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Journal of Invasive and Clinical Cardiology

INSTRUCTION TO AUTHORS

A. Introduction

The Journal of Invasive and Clinical Cardiology is a biannual, peer-reviewed journal and aims to publish work of the highest quality from all sub-specialties of Cardiology. The aim of the publication is to promote research and serve as platform for dissemination of scientific information in Cardiology.

B. Categories of Articles

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

Original Research:

Original, in-depth research article that represents new and significant contributions to medical science. Each manuscript should be accompanied by a structured abstract of up to 250 words using the following headings: Objective, Methods, Results, and Conclusions. 3 to 5 keywords to facilitate indexing should be provided in alphabetical order below the abstract. The text should be arranged in sections on INTRODUCTION, METHODS, RESULTS, and DISCUSSION. The typical text length for such contributions is up to 3000 words (including title page, abstract, tables, figures, acknowledgments and key messages). Number of references should be limited to 50.

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

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

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1. Category of manuscript (original research, review article, case report, cardiovascular image, letter to the Editor)
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The cover letter should also include the mailing address, telephone and fax numbers, and e-mail address of the corresponding author.

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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.

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- Title. Concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations where possible.
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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.).

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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.

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

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

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

- References should be numbered consecutively in the order in which they are first mentioned in the text.
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Examples of correct forms of references are given below:

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

1. *Standard journal article*

   List the first six authors followed by et al.

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

   More than six authors:

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

2. *Organization as author*

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

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

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

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

4. *Volume with supplement*

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

5. *Issue with supplement*

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

6. *Type of article indicated as needed*

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

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

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

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

Books and Other Monographs

1. *Personal author(s)*

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

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

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

3. *Organization(s) as author*

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

4. *Chapter in a book*

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

5. *Conference proceedings*

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

6. *Dissertation or thesis*

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

Other Published Material

Newspaper article

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

Unpublished Material

In press or Forthcoming

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

Electronic Material

1. *Journal article on the Internet*

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

Article published electronically ahead of the print version:

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

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

Article with a Digital Object Identifier (DOI):

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

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

2. *Monograph on the Internet*

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

3. *Homepage/Web site*

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

G. Submission Preparation Checklist

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

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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.

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COVID-19 Cardiovascular Manifestations: Diagnostic Challenges and Therapeutic Ambiguities

Chowdhury Ahsan[1], Christoph W Sossou[2], Jagat Narula[3]

J Inv Clin Cardiol 2021; 3(2): 44-48

Introduction:

The cardiovascular manifestations of coronavirus disease-2019 (COVID-19) are complex, with patients presenting with ST-elevation myocardial infarct (STEMI), non-ST elevation myocardial infarct (NSTEMI), myocarditis mimicking STEMI presentation, stress-induced cardiomyopathy, non-ischemic cardiomyopathy, coronary vasospasm, global myocardial stunning and non-acute coronary syndrome myocardial injuries (non-ACS MI).

The heterogenous presentation of patients with COVID-19 infection coupled with the global disruption of healthcare delivery, make a timely and accurate diagnosis of ACS and subsequently its management in this population extremely challenging. Herein, we discuss the challenges of accurately diagnosing acute coronary syndrome in COVID-19 patients, differentiating true ACS from ACS mimics in patients with COVID-19 and discuss the logistic challenges of managing ACS patients with concurrent COVID-19 infection.

COVID and Acute Coronary Syndrome

The clinical presentation of acute coronary syndrome is heterogenous (Figure 1), particularly in those with concomitant coronavirus disease-2019. Such patients often present late and atypically, leading to delay in initiating the diagnostic and therapeutic cascade, hence conferring upon them high morbidity and mortality.[1] All patients presenting with type I myocardial infarction (MI) during the COVID-19 pandemic should be presumed to be COVID-positive and appropriate precautions should be taken.[2] However, delay from first medical contact to cardiac catheterization laboratory should be minimized. Although high clot burden is

