inflation Pressure (ATM)		
LAD	M	43.5	±	0.3	2.8	±	0.3	16.0	±	1.9
	F	44..0		4.9	2.7		0.3	15.4		1.7
RCA	M	43.7	±	4.7	2.96	±	0.4	15.7	±	1.6
	F	43.7		4.4	2.85		0.3	15.4		1.7
LCX	M	43.4	±	4.5	2.64	±	0.2	15.2	±	1.7
	F	42.0		5.3	2.67		0.1	16.0		1.0

Data were presented as Mean ± SD

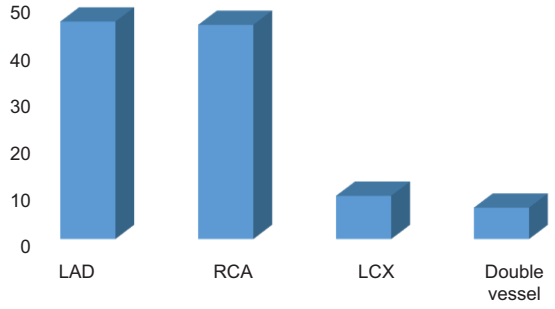

Fig.-1. *Percentage of distribution of Coronary Stent*

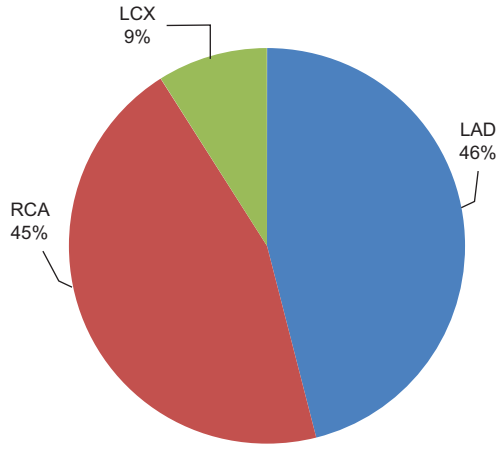

Fig.-2: *Percentage of distribution of Stented territory*

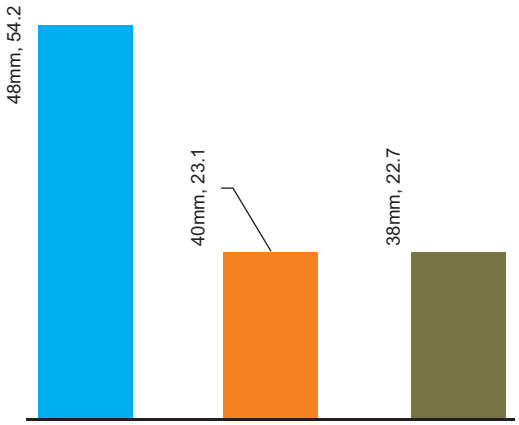

Fig.-3: *Percentage distribution of Stent according to size*

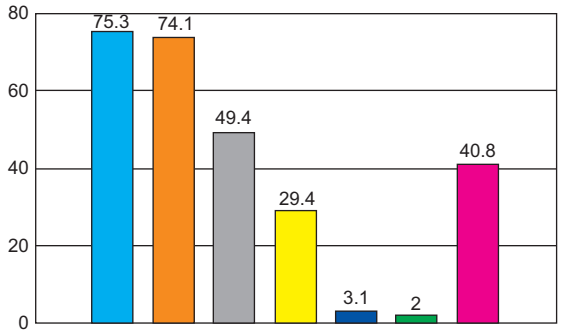

Fig.-4: *Percentage of Distribution of CAD Risk Factors*

Stent positioning *Post PCI*

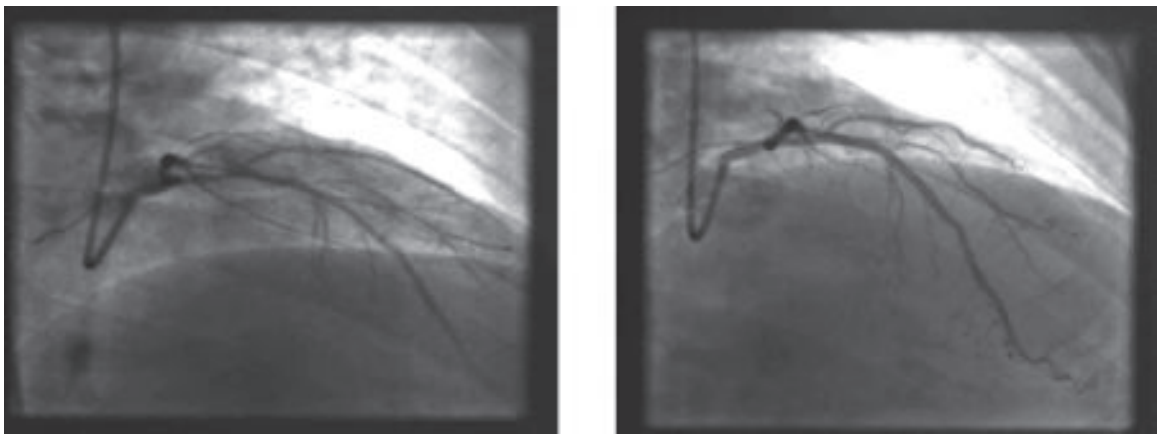

Fig.-5: *Shows PCI of LAD lesion with 2.75 x 48 mm Stent*

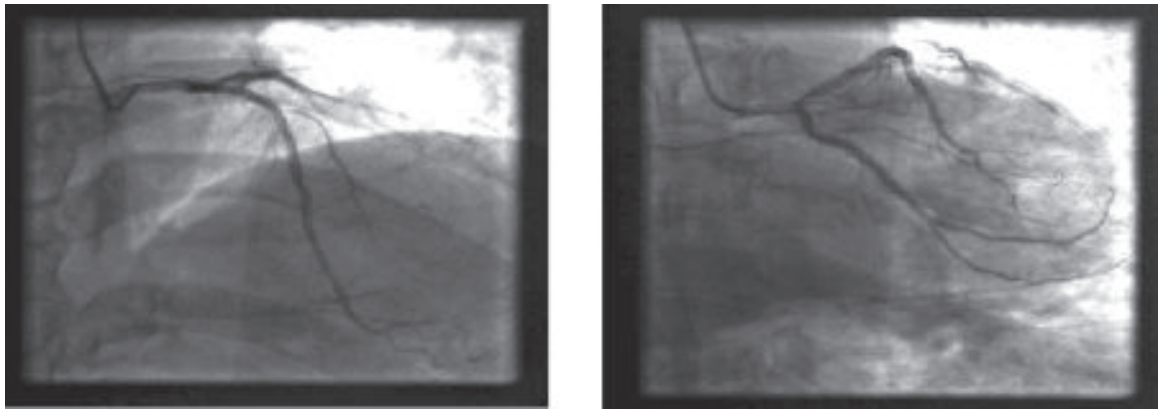

Fig.-6: *Shows Patent LAD and LCX stent after 16 month*

Discussion:

In the era of Percutaneous coronary intervention(PCI) in treating long segment lesion, itself is an important predictor determinant of restenosis. Nonrandomized studies have indicated an increased risk of restenosis after conventional PCI.[13] Multiple or long coronary stents are now being used to treat long lesion or in tandem lesions and shown to have higher restenosis. [6-7]

In this current prospective cohort study, we try to find the stent patency and major adverse cardiac events (MACES) that is stent thrombosis, MI or death after treating long-segment lesion by putting a long stent. In our study, females are more obese than male with poorly controlled diabetes possibly due to lack of exercise or non-compliance to medicine or ignorance. The number of CAD risk factors were more in male than female, possible due to smoking as additive factor in male. This is in favor that suggested, both male and female patients might have different CAD risk factors that trigger the development of coronary artery disease. Interestingly, the average vessel size in all three territories in both male and female were less than 3mm in diameter. In general, we are treating small size vessel in our population where the chances of development of in-stent restenosis is high.[14] Also, the post PCI, Serum Creatinine level didn't change much than the pre PCI Serum Creatinine level, possibly due to controlled uses of ionic contrast uses amount during the entire procedure to keep as much low as possible.[15]

This is the first time; we have carried out this non randomized single center prospective cohort of patients underwent PCI for their occluded coronaries by a long single stent to treat the de novo long lesion. Previously, Islam et al,[16] demonstrated treating a long segment lesion by multiple overlapping stents; where Sirolimus Eluting Stents (Cypher)

showed reduce ISR than other DES. Since, ISR is one of the important drawback in maintaining the integrity of stent patency and thus patient's clinical improvement. So, we designed to treat long de novo lesion at our center by putting a single stents > 38mm in length. We used three different stent size of 38mm, 40mm and 48 mm. We did not find ISR at 1.5 years after PCI in this patient subset. Even though the average vessel diameter is less than 3mm, which is very common in this Asian population.

It is well known that dramatic advances in treating clogged coronary artery to open and keep its patency, thus to reduce myocardial damage either by PCI,using bare metal stents or different drug eluting stents, leads to reduction CABG.[13]It has been shown thatpercutaneous coronary intervention by stenting over plain PTCA has clear advantages in terms of restenosis, and restenosis driven events for an increasing number of indications.[2-3] In addition, the administration of DAPT for given a given times, has dramatically reduces the development of stent thrombosis or subsequent ISR. [16-18]

Long lesion and long stent are considered as important predictors of restenosis after PCI with Bare metal stent (BMS) or Drug eluting stent (DES).DES have consistently shown to reduce restenosis, need for target lesion revascularization or MACE over the Bare metal stent (BMS). A number of nonrandomized studies have indicated an increased likelihood of restenosis after coronary stent implantation in treating long lesion.[19-20] Longer lesion needs a longer stent to cover the lesion. This increase length may require the placement of >1 stent and may exacerbate the risk of restenosis,mask the direct relationbetween lesion length and lumen narrowing after coronary stenting.

The reason why the implantation of more stents causing more restenosis irrespective of lesion length and stented segment is not known. One possible explanation, is that are difficult to avoid unless intentionally overlapping the stents. Tissue prolapse may occur between stents in same as described for the articulation site of Palamz-schatz stents,[21] has suggested spot stenting seems to be preferable over full jacket for PCI in long lesion. As stented length increases the chances of restenosis and stent thrombosis, since metal, polymer and drug disrupt the intimate morphology and physiology.Adnan kastrati et al has suggested in treating long lesion by a single long stent is favorable over multiple shorts stent.[22]

In our study, female patients are more obese, poorly controlled diabetes, developed CAD in advance age than male patients. Although, in our present study, average stent diameter is less than 3mm in both male and female patients. Therefore, long lesion and small vessel diameter in addition to poorly controlled diabetes, specially in female and smoking in male (as all smoker) might be one of the important determinant in stent patency specially in long lesion.

In this study, we put all drug eluting stent in treating long lesion. Follow up CAG has shown no ISR at 1.5 years after PCI which is very much consistent with described else.[23] Kereiakes et al[24]showed that in the BMS group long lesion has the higher rate of ISR, similarly stent length and lesion length is an independent predictor of ISR in various DES.[19,25]

Conclusion:

Treating long segment coronary lesion, has some drawbacks due to the possibility of development of ISR. Previously, PCI by putting multiple overlapping stent was one of the important modality in treating long coronary lesion. But, the development or risk of possible in-stent restenosis, many has changes to one stent strategy to treat long lesion. With the advent of different DES in different length size and availability of IVUS, the ISR rate has come down. In this perspective non randomized single center cohort, we found treating long de novo coronary lesion by a single long drug eluting stent is safe, without any ISR at 1.5 year follow up in ourpatient population. Thus, to reduce hospital re-admission and reduce hospital cost as well.

Future perspective:

Our future plan is to enroll more patient to do long-term follow-up, to see stent patency and MACE in terms of ISR, MI and death, and hence, if possible to enroll and compare multicenter involvement.

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Original Article

Predictors of Short Term Outcomes of Primary Percutaneous Coronary Intervention

Farhana Ahmed[1], Afzalur Rahman[1],Mohammad Arifur Rahman[1], Tariq Ahmed Chowdhury[1], Md. Shahabul Huda Chowdhury[2], Syed Nasir Uddin[1], AKM Monwarul Islam[1], Mohsin Ahmed[1]

Abstract:

Background: Acute myocardial infarction (AMI) is one of the leading causes of death and disability all over the world. Primary percutaneous coronary intervention (PCI) is the treatment of choice for patients with acute ST segment elevation myocardial infarction (STEMI). Primary PCI is being increasingly done in our country also. But the factor influencing the outcome of primary PCI in our setting are mostly unknown. The present study was conducted to investigate factors that influencing the short term outcomes of primary PCI.

Materials and methods: This prospective observational study was conducted from September 2014 to January 2016in the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka. 48 patients were selected by purposive sampling. Patients with acute STEMI treated with primary PCI were included in the study based on inclusion and exclusion criteria. Effect of factors including advanced age, male sex, diabetes mellitus, hypertension, dyslipidemia, serum creatinine, left ventricular ejection fraction, anterior myocardial infarction (MI), thrombolysis in myocardial infarction (TIMI) flow, multi vessel disease, angiographic severity score (Leaman score), thrombus aspiration, door to balloon time and total ischemic time on major adverse cardiac events (MACE) i.e. death, post procedural MI, target vessel revascularization (TVR), stroke as well as, on other adverse events like heart failure, cardiogenic shock, major bleeding, significant arrhythmia and stent thrombosis were studied.

Results: The overall incidence of MACE was 2.1%, major bleeding 2.1%, heart failure 4.2% and cardiogenic shock 2.1%. In multivariate analysis, the factors independently influencing the adverse short term outcomes (MACE and other adverse events) were diabetes mellitus (odds ratio (OR) 2.55, 95% confidence interval (CI) 1.180 to 4.124, p=0.02), anterior MI (OR 1.48, 95% CI 1.020 to 1.926, p=0.04), total ischaemic time (OR 1.49, 95% CI 1.044 to 2.444, p=0.04), multivessel coronary artery disease (OR 1.77, 95% CI 1.26 to 3.261, p=0.03) and Leaman score (OR 2.5, 95% CI 1.100-4.504, p=.03).

Conclusion: According to our finding, diabetes mellitus, anterior myocardial infarction, total ischemic time, multivessel coronary artery disease and high Leaman score are predictors of adverse short term outcomes of primary PCI.

Key word: Percutaneous Coronary Intervention, Primary, STEMI, Short Term Outcomes.

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Introduction:

Coronary artery disease (CAD) is the most common form of heart disease and single most important cause of premature death in most part of the world. Acute myocardial infarction (AMI) is one of the leading causes of death and disability. It generally occurs due to sudden occlusion of a coronary artery by formation of thrombus at the site of fissured or ruptured atherosclerotic plaque.[1]The major aspect of treatment of ST elevated Myocardial Infarction (STEMI) is reperfusion of the infarct related artery.

1. National Institute of Cardiovascular Diseases, Dhaka, Bangladesh
2. Government Employee Hospital, Dhaka , Bangladesh.

Address of Correspondence: Farhana Ahmed, Registrar, Dept. of Cardiology ,National Institute of Cardiovascular Diseases, Dhaka, Bangladesh, Mobile- +8801712282059, Email- nipaariha79@gmail.com

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Reperfusion therapy aims at restoration of antegrade flow in the occluded infarct related artery, which reduce infarct size and improves clinical outcome.[2]Early, effective and sustained reperfusion of the culprit artery is needed to salvage myocardium, maintain left ventricular function, and reduce mortality.

Fibrinolysis and primary PCI are the two options for the patient presenting with STEMI. If high-quality PCI is available, multiple randomized trials have shown enhanced survival compared to fibrinolysis with a lower rate of intracranial hemorrhage and recurrent myocardial infarction (MI).[3]

Outcomes after primary PCI are variable and accurate risk stratification is the clinical importance in guiding the management of relatively high risk patient. Some studies have shown that mortality rates are higher among women than in men.[4] Moreover, some studies have identified high age as a predictor of major adverse cardiac events (MACE) after primary PCI for myocardial infarction.[5] Patients with diabetes who receive primary PCI for STEMI are also at higher risk of mortality especially during hospitalization and the first year following the procedure.[6] In patients with myocardial infarction, high lipoprotein (a) levels have been found to be associated with adverse long-term result.[7]

Finally, shorter interval between the onset of myocardial infarction symptoms and primary PCI will lead to better result. The most favorable interval has been determined as 90 minutes.[8] Diabetes mellitus, poor post-interventional flow in the coronary arteries, three vessels disease, cardiogenic shock, and infarct localization appear to be important factors impacting the outcome in patients with STEMI undergoing primary PCI. Primary PCI is being increasingly done successfully in our country also. No such study done so far in our population to determine factors influence the outcomes of primary PCI. The present study was conducted to determine factors that influence the outcomes of patients who underwent primary PCI due to acute myocardial infarction.

Study Methods:

This prospective observational study conducted in the department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), from September 2014 to January 2016. Objective of the study was to find out the predictors of short term outcomes of primary PCI among Bangladeshi population presented with acute ST segment elevation myocardial infarction (STEMI). Total 48 patients with acute STEMI presented within 12 hours of onset of typical chest pain were included in the study purposively. Patients who received fibrinolytic therapy, having old MI, LBBB, valvular heart disease, cardiomyopathies, renal failure, high bleeding risk, stroke, malignancy were excluded from the study.

All patients were subjected to a thorough assessment of history with a focus on demographic data, analysis of chest pain, including timing variable, risk factors of coronary artery disease, drug history. Physical examination was performed including vital signs and evidence of heart failure (S_3 gallop, pulmonary rales, elevated JVP). ECG was done immediately. STEMI was diagnosed by new ST elevation at the J point in at least 2 contiguous leads of ≥2 mm (0.2 mV) in men or ≥1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥1 mm (0.1 mV) in other contiguous chest leads or the limb leads. Loading dose of aspirin and clopidogrel was given immediately after diagnosis of acute STEMI. Blood sugar was checked immediately by glucometre and managed accordingly. Blood sample send for lipid profile, troponin-I, serum creatinine before coronary angiogram; left ventricular function was assessed by echocardiography by Teichholz method before coronary angiogram.

Immediately coronary angiography was done by femoral route to identify the culprit lesion, thrombus burden, other vessels involvement; severity of coronary artery disease were also assessed by vessel score and Leaman score. Coronary flow in the infarct related artery was assessed visually by the operator and classified according to the TIMI grading system on a scale of 0 to III. During primary PCI, pre-dilatation of lesion and thrombus aspiration was performed by aspiration catheter in selected cases, then PTCA (percutaneous transluminal coronary angioplasty) with stenting was done and post procedural TIMI flow was assessed and post-dilatation was done if necessary. Successful PCI was defined as visually assessed <20% residual stenosis with TIMI III distal flow and absence of major clinical complications (death, AMI and emergency myocardial revascularization). During hospital stay, patients were examined daily to find out any major complications following PCI; troponin-I and serum creatinine were repeated 6 hours after PCI; left ventricular function following PCI was assessed by echocardiography before discharge. All patients were asked for follow up within one month after primary PCI. In each follow up following parameter were seen: haemodynamic (pulse, blood pressure), ECG (ST change) and complications including MACE (death, Post procedural Myocardial Infarction and the need for repeat revascularization, Stroke) and other adverse events including major bleeding (Major bleeding defined as either intracranial bleeding or overt bleeding with a decrease in hemoglobin ≥5 g/dl),heart failure, cardiogenic shock, significant arrhythmia. Follow up evaluation was done by telephone interview for those who could not attend directly and all

parameters were recorded. The primary end point was in-hospital MACE andother adverse events and secondary end point included 30 days outcome from discharge including MACE and other adverse events.

Statistical Methods

All variables were entered into the Statistical Package for Social Sciences, version 16 (*SPSS Inc., Chicago, Illinois*).Data was presented as frequency and percents for categorical variables and as mean with standard deviation for quantitative variables. Univariate and multivariate regression analysis were done with variables may be related to adverse outcome with calculated risk ratios odds ratios [OR] for independent variables with 95% confidence intervals [CI]. P value <0.05 were considered as significant.

Results:

This prospective observational study was conducted in NICVD, Dhaka starting from September 2014 to January 2016. A total of 48 patients with acute STEMI who had fulfilled the inclusion and exclusion criteria were included in the study.

The mean age of this study group was found 47.9 ± 6.5 years and the age limit was 37 to 65 years (Fig-1); 85% study population were male and remaining 15% were female

(Fig-2). Smoking was the most prevalent risk factor affecting 75.0%. Table-1 shows that hypertension, diabetes mellitus,dyslipidemia was found in 41.7%, 45.8%, 54.2% study population respectively; along with chest pain 16.8% and 22.9% patients were presented with shortness of breath and palpitation; the mean systolic blood pressure of study population was 112.5 ± 23.6 mmHg and mean diastolic blood pressure was 69.6 ± 14.3 mmHg; JVP was raised in 4.2% patients on admission; heart failure and arrhythmia were present in 4.2% patients each, mean pain to door time was 4.6 ± 1.8 hrs, door to balloon time was 1.9 ± .9 hrs, total ischemic time was 6.8 ± 2.1 hrs.

Anterior infarction was found in 47.9% patient and rest were presented with inferior MI. Coronary angiogram revealed 2.1% patients had LM disease; LAD and RCA were involved in 33.3% patients each. LCX involved in 4.2% cases, both LAD and LCX were involved in 10.4% patients. LAD and RCA, LCX and RCA were involved in 4.2% patients each. Triple vessel (LAD, LCX and RCA)were involved in 8.3% patients. Fig-3 shows that single vessel involvement was found in 72.9% followed by double vessel 18.8% and triple vessel 8.3% among the study population (n=48).Mean lesion length was found 23.1 ± 9.1 mm.PCI to non culprit lesion was done in 4.2% cases, thrombus aspiration and predilatation were done in 33.3% and 12.5% cases respectively.

Mean Leaman score of study population was 11.4 ± 5.9.Fig-4 shows that drug eluting stent were used in 72.9% of cases and bare metal stent were used in 27.1% of cases.Before PCI 95.8% patients had TIMI 0 and 4.2% patients had TIMI I flow.On the contrary, in post-PCI TIMI III flow resumed in 93.8% cases and 6.2% patients had TIMI II flow

(Fig-5). Table-2 shows adverse in-hospital and 1 month outcomes of the study population (n=48) where it was found that death, heart failure, major bleeding and cardiogenic shock developed in 2.1%, 4.2%, 2.1% and 2.1% cases respectively. Binary logistic regression analysis of odds ratio for characteristics of the population likely to develop adverse outcomes of primary PCI. In regression analysis (Table-3) diabetes mellitus, anterior MI, total ischemic time, multivessel disease and Leaman score were found to be the independent predictors for developing adverse short term outcomes of primary PCI.

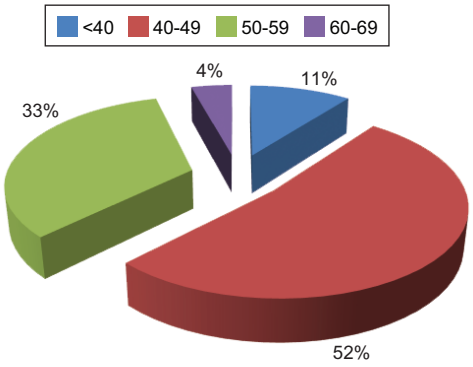

Fig.-1: *Age distribution of study population (n=48)*

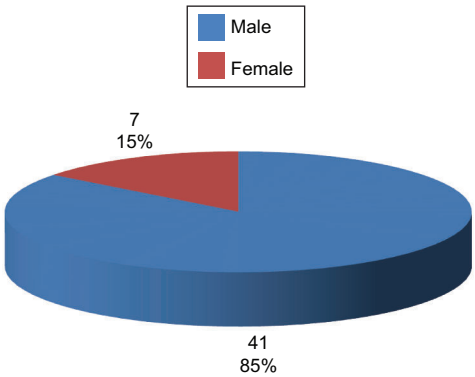

Fig.-2*: Sex distribution of study population (n=48)*

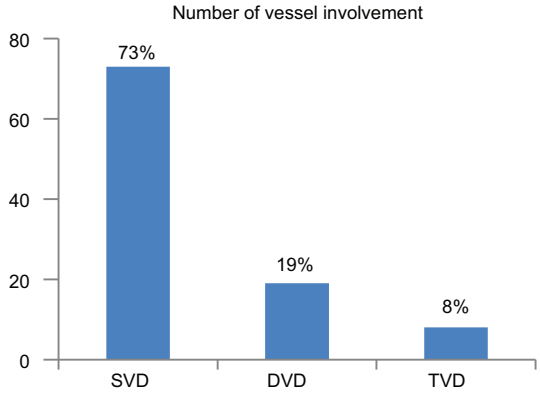

Fig.-3: *Distribution of study population according to number of vessel involvement*

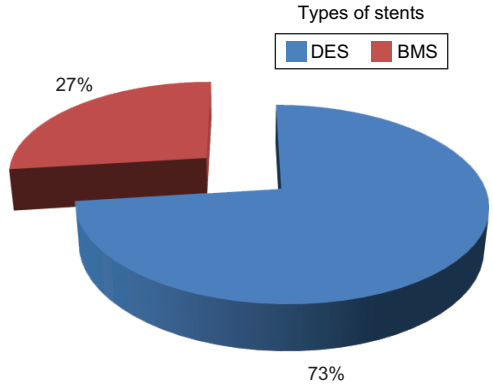

Fig.-4: *Distribution according to type of stent used in study population (n=48)*

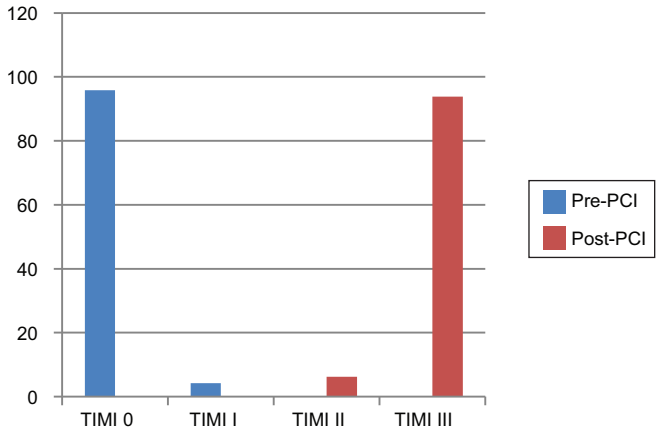

Fig.-5: *Procedural outcomes of the study population according to TIMI flow (n=48)*

Table-I

Baseline characteristics of study population

Body Mass Index (kg/m^2)	Number	Percentage (%)
Normal (18.5-24.9)	10	20.8
Overweight (25-29.9)	22	45.8
Obese (30 – 39.9)	16	33.33
Mean ± SDRange (min – max)	21.4 ± 1.7 (22.3 – 30.4)	
Risk factors	Number	Percentage (%)
Smoking	36	75.0
Hypertension	20	41.7
Diabetes mellitus	22	45.8
Dyslipidemia	26	54.2
Serum Creatinine (mg/dl)	1.0 ± 0.23	
Duration of chest pain	Duration (hrs) Mean ± SD	
Pain to door time	4.6 ± 1.8	
Door to balloon time	1.9 ± 0.9	
Total chest pain duration (Total ischemic time)	6.8 ± 2.1	

Table-II

Adverse in-hospital and 1 month outcomes of the study population (n=48)