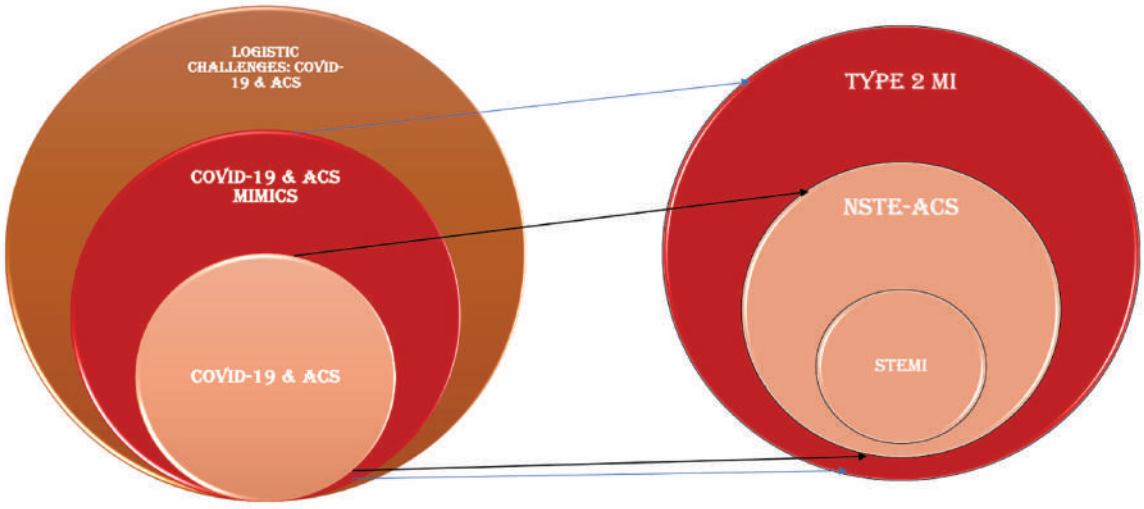


Fig.-1: *COVID-19 and Acute Coronary Syndrome*

1. Clinical Professor of Medicine, University of Nevada, USA.
2. NJ Children's Hospital Medical College, New Jersey, USA
3. Professor of Medicine, Cardiology, The Mount Sinai Hospital, USA

common in this patient population, primary PCI and downstream P_2Y_{12} administration, instead of fibrinolysis therapy remains the gold standard.[2,3] High-grade, hemodynamically significant lesion in non-infarct related artery should also be addressed at index procedure to further minimize exposure of clinical staff during a staged procedure.[2] Intravenous infusion of glycoprotein IIb/IIIA inhibitors in conjunction with anticoagulation after primary percutaneous coronary intervention (PPCI) in COVID-19 patients with type I MI should be individualized, considering the ischemic vs. bleeding risk.

Just because we can, does not mean we should. Not all COVID-19 patients with STEMI will benefit from reperfusion strategy or advanced mechanical support,[2] compassionate care is always an option for some patients in intensive care units given significant pulmonary involvement and multiorgan failure with grave prognosis .

The overwhelming majority of hospitalized patients with COVID-19 infection have positive biomarkers and at times diffuse cardiac structural abnormalities on transthoracic echocardiogram.[4] This creates diagnostic and therapeutic dilemma for clinicians. While Type I MI might be possible, type 2 MI cannot be ruled out with certainty.[2] Given the heterogeneity of presentation and reduced specificity of high-sensitivity cardiac troponin, distinguishing between NSTE-ACS vs. type 2 MI is not only daunting but seem impossible at times. Fibrinolysis therapy is not advisable in this subgroup of patients. Bedside echocardiography and other imaging studies such CT angiogram can help to address other differential diagnoses. Urgent coronary angiography with possible primary percutaneous coronary intervention (PPCI) is not immediately warranted when the overwhelming other clinical issues such as severe hypoxia, acidosis and metabolic disturbances can explain an EKG abnormalities which triggered STEMI activation by the treating physicians not familiar with STEMI mimics in EKG and some patients with high-risk clinical features and hemodynamic instability and significant other comorbidities may not be suitable candidates for revascularization for overall benefits.[2] Patients with COVID-19 infection are inherently complex, targeted differential diagnosis should be undertaken prior to settling on appropriate reperfusion strategy.

Acute Coronary Syndrome mimics in COVID-19

A significant proportion of patients with COVID-19 requiring hospitalization have biomarker evidence of myocardial injury.[2] A good portion of these patients are acidotic with multiple electrolytes derangements (hyperkalemia), some may have coronary artery spasm, focal perimyocarditis, LVH, early repolarization, LBBB, stressed induced cardio-myopathy, dysrhythmia, acute pulmonary embolism, anemia etc. all of which may have ST-elevation or equivocal ECGs, mimicking acute coronary syndrome.[2] Cardiac structural abnormalities on transthoracic echocardiogram are common in this subset of patients as well.[4] Pursuing early reperfusion strategy (fibrinolysis or percutaneous coronary intervention) in this patient population confers bleeding risk with no benefit and can potentially give unnecessary exposure to the personnel involved in cardiovascular care.

It is understandably difficult at times to differentiate type I MI from type 2 MI in this patient group. However, clinical impression, ECG, laboratory, and imaging data can inform a decision between the intensive care physician and interventional cardiologist regarding appropriate action.[2] A close collaboration between the interventional cardiologist, emergency room physician and the intensive care physician fosters an impactful relationship, which may lead to excellent patient care. Resource stewardship, protection of staff and practitioners is paramount, especially during the COVID-19 pandemic. Not all ST-segment elevations equate to type I MI and not all type I MI patients with concurrent COVID-19 infection would benefit from early reperfusion strategy. Patient selection with assessment of overall prognosis and co-existent other COVID complications would need to be considered before considering invasive reperfusion strategies.