Outcomes	In hospital Number (%)	1 month Number (%)	Total Number (%)
MACE	1 (2.1)		
Death	1 (2.1)	0	1 (2.1)
MI	0	0	0
TVR	0	0	0
Stroke	0	0	0
Other adverse events			
Major bleeding	1 (2.1)	0	1 (2.1)
Heart failure	1 (2.1)	1 (2.1)	2 (4.2)
Cardiogenic shock	1 (2.1)	0	1 (2.1)
Significant Arrhythmia	0	0	0
Stent thrombosis	0	0	0

Table-III

Factors related to adverse short term outcomes of primary PCI

Variables of interest	Univariate analysis			Multivariate analysis		
	OR	95% CI of OR	p value	OR	95% CI of OR	p value
Age (>50 years)	1.89	0.797-3.538	0.17^{ns}	1.59	0.699-3.443	0.16^{ns}
Male gender	1.02	0.989-1.431	0.36 ^{ns}	1.00	0.987-1.132	0.42^{ns}
Current smoking	1.58	0.797-3.538	0.16^{ns}	1.55	0.699-3.443	0.26^{ns}
Diabetes mellitus	3.04	1.190-4.931	0.01^{s}	2.55	1.180 – 4.124	0.02^{s}
Hypertension	1.11	0.202-2.401	0.21^{ns}	0.91	0.107-2.349	0.17^{ns}
Dyslipidaemia	1.24	0.404-2.530	0.14^{ns}	1.19	0.301-2.429	0.11^{ns}
Serum creatinine	0.86	0.120-2.549	0.30 ^{ns}	0.77	0.109-2.409	0.39^{ns}
Anterior MI	1.96	1.050-2.599	0.03^{s}	1.48	1.020-1.926	0.04^{s}
LVEF	1.97	0.594-1.789	0.14 ^{ns}	1.11	0.449-1.680	0.20 ^{ns}
Total ischemic time	1.92	1.089-2.779	0.03^{s}	1.49	1.044-2.444	0.04^{s}
Door to balloon time	0.97	0.080-1.608	0.40^{ns}	0.86	0.050-1.480	0.46^{ns}
Multivessel disease	2.01	1.142-3.144	0.02^{s}	1.77	1.26-3.261	0.03^{s}
Pre-TIMI (0)	1.24	0.624-3.540	0.11^{ns}	1.00	0.509-2.780	0.15^{ns}
Leaman score	2.78	1.201-5.404	0.01^{s}	2.50	1.100-4.504	0.03^{s}
Thrombus aspiration	0.97	0.090-1.799	0.25^{ns}	0.86	0.040-1.690	0.22^{ns}

Discussion:

Coronary artery disease is one of the most important causes of death worldwide. However, PCI represents one of the cornerstone management modalities for patients with coronary artery disease. To date, data are lacking on the factors that modify the outcomes of primary PCI in Bangladesh.This study sought to identify factors affecting major adverse cardiac outcomes in patients who underwent primary PCI for STEMI.

In our study, mean age of patient with acute STEMI was 47.9 ± 6.5 years and most of the patients were between 40 to 49 years instead of old age group as seen in Western communities.[9] In our observation, the incidence of MACE were not related with age, increased age generally increase the mortality in patient with myocardial Infarction[10], Since we have lacking patient of very old age group, so this result is justifiable.

Most of our patients were male (85%) which reflect the current scenario that coronary artery disease is more common in man.[9] Though most of our patients were men, we found no significant effect of sex on major adverse cardiac outcomes which is similar to a study done

previously.[11] However, some studies have found higher mortality rates in women than in men.[4] The absence of such a higher risk among our female participants emphasizes the benefits of primary PCI in women.

Smoking is a well known risk factor for coronary artery disease. Most of our study population (75%) were smoker. The importance of smoking in the incidence of major adverse cardiac outcomes after primary PCI is a matter of concern. In this study, smoking did not have any effect on MACE. However Both American College of Cardiology and European Society of Cardiology STEMI guidelines strongly encourage the patient and their family to stop smoking and to avoid second hand smoke.[12]

According to global registry for acute coronary events, approximately one in four patients presented with acute coronary syndrome has history of diabetes mellitus[13]. We found a significant number of our study population (45%) were diabetic and diabetes mellitus was found to be a factor that is responsible for adverse short term outcome after primary PCI.

A very recent study report[14]also described that patients with diabetes mellitus had a worse outcome after primary PCI than non diabetic patients. Various studies have also highlighted the short-term and long-term effects of diabetes on the MACE.[6] Thus diabetes mellitus can be considered as a risk factor for major adverse cardiac outcomes after primary PCI and should give emphasize on good diabetic control for prevention of macro and micro vascular complication and post PCI adverse outcome.

High arterial blood pressure is a risk factor for coronary artery disease and increases the risk of complications after acute coronary syndrome.[15] DESERT database[16] showed that among STEMI patients undergoing primary angioplasty HTN is independently associated with impaired epicardial reperfusion, mortality, re-infarction, TVR and stent thrombosis. In our observation we did not find any significance of hypertension on MACE in patient with primary PCI.

Inter-heart study[17] suggest that 45% heart attacks in Western Europe are due to abnormal blood lipid. We found 45.8% patient with myocardial infarction have dyslipidemia in our study population; a similar study[18] done in India also found similar incidence 43.6%. Despite the fact that hyperlipidemia is a risk factor for coronary artery disease, we did not find any significant effect on the incidence of major adverse cardiac outcomes in this study.

Better left ventricular function is associated with improved survival. On the other hand, low LVEF leads to higher incidence of MACE.[19]Although the incidence of heart failure after myocardial infarction has fallen over the last few decades, still it complicating up to 45% of infarcts. Though in a current study, low LVEF had statistically significant effect on MACE in patients undergoing primary PCI;[19] we did not find any statistically significant correlation of LVEF on MACE after primary PCI as because of our small number of study population.

Total ischemic time is an important determinant of quality of care. Recommended time as per American College of Cardiology (ACC)/ American Heart Association (AHA) guidelines[20] is 90 minutes door to balloon time. However achieving this time is possible only in ideal world scenario. In our study, the median door to balloon time was 1.9 ± .9 hours. However if we look at recent study conducted in China, the median door to balloon time reported for primary PCI was 132 min.[21] In our study mean of total ischemic time is 6.8 ± 2.1 hours. A similar study[22] describes better outcome of primary PCI in patients with total ischemic time d”5 hours than in those with longer ischemic time. Treatment within these golden hours of primary PCI results in better myocardial reperfusion and clinical outcomes. Our findings also confirm that prolonged total ischemic time is associated with adverse outcome after PCI.

Anterior myocardial infarction carries the worst prognosis of all infarct locations, mostly due to larger infarct size. In our study, 47.9% patient were presented with anterior myocardial infarction and rest of the patient with inferior myocardial infarction. A study comparing outcomes from anterior and inferior infarctions found that on an average, patients with anterior MI had higher incidences of in-hospital mortality (11.9% vs. 2.8%), total mortality (27% vs. 11%), heart failure (41% vs. 15%) and significant ventricular ectopic activity (70% vs. 59%) and a lower ejection fraction on admission (38% vs. 55%) compared to patients with inferior MI. [23] In our study, we also observed that anterior MI had significant (OR 1.48, p value 0.04) influence on major adverse cardiac outcomes after primary PCI.

In our observation we found 8.3% patient with TVD and 18.8% patient with DVD. Multi vessel involvement has significant (OR 1.77, p=.03) impact on MACE after primary PCI which is consistent with a study finding where they also found an increased risk of MACE in presence of multi vessel coronary artery disease.[9] In the CADILLAC trialcumulative incidence of death of patients with single, double and triple-vessel disease was 3.2%, 4.4% and 7.8%, respectively (p=0.003), and the composite rate of major adverse cardiac events (MACE) was 14.8%, 19.5% and 23.6% respectively (p= 0.0006).[24] In our study, angiographic severity was assessed by Leaman score and higher score was found to have bad prognostic effect on MACE.

The use of thrombus aspiration during primary PCI has remained controversial. During our procedure we did manual thrombus aspiration in 33.3% cases where there were high thrombus burden and we found no significance impact of thrombus aspiration on outcome of procedure which also support the recent recommendation[20] regarding thrombus aspiration.

Among 48 primary PCI procedure most of the cases (95.8%) we did only culprit lesion PCI, only in few cases (4.2%)we performed non culprit lesion PCI also. Multivessel PCI done in a case where patient presented with AMI anterior with heart failure and in another cases of acute inferior MI where there were difficulties of identification of culprit lesion, both LCX and RCA revascularization done simultaneously. There were no statistically significant relation observed on short term outcomes in our study, though PRAMI [25] trial showed favorable result of non culprit lesion PCI beside culprit lesion PCI in multivessel disease.

Good TIMI flow at the time of angiography and PCI is a determinant of MACE in patients undergoing primary PCI. In our study, most of the patient (93.8%) achieved TIMI III flow with excellent procedural success rate after percutaneous coronary intervention. During the time of procedure eight patients developed no-flow phenomenon, after taking appropriate measures TIMI III flow established in five patients and three patients (6.2%) developed TIMI II flow. Patients with TIMI III flow are expected to have higher survival rates and fewer complications following primary PCI.[26]

Our study showed death rate was only 2.1%; result is consistent with study done in India[27] and from Pakistan[28] where in hospital mortality was 2.2% and 1% respectively. In our country previous single study conductedhad shown in hospital survival rate only 93.8%.[29] In our study population one patient died 3 days after procedure had multiple risk factor including uncontrolled diabetes, hypertension and dyslipidemia,multivessel disease, immediate post procedural period was uneventful, sudden death occur on third day due to ventricular fibrillation.

In our study, among 48 cases in-hospital period death occurred in 2.1% cases, 2.1% patient developed heart failure, 2.1% patient develop cardiogenic shock. Incidence of major bleeding in our study was 2.1% whereas another study[30] found the incidence of major bleeding is less than 2%. In our study population nobody developed significant arrhythmia though 4.2% patient developed arrhythmia in the form of ventricular tachycardia, atrial fibrillation immediately after procedure which terminate spontaneously. The frequency of these arrhythmias after primary PCI was analyzed in Primary Angioplasty in Myocardial Infarction (PAMI) trials.[31]Within one month period among 48 patient 1 patient took readmission with heart failure all other patients were symptoms free.

Conclusion:

The study revealed that diabetes mellitus, anterior myocardial infarction, prolonged total ischemic time, multivessel coronary artery disease and high Leaman score of coronary artery disease were predictors of adverse short term outcomes after primary PCI in Bangladeshi population.

Recommendation:

Primary PCI should be encouraged as a management of acute STEMI. Large scale, multicentre study should be done to evaluate the factors which influence the short term outcomes of primary PCI. Diabetic patient should need special attention during primary PCI and post PCI period. Multivessel coronary artery disease patient need individualized management strategy.

Conflict of interest - none.

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Original Article

Impact of SYNTAX Score on In-hospital Outcome after Primary Percutaneous Coronary Intervention

Md. Shariful Islam[1], Md. Afzalur Rahman[2], Abdul Wadud Chowdhury[3], Sayed Nasir Uddin[4], Nupur Kar[5], Kajal Kumar Karmakar[5], Mohammad Ullah Firoze[6], Mohammad Arifur Rahman[7] , Monir Hossen Khan[8], Md. Nure Alam Ashrafi[1], Muhammad Ruhul Amin[9], Md Minhaj Arefin[1], Fathima Aaysha Cader[10]

Abstract:

Background: Limited contemporary data exist regarding the impact of SYNTAX score on in-hospital outcomes undergoing primary percutaneous coronary intervention(PCI) in acute STEMI patients.

Objectives: To evaluate the significance of the SYNTAX score for predicting in- hospital outcome after primary PCI in patient with acute STEMI.

Methods: This cohort study was conducted in the department of cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh from September, 2015 to September, 2016. 42 patients with acute STEMI who underwent primary PCI were considered for the study. But 2 patients were excluded from the study due to failure of primary PCI. The patients were divided into two groups: Group I (low SYNTAX score d"22) and Group II (high Syntax score > 22). The Syntax score of all patients were calculated from an initial coronary angiogram before primary PCI. In-hospital outcome was observed in between two groups.

Results: Among traditional cardiovascular risk factors diabetes was significantly more prevalent in the Group II than Group I (82.4% vs 34.8%, p Â 0.003). Angiographic profile revealed maximum (69.6% vs 17.6%) culprit lesion in LAD artery in Group I and maximum culprit lesion (64.7% vs 21.7%) in RCA in Group II, these were the statistically significant between Group I and Group II (P<0.05). The high SYNTAX score group had lower TIMI 3 (76.47% vs 91.3%, p Â 0.03) compared to the low SYNTAX score group. But there were no significant difference in complications as arrhythmia (2.5% vs 0%), cardiogenic shock (2.5% vs 0%), heart failure (5% vs 2.5%) and mortality (5%vs 0%) between high and low SYNTAX score. Multivariate logistic regression analysis revealed SYNTAX score (OR = 5.95, p Â 0.001) was an independent predictor of in-hospital outcome in patients under going primary PCI. Performance test of SYNTEX score in the setting of Primary PCI outcome showed positive predictive value 83%. Conclusions: SYNTAX score was an independent variable that can predict in-hospital outcomes of patients with acute STEMI undergoing primary PCI.

Key wards : SYNTAX score, Primary PCI, STEMI

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1. Assistant Registrar, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka
2. Professor and Director, National Institute of Cardiovascular Diseases, Dhaka
3. Professor and Head, Department of Cardiology, Dhaka Medical College, Dhaka
4. Professor, National Institute of Cardiovascular Diseases, Dhaka
5. Associate Professor, Department of Cardiology National Institute of Cardiovascular Diseases, Dhaka
6. Associate Professor, Department of Cardiology, Manikganj Medical College, Manikganj
7. Junior Consultant, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka
8. Registrar, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka
9. Emergency medical officer, Shahid Ziaur Rahman Medical College Hospital, Bogra
10. Assistant Registrar, Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka.

Address of Correspondence: Dr Md Shariful Islam, Assistant Registrar, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka., Mobile: +8801716424258, Email: mistyratan@gmail.com.

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Introduction:

Cardiovascular diseases account for more than 17 million deaths globally each year. This figure is to grow to 23.6 million by the year 2030.[1] Estimates from the global burden of disease study suggests that by the year 2020 the South Asian part of the world (India, Pakistan, Bangladesh, Nepal) will have more individuals with atherosclerotic cardiovascular diseases than any other region.[2]

Primary PCI reduces the risk for mortality and subsequent myocardial infarction when compared with Thrombolytic therapy in patients with acute coronary syndromes. However, the invasive mechanical reperfusion strategies have their own complications. Major complications include death, MI, or stroke, and minor complications include transient ischemic attacks, vascular complications, contrast induced nephropathy, and angiographic complications.

Originally, the SYNTAX score was designed to grade the complexity of stable coronary artery disease. Higher values of this score, reflecting a more challenging coronary anatomy for the interventional cardiologist, also predict a worse prognosis after acute STEMI.[3] Patients with very low predicted mortality could benefit from early discharge from the intensive care unit and from the hospital, resulting in better clinical care and optimization of health resources. In contrast, morbidity and mortality after STEMI are still high in other subgroups.[4]

The aim of the study is to investigate the usefulness of SYNTAX score in predicting outcome of primary PCI in acute STEMI patients in terms of severity and complexity of CAD.

Methods:

This prospective cohort study was conducted in the Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh.from September, 2015 to September, 2016. The study included patients with acute STEMI who undergone primary PCI during the study period. Patients with valvular heart diseases, congenital heart diseases,prior thrombolytic therapy, prior MI, PCI or CABG and severe comorbidities were excluded.

Proper medications was given in CCU. After adequate explanation Coronary angiogram (CA) was done by conventional method. Angiographic pattern and CAD severity assessment was done by visual estimation.

The SYNTAX scores of all patients were calculated by 2 independent experienced interventional cardiologists who were blinded to the identities. The patients were divided into 2 groups, those with low SYNTAX scores d”22 (Group I) and those with intermediate to high SYNTAX scores e”23 (Group II).[5]

Data analysis was performed using SPSS version 16. Categorical variables were expressed as frequency and percentage and continuous variables as mean and standard deviation. Data was analyzed by student’s t-test, chi-square test and Fisher exact test. Multivariate logistic regression analysis was done to assess the effect of independent variable and adjustment was done for confounding variable.

Results:

Total 42 patients with acute STEMI who underwent primary PCI were enrolled in this study. The main objective of the study was to determine impact of SYNTAX score for predicting In-hospital outcome after primary PCI in patients with acute STEMI. Two patients were excluded from this study due to primary PCI failure. In our study 57.5% were in SYNTAX score ≤22 (Group I) and 42.5% were in SYNTAX score >22 (Group II).

The mean age ± SD was 51.40±13.20 years in Group I and 46.00±13.56 years in Group II (Table I). The difference was not statistically significant. There are no significant difference of traditional cardiovascular risk factors among the Group I and Group II except DM which was statistically significant different in between two group. Angiographic profile (Table I) revealed maximum (69.6% vs 17.6%) culprit lesion in LAD artery in Group I and maximum culprit lesion (64.7% vs 21.7%) in RCA in Group II, these were the statistically significant between Group I and Group II (P<0.05).

Group I= SYNTAX score ≤22; Group II: SYNTAX score >22 p value >0.05^{ns}

Regarding sex 69.5% and 30.5% patients were male and female in Group I and 88.2% and 11.8% were male and female in Group II respectively.

Angiographic outcome showed that 91.3% patient in Group I and 76.47% in Group II achieved TIMI flow 3 and the difference was statistically significant (p= 0.03). In Group I 8.7% patients and 11.76% patient in Group II achieved TIMI flow 2 and the difference was not statistically significant (p= 0.8). No patient in Group I and 11.76% patient in Group II achieved TIMI flow 1 and the difference was not statistically significant (p= 0.14).

Complications of primary PCI in Group I Vs Group II: acute heart failure 2.5. % Vs 2.5% cardiogenic shock 00 % Vs 2.5% significant arrhythmia 00 % Vs 2.5% and death 00 % Vs 5% and total in-hospital outcome: acute heart failure 5%, cardiogenic shock 2.5%, significant arrhythmia 2.5% and death 5% (Table III).

Multivariate analysis revealed that out of ten variable DM, ejection fraction and SYNTAX score were found to be the independently significant predictors outcome of the patients undergoing primary PCI with Odds ratio being 4.75 (p=0.001), 1.71(p=0.01) and 5.95 (p=0.001) respectively.

Table-I

Distribution of age, cardiac risk factor and culprit vessel among the study patients

	Group I(n=23)		Group II(n=17)		Total (n=40)		p value
Mean age±SD	51.40±13.20		46.00±13.56				0.397[ns]
Risk factors	No	%	No	%	No	%	
Smoking	13	56.5	14	82.4	27	67.5	0.085[ns]
Hypertension	14	60.9	11	64.7	25	62.5	0.804 [ns]
DM	8	34.8	14	82.4	22	75.0	0.003 [s]
Dyslipidemia	11	47.8	13	76.5	24	60.0	0.068 [ns]
Family history of IHD	5	21.7	6	35.3	11	27.5	0.343 [ns]
Culprit vessel							
LAD	16	69.6	3	17.6	19	47.5	
LCX	2	8.7	3	17.6	5	12.5	0.014[s]
RCA	5	21.7	11	64.7	16	40.0	

Data were analysis using chi-square and Fisher exact test
Group I= SYNTAX score ≤22; Group II: SYNTAX score >22;
ns-not significant; s- significant

Table-II

Angiographic outcome between two groups according to TIMI flow after primary PCI (n=40)

TIMI flow	Group I(n=23)		Group II(n=17)		p value
	No	%	No	%	
0	0		0		
1	0	0	2	11.76	0.14[ns]
2	2	8.7	2	11.76	0.8[ns]
3	21	91.3	13	76.47	0.03[s]

Data were analysis using chi-square test and Fisher exact test
Group I= SYNTAX score ≤22; Group II: SYNTAX score >22

Table-III

Complications of study patients according to SYNTAX score (n=40)

Outcome	Group I (n=23)		Group II (n=17)		Total		p value
	No	%	No	%	No	%	
Acute heart failure	1	2.5%	1	2.5	2	5.0	0.863 [ns]
Cardiogenic shock	0	00	1	2.5	1	2,5	0.453 [ns]
Significant arrhythmia	0	00	1	2.5	1	2.5	0.453 [ns]
Death	0	00	2	5.0	2	5.0	0.371 [ns]

Data were analysis using chi-square test and Fisher exact test Group I= SYNTAX score ≤22; Group II: SYNTAX score >22

Table-IV

Multivariate logistic regression analysis of determinants including SYNTAX score that affect the outcome of patients undergoing primary PCI (n=40)

	Beta	S.E	p value	OR	95% CI	
					Lower	Upper
Age	0.379	0.793	0.632	0.684	0.144	3.240
Sex	0.973	0.890	0.274	0.378	0.066	2.162
Smoking	1.278	0.763	0.094	0.279	0.062	1.242
Hypertension	0.405	0.527	0.442	0.667	0.321	4.326
DM	2.169	0.772	0.001[s]	4.750	1.926	9.754
Dyslipidemia	0.956	.0.677	0.158	2.600	0.689	9.806
Family history	1.052	0.734	0.152	0.349	0.083	1.473
Creatinine	0.091	0.376	0.809	0.913	0.437	1.909
LVEF	1.702	0.699	0.015[s]	1.714	0.046	2.717
SYNTAX score	2.973	1.132	0.001[s]	5.955	2.216	19.839

s- significance

Discussion:

There are few literatures regarding the usefulness of the SYNTAX score for predicting in-hospital outcome after primary percutaneous coronary intervention in patient with acute STEMI.

We found diabetes to increase the risk of MACE in patients who received primary PCI for acute STEMI. In other words, diabetes was significantly more prevalent in the Group II. Various studies have also highlighted the short term (during hospitalization and the first year after the disease) and long term effects of diabetes on the MACE. [6,7] Diabetes can thus be considered as a risk factor for MACE after primary PCI.

In this study, smoking did not have any negative effects on MACE. However, a previous research reported better reperfusion rate after primary PCI in smokers .[8] High arterial blood pressure is a risk factor for coronary artery disease and increases the risk of complications after acute coronary syndrome.[9,10] However, we did not find significant differences in level of blood pressure between the two groups. Hyperlipidemia is a risk factor for coronary artery disease, it has no significant effects on the incidence of MACE in our study. Some studies have identified high levels of lipoprotein (a) to be associated with poor outcome in patients with acute myocardial infarction.[11]

Angiographic profile in our study showed that left anterior descending (LAD) artery was the most common (47.5%) culprit vessel followed by right coronary artery (RCA) was 40% and left circumflex artery (LCX) was 12.5%. These were similar to the finding (LAD 51.2%, RCA 38.5% and LCX 10.2%). [12] In this study we found cardiogenic shock 2.5%, acute heart failure 5% and death 5%.[13] reported that cardiogenic shock developed in 3.4% post-randomization, and mortality at 90 days was 54.6%. Congestive heart failure occurred in 4.4% which are also compatible with our study.[14]

TIMI flow is one of the important factor in determining the outcome of primary PCI in patient with acute STEMI. In our study, TIMI 3 flow was established among 91.3% patients in Group I and 76.47% in Group II. Some study found similar finding (87.5%).[12] Multiple regression analysis showed that DM, LVEF and SYNTAX score are independent predictors of adverse in-hospital outcome following primary PCI (OR=4.75, 1.71 & 5.95, respectively). Another author also found Killip class and SYNTAX score (OR=1.12 & 1.08) as the predictors of in-hospital adverse outcome.[15]

Conclusion:

This study demonstrates that a high SYNTAX score before Primary PCI provides important prognostic information with regards to in-hospital clinical outcomes including mortality. In our acute STEMI cohort, risk of mortality was notably increased during the early periprocedural period. The SYNTAX score can be used for risk stratification in patients undergoing primary PCI.

Limitations:

Interpretation of angiograms and assessment of the SYNTAX score was not performed by QCA. Although the result of the study supports the hypothesis there were several limitations such as coronary angiogram was evaluated by visual estimation. So there was chance of interobserver and intraobserver variation to calculate the SYNTAX score. The study was carried out only in single centre, study period was short, sample size was small and it was a non randomized study.

Conflict of interest- None.

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Original Article

On-shelf Streptokinse EnsuRes More Favorable In-hospital Outcome after Acute STEMI (OSTRIC trial) - A Single Centre Randomized Controlled Trial

Afzalur Rahman[1], Mohammad Arifur Rahman[2], Farhana Ahmed[3], Rezvey Sultana[4], Nabil Amin Khan[5]

Abstract:

Introduction: The burden of CAD is increasing at a greater rate in South Asia than in any other region globally. Among them acute ST elevation myocardial infarction (STEMI) is one of the leading causes of death and disability. Major aspect of treatment of acute STEMI is reperfusion of the infarct related artery. Delay in reperfusion is associated with higher mortality and morbidity rates. While primary percutaneous coronary intervention (PCI) is the preferred mode of reperfusion, only few patients can get this form of reperfusion within recommended timelines. On the other hand, thrombolysis is easily available, economical and evaluated in several clinical studies. Thrombolysis is an important reperfusion strategy, especially when primary PCI cannot be offered to STEMI patients, with a time dependent fashion.

Methods: This randomized controlled trial was conducted in the department of Cardiology of National Institute of Cardiovascular Diseases since January 2016 to June 2018. Objective of the study was to find out the outcomes of acute STEMI patients after getting on-shelve or purchased Streptokinase (STK). Initially there was no free supply of STK in our hospital as it is an expensive drug, later on fund was arranged and STK was made available at free of cost by the hospital authority. Total 300 patients fulfilling inclusion and exclusion criteria were included in the study. Group I: 150 patients received on-shelf STK when it was made free by the authority and Group II: 150 patients received purchased STK when it was not available at free of cost. Study populations were analyzed for LVF, Cardiogenic shock, MACE (re-infarction, stroke and death) and duration of hospital stay.

Results: The mean age of the study population in group I and II were 53.88 ± 14.51 vs. 57.18 ± 15.28 years (p= 0.46). Mean door to injection time in group I and II were 25.51 ± 7.9 vs. 70.36 ± 16.6 minutes (p=<0.001). ST segment resolution was significantly higher in on-shelf STK group then purchased group which were 109 (72.7%) vs. 92 (61.3%), p=0.03. Considering the in-hospital outcome we found that in group I and group II LVF (killip III/IV) was 10 (6.7%) vs. 23 (15.3%) , Cardiogenic shock was 11 (7.3%) vs. 24(16%) , re-infarction was 9(6%) vs. 13 (8.7%) , Stroke was 6 (4%) vs. 8 (5.3%) and death was 12 (8%) vs. 23(15.3%). Among them LVF (killip III/IV), Cardiogenic shock and Death were significantly higher in group II (p=0.02, 0.01 and 0.04 respectively). Major adverse cardiac events (MACE) included re-infarction, Stroke and death, were significantly higher in group II [27 (18%) vs. 44(29.3), p= 0.02]. Mean hospital stay was significantly higher in group

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1. Director & Professor of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh
2. Junior Consultant, Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh
3. Registrar, Cardiology, National Institute of Cardiovascular Diseases, Bangladesh
4. Assistant Registrar, Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh
5. Medical Officer, Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh.