LOGISTIC CHALLENGES: Acute Coronary Syndrome & COVID-19

The COVID-19 pandemic has greatly disrupted healthcare delivery globally. The fear of contagion or burdening already overwhelmed emergency departments has refrained patients with chest pain and ACS-related symptoms from immediately seeking medical care.[1,5-8] This is evidentially reflected in the significantly reduced number of primary PCI procedures performed during the peak of the COVID-19 pandemic.[5] The heterogeneities of COVID-19 patients presentation coupled with the several ACS mimickers commonly seen in such patient population further lead to diagnostic dilemmas and therapeutic ambiguities.[1] Additionally, institutional specific COVID-19 protocols,

instead of national, universally adapted protocols for screening patients, preparing catheterization laboratory and clinical staff further delay diagnosed, hence prolonging ischemia time. A longer ischemic time has grave morbidity and mortality consequences, especially in patients with type 1 MI in whom a longer time delay has a significant negative impact on myocardial salvage, preservation of left ventricular systolic function, and overall survival.[6-7] It is undoubtedly true that the COVID-19 crisis poses significant logistic challenges to healthcare systems. Nonetheless, attempt to contain the pandemic should not wane the efforts spent in minimization of total myocardial ischemia time, as this is directly correlated with late sequelae of myocardial necrosis and worse overall prognosis.[1, 9]

Universal adoption of standardized fast-track pathways for the exclusion of COVID status would likely lead to rapid yet accurate recognition of ACS and ACS-mimickers, leading to immediate institution of optimal therapeutic interventions while avoiding additional risks to patients and personnel (i.e., avoidance of fibrinolysis or PPCI in COVID-19-related focal myocarditis, or stress-induced cardiomyopathy, or performing