Address of Correspondence: Professor Afzalur Rahman, Director and Professor of Cardiology, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh. E mail: afzalur.bit@gmail.com. Mobile no. +8801711522615

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II (6.05 ± 1.81) then group I (5.33±1.26), (p=<0.001). Multivariate logistic regression analysis showed hypertension (p=.025) and door to injection time (p=.002) were statistically significant predictors for in-hospital major advance cardiac events (re-infarction, stroke and death) after streptokinase therapy.

Conclusion: Despite the strength of evidence based medicine pertaining to the benefits of primary PCI in STEMI, treatment options in Bangladesh are often dictated by resources, logistics, availability and affordability. In our country, not many hospitals offer primary PCI services round the clock. So thrombolysis by streptokinase it the potential reperfusion strategy in our context. In our study it has been found that on-shelf Streptokinase significantly reduce door to injection time which ultimately reduce cardiovascular mortality and mortality and also significantly reduce hospital stay. Hospitals intended to treat acute STEMI patients should have on-shelve Streptokinase to reduce door to injection time which affect the in-hospital outcome by reducing significant cardiovascular mortality and morbidity.

Key words: STEMI, Thrombolytic Therapy, Streptokinase, In-Hospital Outcome

Introduction:

CVD is the number one killer worldwide.[1,2] According to the heart disease and stroke statistics 2016 update by the American Heart Association, heart disease and stroke continue to be the top two killers worldwide. As of 2013, 31% of all deaths were from CVD, with 80% occurring in low- and middle-income countries. The burden of CVD, especially the CAD is increasing at a greater rate in South Asia than in any other region globally. The prevalence of CVD in India has been estimated to be nearly 3% in 2000, and upto10% in recent years, indicating rising prevalence.[3,4] The health care in Bangladesh is the reflection of mixed economy. Only 20% of the population has the affordability to take proper medical care either with government supported schemes or private insurance. In this scenario, it is not difficult to understand the challenges in delivery of modern evidence based management of STEMI to the majority of the population.[5]

Acute ST elevation myocardial infarction (STEMI) is one of the leading causes of death and disability. It generally occurs due to sudden occlusion of a coronary artery by formation of thrombus at the site of fissured or ruptured atherosclerotic plaque.[6] Major aspect of treatment of acute STEMI is reperfusion of the infarct related artery. Reperfusion therapy aims at restoration of antegrade flow in the occluded infarct related artery, which reduce infarct size and improves clinical outcome.[7] The management of acute STEMI has rapidly evolved worldwide during the last two decades. Despite global agreement on most issues related to the management of STEMI, wide discrepancies exist in implementation of Western guidelines in most of the developing world. The need has been felt that every country and society should adopt the existing scientific data, in combination with local limitations and strengths, and develop protocols that work best in their community.

Selection of reperfusion strategy in STEMI the prompt restoration of antegrade flow is the core aim. Delay in reperfusion is associated with higher mortality and morbidity rates. Timely reperfusion results in better myocardial salvage and preservation of left ventricular function. Despite recent advances in pharmacological and interventional reperfusion strategies, timely reperfusion still remains suboptimal in patients with STEMI. While primary percutaneous coronary intervention (PCI) is the preferred mode of reperfusion by most guidelines, only few patients with STEMI can avail this form of reperfusion within recommended timelines. On the other hand, thrombolysis is easily available, economical and evaluated in several clinical studies but fraught with dangers of re-occlusion of infarct related artery (IRA). Initial timely thrombolysis followed by early PCI to improve the patency rates, labeled as pharmaco-invasive (PI) strategy, is an attractive option of reperfusion in STEMI and may bridge the gaps in systems of care.

There are currently three reperfusion strategies recommended worldwide. Primary PCI is the preferred reperfusion strategy in patients with STEMI, provided it can be performed within guideline mandated time-frame, by an experienced team.[8] Primary PCI produces higher rates of IRA patency, TIMI 3 flow, and lower rates of recurrent ischemia, re-infarction, emergency repeat revascularization procedures, intracranial haemorrhage and death.[9] Randomized clinical trials have repeatedly shown that primary PCI is superior to thrombolysis, when performed in a timely manner, in high-volume, experienced centres.[10,11] Primary PCI results in TIMI 3 flow of IRA in over 90% of patients.[12] PCI related delay of >60 min negates any mortality benefit compared to immediate thrombolysis.[13]

Thrombolysis is an important reperfusion strategy, especially when primary PCI cannot be offered to STEMI patients, with a time dependent reduction in mortality and morbidity rates within 12 h after symptom onset. Thrombolytic therapy has greater benefit in patients treated within 1 h of symptom onset, with a sharp drop off after 3 h. Thrombolysis prevents

approximately 30 early deaths per 1000 patients treated within 6 h after symptom onset.[14]

Thrombolysis is currently the most practiced form of reperfusion method in Bangladesh. The earlier studies examined thrombolytics, initially with streptokinase (STK) and subsequently with tissue plasminogen activator (tPA) and its analogs. A meta-analysis of thrombolytics showed that this was a good way of reperfusion with improved outcomes across subsets except in those presented beyond 12 h of symptom onset.[14] Benefit from thrombolytic therapy in patients with STEMI who present more than 12 h after symptom onset has not been established, although consideration should be given in patients with on-going chest pain, with large myocardium at risk or hemodynamic instability, if primary PCI is not available.

Fibrin specific agents have some advantages over streptokinase, but are more expensive and not widely available. However, the final decision of choice of thrombolytic agent is at the discretion of the treating physician and the patient's choice. Pharmaco-invasive (PI) strategy PI strategy consists of early thrombolysis followed by either rescue PCI for patients with failed thrombolysis, or non-urgent coronary angiography to determine the need for additional revascularization within 3–24 h.[15,16] It differs from a 'facilitated' approach which consists of an immediate PCI following fibrinolysis and has shown adverse outcomes.[17]

Streptokinase is cheap, easily available and is the most frequently used thrombolytic agent in Bangladesh. Most of the hospitals of the country do not keep Streptokinase. in their shelf and it kills time to collect it from outside. Considering all points we designed this study to find out in-hospital outcomes of on-shelf and purchased Streptokinase.

Materials and methods:

This randomized controlled trial was conducted in the department of Cardiology of National Institute of Cardiovascular Diseases since January 2016 to June 2018. Objective of the study was to find out the outcomes of acute STEMI patients after getting on-shelf or purchased Streptokinase. In Bangladesh most of the patients purchase STK, as it is an expensive drug it is not kept on the shelf. In our hospital initially there was no free supply of STK later on fund was arranged and STK was made available at free of cost by the hospital authority. Total 300 patients fulfilling inclusion and exclusion criteria were included in the study. Group I: 150 patients received on-shelf STK when it was made free by the authority and Group II: 150 patients received purchased STK when it was not available at free of cost. Inclusion criteria were age >18 and <80, clinical and ECG diagnosis of acute STEMI, presented within 12 hours of onset of chest pain) and exclusion criteria were uncontrolled hypertension, old myocardial infarction, post PCI or CABG, cardiomyopathies, valvular heart disease, congenital heart disease, severe co morbidities i.e. CVD, CKD. ST segment resolution considered significant when it is reduced by 50% from the baseline. Study populations were analyzed for LVF, Cardiogenic shock, MACE (re-infarction, stroke and death) and duration of hospital stay. Informed written consent were taken from all patients.

Statistical Methods:

Data obtained from the study were analyzed and significance of differences were estimated by using statistical methods. Variables were analyzed by chi-square test, t-test where applicable. Multivariate logistic regression analysis was done. P value $p < 0.05$ were considered as significant. Statistical analyses were performed with SPSS, version 20.0 (SPSS Inc).

Results and discussion:

The mean age of the study population in group I were 53.88 ± 14.51 years and group II were 57.18 ± 15.28. There was no statistical difference (p= 0.46). Age group 41-60 years were height in both group, group I were 64 (42.7%) and group II were 60 (40%) (Table 1). Although ischaemic heart disease develops on average 7–10 years later in women compared with men, MI remains a leading cause of death in women. Acute coronary syndrome (ACS) occurs three to four times more often in men than in women below the age of 60 years, but after the age of 75, women represent the majority of patients.[7]

Male patients were 70.7% and 66.7% in group I & II respectively. Female patients were 29.3% and 33.3% in group I and II respectively with no statistical difference (p= 0.43). Risk factors analysis of our study population showed hypertension were 56 (27.3%), diabetes were 47 (31.3%), smoking were 41 (27.3%) in group I and hypertension were 60(40%), diabetes were 49 (32.7%) and smoking were 39 (26%) in group II with no statistical difference (p= 0.63, 0.80 and 0.79 (Table I).

Several recent studies have highlighted a fall in acute and long-term mortality following STEMI in parallel with greater use of reperfusion therapy, primary percutaneous coronary intervention (PCI), modern antithrombotic therapy, and secondary prevention.[18,19,20]

Among the study population it was observed that mean door to injection time were 25.51 ± 7.9 minutes in group I and it was 70.36 ± 16.6 minutes in group II. Mean door to injection time were significantly lower in on-shelf STK group then purchased STK group which is three times lower

(p=<0.001) (Figure II). If the reperfusion strategy is fibrinolysis, the goal is to inject the bolus of fibrinolytics within 10 min from STEMI diagnosis. This time is selected based on the median time from randomization to bolus recorded in the STREAM trial, which was 9 min.[21] In previous ESC STEMI guidelines,[22] the target time was 30 min, but this was calculated from FMC (as opposed to STEMI diagnosis). STEMI diagnosis should occur within 10 min from FMC.

There are several features of successful thrombolysis. ST segment resolution along with relieve of chest pain and hemodynamic improvement are prominent. In our study it has been found that ST segment resolution was significantly higher in on-shelf STK group then purchased group which were 109 (72.7%) vs. 92 (61.3%) respectively with p=0.03. patients with failed thrombolysis was treated by additional Injection Low molecular heparin. As the time elapsed thrombus become organized and resist lyses. For this reason the early thrombolytic is administered the early ST segment resolution occur.

In our study it has been found that acute extensive anterior MI were highest in groups I which was 69 (46%) followed by Antero septal 27 (18%), Inferior 24 (16%), Inferior+ RVI 19(12.7%) and High lateral 11(7.3%) on the other hand acute extensive anterior MI were also highest in groups II which was 74(49.3%) followed by Inferior 27 (18%), Antero septal 24 (16%), Inferior+ RVI 15 (10%) and High lateral 10 (6.7%) with no statistically significant difference between groups (p= 0.98) (Table III).

Considering the in-hospital outcome in our study we found that in group I significant arrhythmia was 12(8%), LVF (killip III/IV) was 10 (6.7%), Cardiogenic shock was 11 (7.3%), re-infarction was 9(6%), Stroke was 6 (4%) and death was 12 (8%) whereas in group II significant arrhythmia was 10(6.7%), LVF (killip III/IV) was 23 (15.3%), Cardiogenic shock was 24(16%), re-infarction was 13 (8.7%), Stroke was 8 (5.3%) and death was 23(15.3%). Among these variables LVF (killip III/IV), Cardiogenic shock and Death were significantly higher in group II (p=0.02, 0.01 and 0.04 respectively) (Table IV). Patients having primary PCI also had lower rates of heart failure, mechanical complications, and cardiac arrest compared with fibrinolysis and no reperfusion (P < 0.05). The rates of hemorrhage stroke (0.3%, 0.6%, and 0.1%) and other major bleeding (3.0%, 5.0%, and 3.1%) were similar in the primary PCI, fibrinolysis, and no reperfusion group (P > 0.05).[21]

In our study Major adverse cardiac events (MACE) included re-infarction, Stroke and death, were significantly higher in group II [27 (18%) vs. 44(29.3) , p= 0.02]. Poor in-hospital outcome prolonged mean hospital stay. Mean hospital stay was significantly higher in group II (6.05 ± 1.81) then group I (5.33±1.26), (p=<0.001). Multivariate logistic regression analysis showed hypertension (p=.025) and door to injection time (p=.002) were statistically significant predictors for in-hospital major advance cardiac events (re-infarction, stroke and death) after STK therapy (Table V).

Table-I

Baseline characteristics of the study population (N-300)

Variables	On-shelf STK (n=150)n (%)	Purchased STK (n=150)n (%)	P value
Age (yrs)	53.88 ± 14.51	57.18 ± 15.28	0.46
Male	106 (70.7)	100 (66.7)	0.43
Female	44 (29.3)	50 (33.3)	
Hypertension	56 (37.3)	60 (40)	0.63
Diabetes	47 (31.3)	49 (32.7)	0.80
Smoking	41 (27.3)	39 (26)	0.79
Door to injection time (min)	25.51 ± 7.9	70.36 ± 16.6	<0.001
ST resolution	109 (72.7)	92 (61.3)	0.03

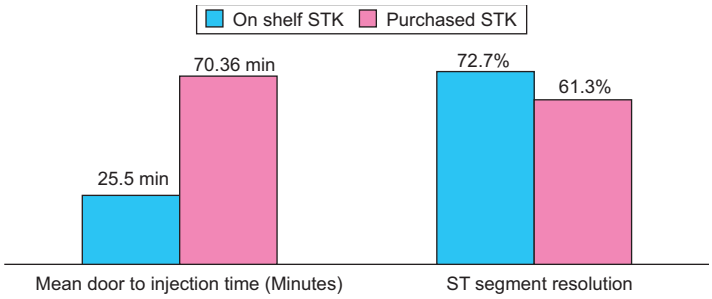

Fig.-1: *distribution of study population according to mean door to injection time and ST segment resolution.*

Table-II

Age distribution of the study population (N=300)

Age group	On-shelf STK (n=150)n (%)	Purchased STK (n=150)n (%)	P value
≤40	38(26)	27 (18)	
41-60	64(42.7)	60 (40)	0.25
61-80	42(28)	56(37.3)	
>80	5(3.3)	7(4.7)	

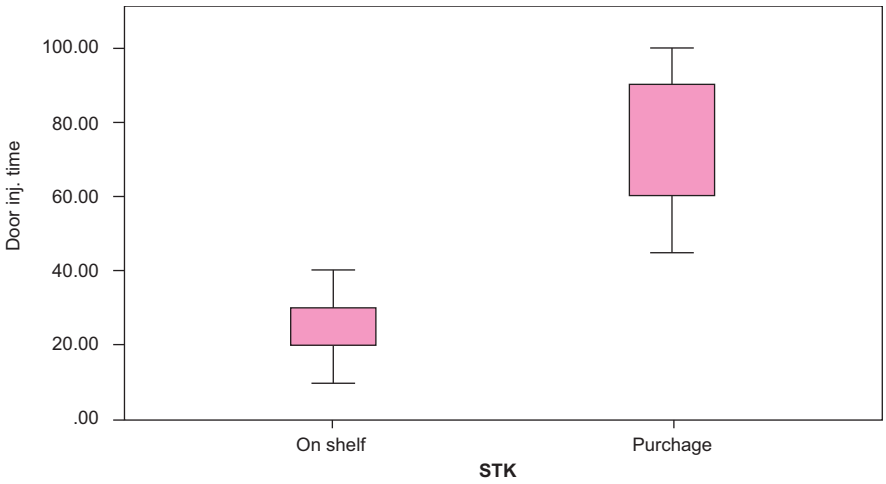

Fig.-2: *Box whisker plot showing mean door to injection time difference between groups.*

Table-III

Distribution of the study population according to ECG diagnosis (N=300)

Variables	On-shelf STK(n=150) n (%)	Purchased STK (n=150)n (%)	P value
Extensive Anterior	69 (46)	74(49.3)	
Antero septal	27 (18)	24 (16)	
High lateral	11(7.3)	10 (6.7)	0.98
Inferior	24 (16)	27 (18)	
Inferior+ RVI	19 (12.7)	15 (10)	

Table-IV

Distribution of the study population according to in-hospital outcome (N=300)

Outcome	On-shelf STK (n=150)n (%)	Purchased STK (n=150)n (%)	P value
Significant arrhythmia	12(8)	10(6.7)	0.54
LVF (Killip III/IV)	10 (6.7)	23 (15.3)	0.02
Cardiogenic shock	11 (7.3)	24(16)	0.01
Hospital stay (days)	5.33±1.26	6.05 ± 1.81	<0.001
MACE	27 (18)	44(29.3)	0.02
Re-infarction	9(6)	13 (8.7)	0.37
Stroke	6 (4)	8 (5.3)	0.58
Death	12 (8)	23(15.3)	0.04

Table-V

Multivariate logistic regression analysis for Major adverse cardiac events (MACE)

Variables	B	S.E.	Wald	df	p	Exp(B)
Age	.005	.011	.251	1	.616	1.005
Male sex	.384	.357	1.157	1	.282	1.468
HTN	.689	.308	5.007	1	.025	.502
DM	.305	.358	.726	1	.394	.737
Smoking	.472	.367	1.656	1	.198	.623
Antero septal MI	.186	.470	.156	1	.693	1.204
Door to Inj. time	.036	.012	9.414	1	.002	.965
ST resolution	.234	.325	.519	1	.471	.791

Discussion:

Acute ST elevation myocardial infarction (STEMI) is one of the leading causes of death and disability. Wide discrepancies exist in implementation of western guidelines in most of the developing world. Primary PCI is the preferred mode of reperfusion by most guidelines but in Bangladesh it is very difficult to ensure. When a patient with acute STEMI reach to coronary care unit it takes time to counsel patient and relatives about treatment options. There are several options according to time of arrival and mode of presentation. If patients present within 12 hours there are two options, one is Primary percutaneous intervention (PPCI) and another one is fibrinolysis. In our country cardiac care hospital with PPCI facilities are very limited, so large group of population are out of PPCI coverage.

On the other hand fibrinolytic therapy is alternative to PPCI as treatment option for acute STEMI patients which is cheap, available at affordable cost all over the country. STK can be given in any hospital with coronary care unit facilities thus immediate and prompt administration of STK should be ensured at every cardiac care hospital. After administration of STK there is option for Pharmaco-invasive therapy for selected cases. After advised for STK for the patients it has been observed that there is undue delay. Several factors playing role for this delay such as 1.literacy level of our population is poor, 2. Delay in decision making 3. Instant cash money may not be available in the pocket, 4. It may not be available at nearby pharmacy, 5. If available may not be preserved at proper way, 6. Price may be higher, 7. Advised drug brand may not be available 8. Supply may be hampered due to some other causes.

Aim of our study was to find out the outcomes of acute STEMI patients after getting on-shelf or purchased Streptokinase. In Bangladesh most of the patients purchase STK, as it is an expensive drug it is not kept on the shelf. In our hospital initially there was no free supply of STK later on fund was arranged and STK was made available at free of cost by the hospital authority. As door to injection time is an established predictor of post STEMI outcome, if we can cut down the time by shelving STK in the CCU it will definitely bring better outcome. In our study it was observed that mean door to injection time were 25.51 ± 7.9 minutes in group I and it was 70.36 ± 16.6 minutes in group II. Mean door to injection time were significantly lower in on-shelf STK group then purchased STK group which is three times lower (p=<0.001) (Figure II). As a result In our study it has been found that ST segment resolution was significantly higher in on-shelf STK group then purchased group which were 109 (72.7%) vs. 92 (61.3%), p=0.03. The better the early reperfusion the better the outcome. In our study we found that in on shelf STK group LVF (killip III/IV) was 10 (6.7%), Cardiogenic shock was 11 (7.3%), Stroke was 6 (4%) and death was 12 (8%) whereas in purchased group LVF (killip III/IV) was 23 (15.3%), Cardiogenic shock was 24(16%), Stroke was 8 (5.3%) and death was 23(15.3%). Among these variables LVF (killip III/IV), Cardiogenic shock and Death were significantly higher in group II (p=0.02, 0.01 and 0.04 respectively) (Table IV). MACE were significantly higher in group II [27 (18%) vs. 44(29.3), p= 0.02]. Poor in-hospital outcome prolonged mean hospital stay. Mean hospital stay was significantly higher in group II (p=<0.001). Shortened hospital stay reduce treatment cost which ultimately lessen this huge economic burden both for the government as well as of the patients. As it is evident that on shelf STK ensure early reperfusion which ultimately positively affect better outcome. As most of our STEMI patients are being treated by STK, provision should be there to keep on shelf STK in all the CCUs of the country both government and private hospitals to reduce cardiovascular morbidity and mortality.

Conclusion:

Timely delivery of reperfusion therapy (whether pharmacological or mechanical) in patients with STEMI is more important than the choice of therapy and the entire emphasis should be to deliver reperfusion therapy to a patient

of STEMI as rapidly as possible. Moreover, despite the strength of evidence based medicine pertaining to the benefits of primary PCI in STEMI, treatment options in Bangladesh are often dictated by resources, logistics, availability and affordability. In our country, not many hospitals offer primary PCI services round the clock in the urban areas and this inadequacy is pronounced more in rural areas where penetration of medical care is modest at best. In our study it has been found that on-shelf Streptokinase significantly reduce door in injection time which ultimately reduce cardiovascular mortality and mortality and door to injection time is proved to be one of the predictor for major adverse cardiac events.

Recommendations

Despite recent advances in pharmacological and interventional reperfusion strategies, timely reperfusion still remains suboptimal in patients with acute STEMI in Bangladesh. Initiation of reperfusion therapy varies, mandating uniform guidelines across the country. The right reperfusion strategy should be timely, effective, complete, safe and easily accessible. Hospitals intended to treat acute STEMI patients should have on-shelve Streptokinase to reduce door to injection time which effect the in-hospital outcome by reducing significant cardiovascular mortality and morbidity.

Conflict of interest- None.

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Case Report

Brachio- Axillary Translocation Fistula with Reverse Saphenous Venous Graft: a New Hope for the Patients of End stage Renal Disease

SMG Saklayen Russel[1], Jubayer Ahmad[2], Raju Ahmed[3], Jashim Uddin[4], Suman Nazmul Hosain[5]

Abstract:

Native arterio-venous fistula (AVF) are the preferred mode of repeated vascular access for the chronic renal failure patients surviving on hemodialysis because of their easy accessibility, good long term patency, low complication rate and cost-effectiveness. Creation of a fistula between the radial or brachial artery and a suitable adjacent vein is the most commonly practiced option. However the major upper arm veins of the CKD patients are often found thrombosed, cord like and not suitable for AV anastomosis. A 48 years old male patient of chronic kidney disease with a permanent catheter placed in the right subclavian vein was referred to create an AV fistula. On exploration none of the upper limb veins was found suitable for fistula formation. The proximal part of the left GSV was harvested from patient's left upper thigh and was used to make a connection between left brachial artery at cubital fossa and the left axillary vein. To avoid over flooding of the limb vasculature, partial banding of the left axillary vein was done distal to this anastomosis. When all options in both upper limbs are exhausted, autologous great saphenous grafts may be a very useful tool for the surgeons in creating upper limb AV fistulas in difficult situations.

Keywords: Arteriovenous fistula, autogenous AVF, graft AVF, Great Saphenous Vein.

(Bangladesh Heart Journal 2018; 33(2): 134-137)

Introduction:

Repeated vascular access is an obligatory requirement for the chronic renal failure patients surviving on hemodialysis. Native arterio-venous fistula (AVF) are the preferred mode of vascular access for the maintenance of haemodialysis (HD) in the patients with end stage renal disease because of their easy accessibility, good long term patency and low complication rate[1]. It is also cost effective for the patient. The high frequency of thrombosis and infection associated with prosthetic channels also make native fistula the first choice. As it is the best access for longevity and has the lowest association with morbidity and mortality, this is strongly recommended by guidelines from different countries[2]. Creation of a fistula between the radial artery and a suitable adjacent vein is the most commonly practiced option. Next choice is fistula involving more proximal vessels with larger caliber like brachio-basilic fistula. However the problem is the major upper arm veins of the Chronic Kidney Disease patients are often found thrombosed, cord like and not suitable for AV anastomosis. Alternate maneuvers involving more proximal veins may be engaged to overcome the situation. Their vascular access also needs maintenance and management of various related complications. There are a large number of patients in whom AVF has either failed in both upper limbs or is not feasible because of unsuitable veins. The management of these patients to get a good long-term vascular access has been a matter of debate and discussion over the years[1].

1. Assistant Professor and Associate Consultant, Dept. of Vascular Surgery, Ibrahim Cardiac Hospital and Research Institute, Dhaka.
2. Registrar, Dept. of Vascular Surgery, Ibrahim Cardiac Hospital and Research Institute, Dhaka.
3. Assistant Prof of Anaesthesia, Ibrahim Cardiac Hospital and Research Institute, Dhaka.
4. Registrar, Dept of Anaesthesia, Ibrahim Cardiac Hospital and Research Institute, Dhaka.
5. Head of the Department of Cardiac Surgery, Chittagong Medical College & Hospital, Chittagong.

Corresponding Address: Dr SMG Saklayen Russel, Assistant Professor and Associate Consultant, Dept. of Vascular Surgery, Ibrahim Cardiac Hospital and Research Institute, Dhaka. Email: saklayendmc@gmail.com.

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Case report:

A 48 years old male patient of chronic kidney disease, diabetes mellitus and hypertension with a permanent catheter placed in the right subclavian vein was referred to the department of Vascular Surgery of Ibrahim Cardiac Hospital and Research Institute to create an AV fistula for the maintainance of haemodialysis. Duplex study of right upper limb vessels couldn't reveal any flow in the cephalic vein either on arm or forearm. Only left basilic vein was visible at the arm level with a diameter of 3 mm on duplex evaluation. So we planned for left Brachio-Basilic transposition fistula. All base line investigations were done. S Creatinin level was 9.3. Patient was found to be both HBsAg positive and Anti-HCV positive. He is an ex-drug abuser.

After informed consent the patient underwent surgery with aseptic precaution on 18th July 2017. Surgical team took extra-precaution due to the seropositive status of the patient with both hepatitis B and C virus. Initially a brachial block and local anesthesia with injection lignocaine was used to create a brachio-basilic transposition fistula according to original operative plan. But on exploration the left Basilic vein was also found to be cord like and not suitable for anastomosis contradicting the preoperative duplex findings. So the operative plan had to be changed.

The patient and the party were briefed about the situation. They were offered Brachio-Basilic fistula with a prosthetic interposition graft. But patient refused that option due to financial constraint and the possible complications related to prosthetic graft. So we offered them for Brachio-Basilic translocation fistula with autologous graft i.e Great Saphenous Vein (GSV). Patient party agreed to do it. For this additional procedure the brachial block and local anesthesia wouldn't suffice. So the anesthetists had applied general anesthesia with laryngeal mask. The proximal part of the left GSV was harvested from patient's left upper thigh. End to side anastomosis was first made with the distal end of the harvested saphenous vein segment with the brachial artery at cubital fossa. This segment of vein was then passed through a subcutaneous tunnel to the left axillary region. End to side anastomosis was made with the proximal end of the harvested segment of GSV with the left axillary vein. To avoid over flooding of the limb vasculature, partial banding of the left axilary vein was done distal to this anastomosis. After proper haemostasis wound closed in layers kept a drain tube in situ. Good bruit and thrill found. The next day the drain was removed and the patient was discharged in good shape.

The postoperative clinical examination and Duplex scanning were found satisfactory on 14th postoperative day and on 3rd and 6th month. The fistula was mature by 6 weeks to start dialysis. The patient is having regular hemodialysis with this brachio-axilary fistula with translocation graft.

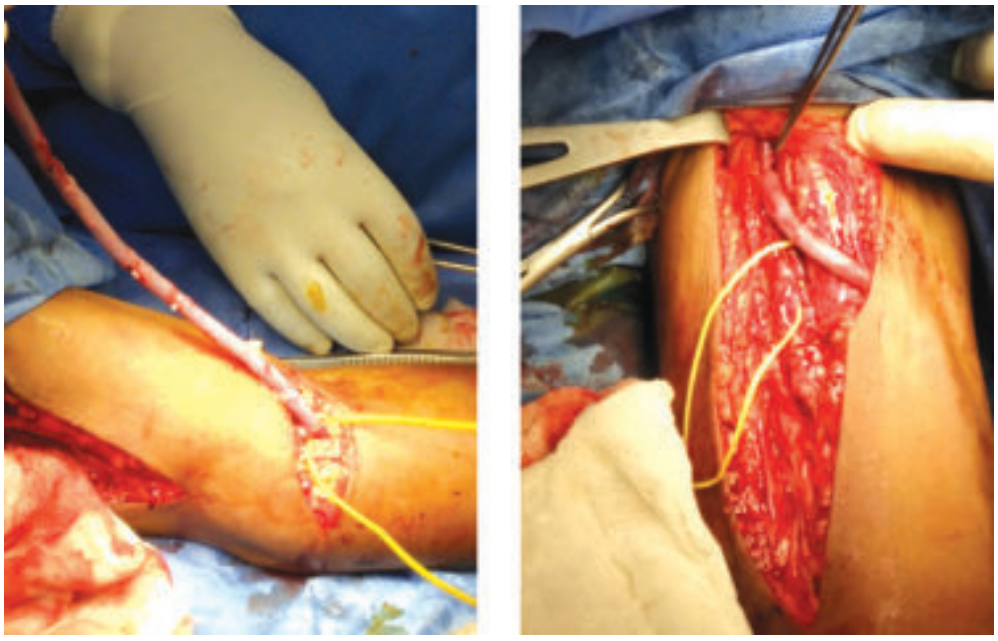

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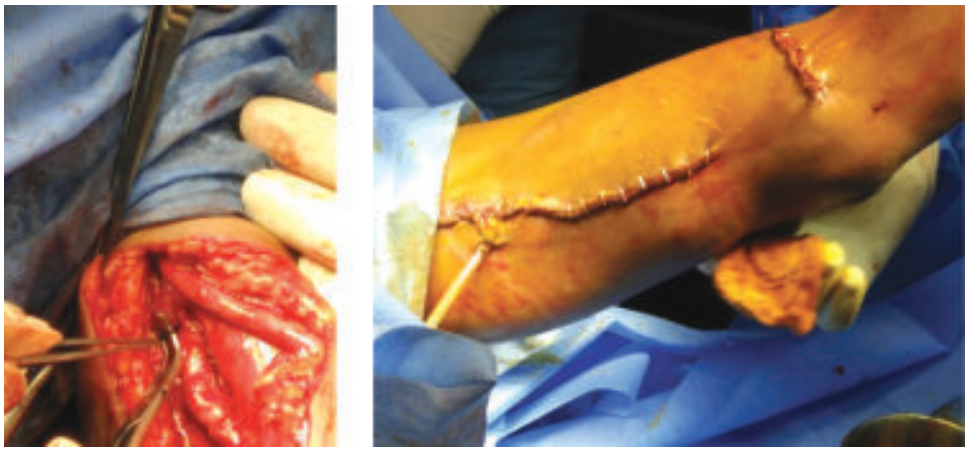
Fig.-1: *A. Brachiobasilic Translocation fistula: Proximal Anastomosis. B. Distal Anastomosis.*



A B

Fig.-2: *A. Banding of distal Axillary vein to prevent venous hypertension of left upper limb. B. After completion of surgery.*

Discussion:

It is quite evident that, whenever possible, native fistula should be preferred over prosthetic grafts. Distal upper arm fistulas involving radial artery is the first choice. If this or Bracial-Basilic fistulas are not possible then of upper arm cephalic and basilic veins with transpositions and great sapheneous vein with translocation wherever required can enhance autogenous fistula options to a large extent. Upper arm grafts should be used when no autogenous fistula is possible. Lower limb and body wall fistula sites are to be considered at the end, when all options in both upper limbs are exhausted. Autogenous arteriovenous fistulas (AVF) are the preferred mode of vascular access for maintenance hemodialysis (HD) in patients with end-stage renal disease (ESRD) because of their good long-term patency and low complication rate.

As the life expectancy of patients on long-term HD has improved with better healthcare facilities, most of them now stay on maintenance HD for much longer periods of time. Their vascular access also needs maintenance and management of various related complications. There is a large group of patients in whom Brescia-Cimino AVF has either failed in both upper limbs or is not feasible because of unsuitable veins. The best possible approach in the management of these patients to get a good long-term vascular access has been a matter of debate and discussion over the years.

The saphenous vein may be transposed either straight or in a loop fashion to make AVF. There are conflicting results in the literature about the outcome of sapheno-femoral-transposed fistulae. Lynggaard reported very poor patency rates and unacceptably high complications in their series[3]. Pierre-Paul *et al*. published their results that straight transposition of saphenous vein has better outcomes compared with loop configuration[4]. The mean primary patency was 7 months, primary-assisted patency was 15 months and secondary patency was 16 months. The fistula was functional for hemodialysis in 71.4%. But compared to its native location Great Saphenous venous grafts may be much more patent when used as a free translocation graft in creating an upper limb AV fistula.

Prosthetic grafts have inferior primary and secondary patency rates and higher incidence of some complications such as infections and thrombosis compared with autogenous fistulae[5,6,7]. As a result, the last decade has seen a gradual and intentional shift toward increasing the use of autogenous AVF[8,9]. Autologous great saphenous grafts may be a very useful tool for the surgeons in creating upper limb AV fistulas in difficult situations.

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Case Report

Atypical Mixed Total Anomalous Pulmonary Venous Connection: A Case Report

Md. Abul Kalam Azad[1], Naharuma Aive Haider Chowdhury[2], Abul Kalam Shamsuddin[3]

Abstract:

A 2 years old boy presented to us with a history of repeated respiratory tract infections and bluish discoloration of tongue, lips and figure tips for last 18 months. Echocardiography and Computed tomography (CT) angiogram revealed total anomalous pulmonary venous connection (TAPVC) mixed type (supracardiac and cardiac);all pulmonary veins drain into a common chamber behind left atrium (LA) and left lower pulmonary vein (LUPV) drains to vertical vein and common chamber both.The patient underwent rerouting of pulmonary veins and vertical vein ligation above the drainage of LUPV.

Key words:TAPVC, mixed type

(Bangladesh Heart Journal 2018; 33(2): 138-140)

Introduction:

The occurrence of multiple drainage sites in total anomalous pulmonary venous connection (TAPVC) has important implications in preoperative diagnosis and surgical treatment. Although the surgical outcome of total anomalous pulmonary venous connection (TAPVC) has improved, repair of mixed-type TAPVC is still technically challenging.[1-4]The pattern of the pulmonary venous drainage has a wide variety, most of the patients had confluence of three pulmonary veins (major drainage) and a single pulmonary vein connected to the systemic vein independently (minor drainage).[2]Here, we report a case where the minor drainage has dual connection both with vertical vein and common chamber.

Case Report:

A 2 years old boy presented to us with a history of repeated respiratory tract infections and bluish discoloration of tongue, lips and figure tips for last 18 months. He has history of hospitalization for respiratory distress at the age of 7 months. Physical examination revealed cyanosis (SPO_2 85%), grade I clubbing and systolic murmur (grade 3/6) at pulmonary area. A routine chest X-ray showed cardiomegaly, widening of superior mediastinum and pulmonary plethora with normal viscera-bronchial situs with gastric air bubble on the left side and a liver shadow on right side. In echocardiography, the patient had situs solitus and levocardia; TAPVC mixed type (supracardiac and cardiac), and all pulmonary veins drain into a common chamber behind left atrium (LA) and LUPV drains to vertical vein and common chamber both (Figure 1). CT angiogram revealed mixed TAPVC and confirmed the communication between vertical vein and common chamber (Figure 2). With these investigations, the patient was taken up for surgery.

After induction, and establishment of standard invasive and noninvasive monitoring median sternotomy was done. Anatomy was checked preoperatively. Extra pericardial vertical vein was dissected free up to the common chamber and LUPV drainage site. An incision is made in the right atrium parallel to the atrioventricular groove. The large coronary sinus is identified, which is the point of the anomalous pulmonary venous connection. The membrane of the foramen ovale is excised to provide a large opening in the atrial septum. The common wall of the coronary sinus and left atrium is incised by placing scissors through the foramen ovale. The incision is made well back into the left

1. Registrar, Pediatric Cardiac Surgery, National Heart Foundation Hospital & Research Institute, Dhaka, Bangladesh.
2. Assistant Professor, Pediatric Cardiology, National Heart Foundation Hospital & Research Institute, Dhaka, Bangladesh.
3. Associate Professor, Pediatric Cardiac Surgery, National Heart Foundation Hospital & Research Institute, Dhaka, Bangladesh.

Address of Correspondence: MAK Azad, Pediatric & Congenital Cardiac Program, Children &Women Cardiac Unit, National Heart Foundation Hospital & Research Institute, 26/4, Darus Salam, Mirpur-1, Dhaka-1216, E-mail: mak.azadcts@gmail.com, Mobile: +880 1716 297225

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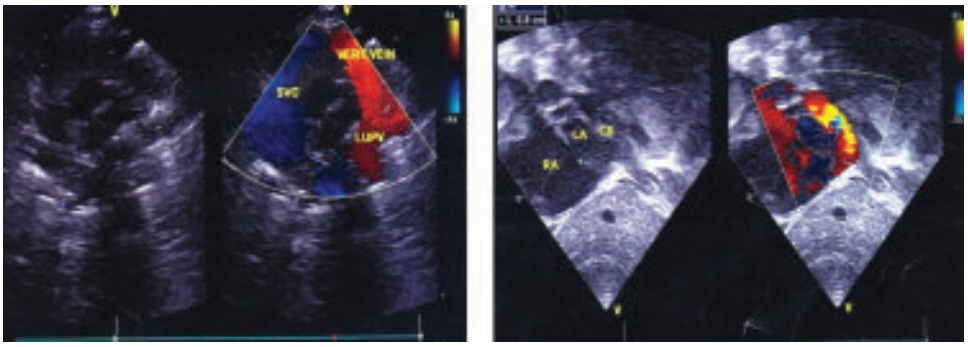

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Fig.-1 (a,b): *Preoperative Echocardiography*

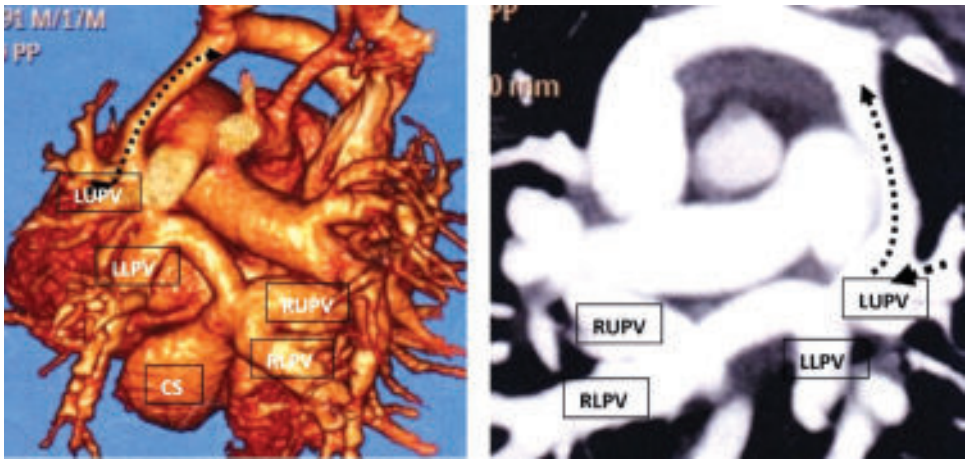

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Fig.-2 (a,b): *CT angiogram*

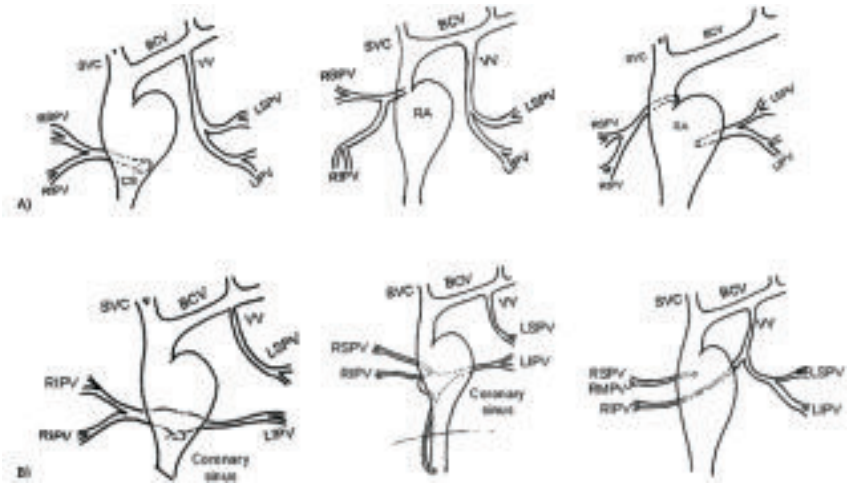

Fig.-3 (A, B): *A: bilateral and symmetrical connections ("2+2" pattern of drainage); B: bilateral and asymmetrical connections ("3+1" pattern of drainage; BCV, Brachiocephalic vein; CS, coronary sinus; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; RA, right atrium; RIPV right inferior pulmonary vein; RSPV, right superior pulmonary vein; SVC, superior vena cava; VV, vertical vein.*

atrium, to allow free drainage of the coronary sinus into the main portion of the left atrium. The atrioventricular node and bundle of His should be anterior to the incision and protected from injury. An appropriately sized pericardial patch is fashioned from the anterior pericardium to close the atrial septum. Finally, ligation of vertical vein was done just above the insertion of LUPV at vertical vein under standard Cardiopulmonary bypass (CPB) and cooled to 30^0C. Postoperative recovery was uneventful.

Discussion:

Mixed type of TAPVC is a rare cardiac anomaly, the frequency of which is 5% to 10% of the patients with TAPVC in the literature.[4]Although the surgical outcome of TAPVC correction has improved, but repair of mixed TAPVC is still technically challenging. In most cases of TAPVC, echocardiography is sufficient for diagnosis. Some authors have stated that cardiac catheterization may be unnecessary if three veins are well visualized and there is no clinical evidence of obstruction.[4]Apical and subcostal 4-chamber echocardiographic views usually best identify individual pulmonary veins and their confluence.[5, 6]

Mixed TAVCs are allocated into three categories. Category I: Bilateral & symmetrical connection, “2+2” pulmonary venous drainage pattern (Figure 3a); category II: Bilateral & asymmetrical connection, “3+1” pulmonary venous drainage pattern (Figure 3b) & category III: Bizarre anatomic variants.[1]

In this case of mixed TAPVC, the left upper pulmonary vein is left unrepaired, because it has a dual communication between vertical vein and common chamber. Though, we left it untreated, it can drain easily into common chamber.Kasama and colleagues reported two stage approach for mixed TAPVC due to single connection of LUPV to vertical vein.[7]Delius and colleagues stated that if there is evidence that the isolated anomalous pulmonary vein of mixed TAPVC is obstructed, anastomosis to the left atrium is mandatory, but the vein may be left uncorrected if it is not obstructed[4]. So, if LUPV had no dual connection, it can be left untreated. In our case the patient is lucky enough due to dual connection of LUPV.

Conclusion:

Mixed type of TAPVC is a rare cardiac anomaly and has a wide variety in pulmonary venous anatomy. There seems to be a limitation in diagnosis by echocardiography only; CT angiogram is a good alternative and recommended when mixed-type TAPVC is suspected.

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Case Report

Severe mitral stenosis with giant LA with LA thrombus - A case report

Asraful Hoque[1], Shahriar Moinuddin[2], Md. Monzur Hossain[2], Ahsanara Binte Ahmed[2], Ashfaq Nazmi[3], Souda Sultana[3], Musfeq Us Saleheen Khan[3], Md. Tanvir Hosaain[3].

Abstract:

Excess dilatation of the left atrium >60 mm is known in the literature as a gigantic atrium. This dilation is most commonly encountered in the mitral insufficiency of rheumatic etiology, but also in severe prolapses of the mitral valve, permanent atrial fibrillation, and at the left-right shunt with cardiac insufficiency. In this paper, we presented a case study of severe mitral stenosis with giant LA with LA thrombus in a 42 years old female patient. The patient underwent successful mitral valve replacement and removal of LA thrombus and discharged from the hospital with advice.

Key words: Mitral Stenosis, Giant LA, LA Thrombus and Atrial fibrillation

(Bangladesh Heart Journal 2018; 33(2): 141-144)

Introduction:

Mitral stenosis (MS) is a common finding in rheumatic heart disease and can lead to enlargement of the left atrium and stasis of blood in this heart chamber. A community based study showed the prevalence of rheumatic heart disease is 1.3 per 1000 in rural Bangladesh.[1] Current prevalence of RF and RHD may be <1/1000.[2] About 535 and 509 Mitral valve replacement were performed in our country in the year of 2015 and 2016 respectively.[3, 4] The exact aetiology is not known, Both increased left atrial pressure and weakening of left atrial wall by rheumatic pancarditis are implicated in its development.[5,6] The condition can be associated with atrial fibrillation, thromboembolic complications, hemodynamic and respiratory complications.[7] Other causes of left atrial enlargement are left ventricular failure, chronic atrial fibrillation, and significant left to right shunts as seen in patent ductus arteriosus and ventricular septal defect. Advanced rheumatic mitral stenosis with enlarged left atrium and atrial fibrillation can predispose to the formation of thrombi in the left atrium and the left atrial appendage over time if anticoagulation treatment is ineffective. Giant left atrium is a feature of rheumatic valve disease with severe mitral regurgitation.[8] We present a case of mitral stenosis associated with Giant LA with huge thrombus formation. The patient was successfully managed by mitral valve replacement and removal of LA thrombus.

Case Report:

A 42 years female, named Masuda Begum, normotensive, nondiabetic was suffering from breathlessness during moderate to severe exertion for 1 year, palpitation for 6 months and generalized weakness for 3 months. Two months back she admitted in NICVD for better treatment. Physical examination revealed she was anxious, co-operative, decubitus on choice, no anemia, non-icteric, no cyanosis with normal blood pressure and an irregular pulse.

1. Assistant Professor, Department of Cardiac Surgery, National Institute of Cardiovascular Diseases & Hospital, Sher-e-Bangla Nagar, Dhaka.
2. Assistant Registrar, Department of cardiac Surgery, National Institute of Cardiovascular Diseases & Hospital, Sher-e-Bangla Nagar, Dhaka.
3. Resident, Department of cardiac Surgery, National Institute of Cardiovascular Diseases & Hospital, Sher-e-Bangla Nagar, Dhaka

Address for Correspondence: Dr. Asraful Hoque, Assistant Professor, Department of Cardiac Surgery, National Institute of Cardiovascular Diseases & Hospital, Sher-e-Bangla Nagar, Dhaka. Tele: 01720834878, e-mail-dr_asraf_sium@yahoo.com

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Cardiovascular examination revealed visible apex beat present in a normal position, no chest deformity and no scar present. The apical impulse in normal position tapping in nature, a diastolic thrill present in the mitral area but there is no parasternal heave and no palpable P2. On auscultation, first heart sound is loud in the mitral area and low pitched, localized, rough and rumbling mid-diastolic murmur also present in the mitral area.

Chest x-ray P/A view showed cardiomegaly (enlarged in transverse diameter, straightening of left heart border with double rt. Heart border), lung fields were normal to congestive. ECG showed atrial fibrillation. Echocardiography confirmed severe mitral stenosis (MVA -0.7cm^2) with AML & PML both thickened and less pliable, both commissures are fused with subvalvular changes grade IV and moderate MR. LA was dilated (63x37 mm), thrombus is seen in posterior wall of left atrium without compressing RA. Pulmonary hypertension present with PASP 41 mm of Hg with grade I tricuspid regurgitation. The patient was getting anticoagulation before surgery and the dosage of anticoagulation was optimized to achieve the INR 2.5.

With all aseptic precaution, a median sternotomy was done. The thymus was dissected and ligated. Pericardiotomy was done and the Cardiopulmonary bypass was established with the bi-caval cannula. There was a 6x4x3 cm in size organized LA thrombus was found and thrombus was removed. (Fig 1, Fig 2 and Fig 3) Pulmonary veins opening were found free of thrombus. Mitral valve found both AML and PML thickened with severe subvalvular change. Mitral valve and it's apparatus excised and replaced with 27 mm

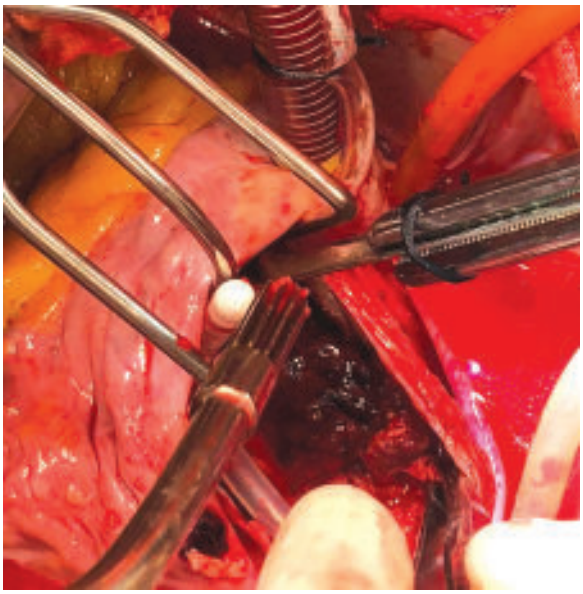

Fig.-1: *Thrombus seen inside LA.*

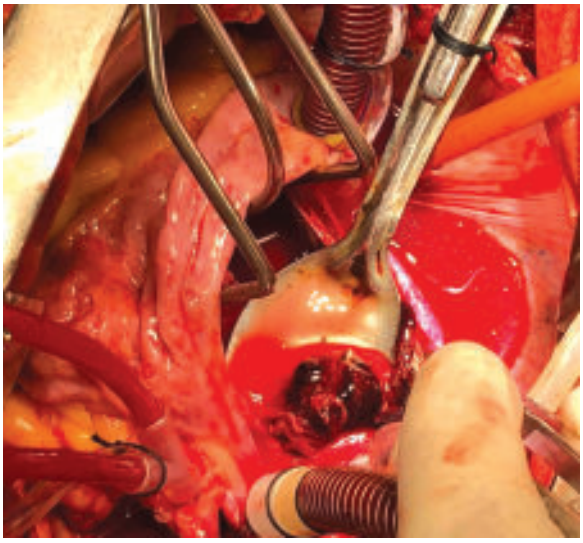

Fig.-2: *Removal of thrombus*

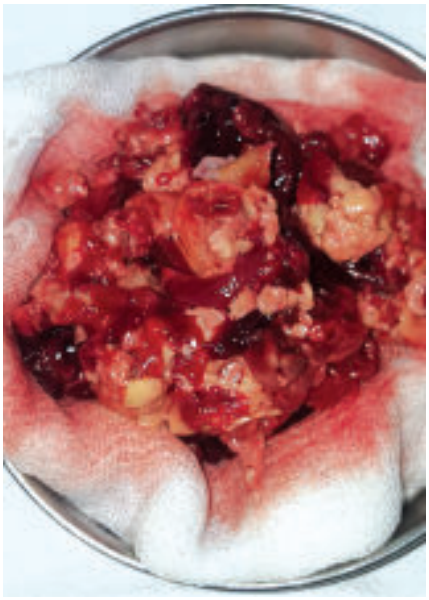

Fig.-3: *Cauliflower like thrombus*

St. Jude bi-leaflet mechanical valve. Maze procedure was performed for atrial fibrillation. Left atriotomy was closed in layers. Pacing wires were placed in RV wall and skin. Chest closed in layers after keeping 2 chest drain tube in retrosternal space. Proper hemostasis was ensured. The per-operative period was uneventful. The patient was extubated on 8 hours after the operation. The drain tube was removed on the 2nd POD. Patient shifted from ICU to ward on the 4th POD. Her GCS level is 15, no peripheral artery blockage is seen, heart rate is 80 bpm. Tab. Warfarin 5 mg is continued. Patient discharged on the 8th POD.

Discussion:

The differential diagnosis of intracavitary masses, particularly LA, may be quite wide and can be a case of a primary intracardial tumor (usually myxoma), thrombus, intracardial cyst or vegetation. The diagnostic method of choice is echocardiography (TTE or TEE).[9] The incidence of the left atrial thrombus in a patient with mitral stenosis and atrial fibrillation varies between 7 and 38%.[10,11] The cause of left atrial dilatation is not only an increase in intracavitary pressure but also a consequence of rheumatic carditis with chronic inflammation and myocardial fibrosis.[5,12] Such dilation and atrophic fibrosis are the basis for the formation of atrial fibrillation the most common cause of ischemic cerebrovascular incidents, and in itself causes further dilation of LA. Patients with severe mitral stenosis and atrial fibrillation, as shown, have an increased risk and require adequate anticoagulant treatment. In patients with mitral stenosis, the level of fibrinopeptide A, thrombin-antithrombin III complex and von Willebrand factor in the left atrium is increased.[13] The dilation of LA is associated with the blood trauma and the formation of a thrombus, and the risk of thromboembolism increases with the enlargement of the left atrial dimension, independent of the administration of an anticoagulant.[14]

According to Isomura and co-workers, left atria larger than 6 cm diameter is termed as giant left atria.[15] In our patient had LA size was 63 mm, which is consistent with the above study. Hurst states that in cases of a giant left atrium, mitral regurgitation is more profound than mitral stenosis, and atrial fibrillation is almost always present.[5] Giant left atria may rarely present in severe mitral stenosis alone, mitral regurgitation should also be present.[16] In our case, X-ray chest of the patient revealed cardiomegaly. On echocardiography, there was severe mitral stenosis with enlarged LA (63 mm) not compressing RA with moderate MR which is consistent with the above study.

The patient of giant left atria usually presents with complaints of shortness of breath and/or dysphagia.[17] Our patient had complaints of shortness of breath on exertion, palpitation without any compressive symptoms like dysphagia which is consistent with the above study.

Patients with severe mitral stenosis and atrial fibrillation, have an increased risk embolic events and require adequate anticoagulant treatment. We also optimized anti-coagulation therapy to achieve the INR 2.5.

In the view of such an enlarged left atrium with the progression of symptoms with atrial fibrillation, we advised our patient for mitral valvular replacement surgery to avoid further complications. Successful mitral valve replacement surgery was done and the patient discharged from the hospital with advice. Ahmad and his colleagues also published a Case Report of Giant Left Atrium in a case of Mitral stenosis with LA thrombus.[18]

Such a giant left atrium with severe mitral stenosis with mild mitral regurgitation with AF is a rare entity[19] and hence we have reported this case.

Conclusion:

Rheumatic mitral stenosis, even of moderate severity, associated with an enlarged left atrium and atrial fibrillation can be complicated by the formation of thrombi. Surgical management was done successfully and the patient discharged from the hospital.

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