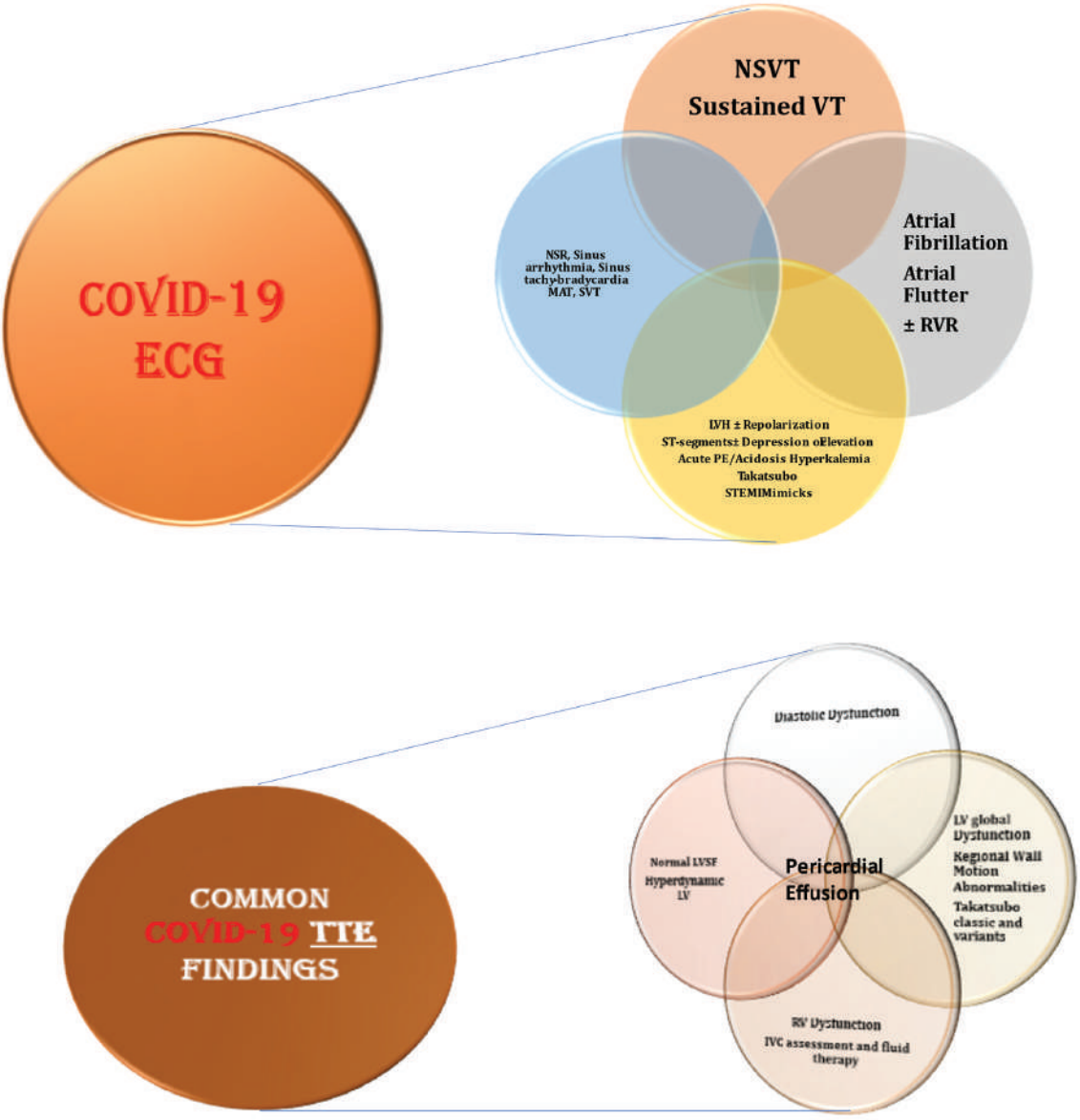


Fig.-2: *Non-specificity of Commonly used Diagnostic Tests*

echocardiography in otherwise asymptomatic, stable patient). Although ECG, cardiac biomarkers, and TTE are the bedrock of cardiology, these are not substitute for detail history, excellent physical exam, and clinical acumen.

The use of ECG and TTE which have always been regarded as "gatekeepers" for differential diagnosis of cardiovascular disease, should be reconsidered during this pandemic.[10] These tests have broad nonspecific findings (Figure 2), hence should not be routinely performed if patients are asymptomatic and stable, but they remain the first line approach in patients with high suspicion of ACS with concurrent COVID-19.[10,11] Likewise, invasive coronary angiography should be reserved for type 1 MI or high-risk patients with crescendo angina and unexplained, significant elevation in biomarkers who are expected to derive meaningful benefits.[10,12-13] Patients with multiple, terminal comorbidities, poor quality of life, and significant frailty should be considered for conservative management early on, as further diagnostic investigations may be futile.[10]

Asserted effort ought to be made [via local radio/TV stations, social media, or journals to educate the public about the early recognition of high-risk ACS symptoms during the pandemic and importance of promptly presenting to the nearest emergency department. It is vital to distinguish true ACS from ACS-mimickers in patients with COVID-19 by judiciously employing sacred diagnostic resources and protective measures, to ensure safe conditions for patients and staff. Though there is a role for fibrinolysis therapy in true STEMI only, pharmaco-invasive therapy and primary PCI remain the standard of care for type 1 MI and clinicians should evaluate the feasibility of a timely interventional therapy based on staff, PPE, and Cath-lab availability.

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Intermediate Coronary Lesion in Our Patient Perspective: FFR/dFR guided Percutaneous Coronary Intervention

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Abstract:

Background: Intermediate coronary artery stenotic lesion, defined as visual angiographic stenosis severity of between >40% to <70% is not uncommon in our patient population. Exact Data on Pre-PCI physiological study by Fractional Flow Reserve (FFR) or diastolic Flow Ratio(dFR) in angiographic intermediate lesion in our population not well known.

Objectives: Therefore, our primary aim was to assess the lesion severity of angiographic intermediate coronary artery stenosis by performing pre-PCI FFR/dFR and thereby, to perform percutaneous coronary intervention (PCI) in significant lesion and to keep on medical therapy in non-significant lesion.

Methods: Total 50 patients (Male 39, Female 11) were enrolled in this observational non-randomized prospective cohort. Patient who had angiographically intermediate lesion were taken into consideration for physiological study by pre-PCI FFR and studied. Total 55 vessel of 50 patients were studied. Cut off value for FFR were clinically significant only if Pd/Pa ratio <0.75 and dFR <0.85.

Results: In the studied patient population, FFR done in total 80%(n=44), dFR 7.3% (n=4) and both FFR & dFR in 12.7% (n=7) patients. Among vessel wise distribution physiological study (FFR/dFR) were done in LAD 69% (n=38), LCX 12.7%(n=7), RCA 16.3%(n=9) and Ramus Intermedius 1.8% (n=1). Physiological study (FFR/dFR) was nonsignificant in 62%(n=31), were kept on medical treatment and significant in 38% (n=19) patients, were treated by drug eluting stents. Among, the significant intermediate lesion all were male.

Conclusion: We found, a good number of intermediate coronary lesion are significant by physiological (FFR/dFR) study, which may develop MI if left untreated and quite a large number patient can be left on medical treatment, thus, to avoid from inappropriate stenting and subsequent consequences. Individual assessment by FFR/dFR of these intermediate lesions prior decision making is recommended.

Key Words: BF lesion, PCI, FFR, dFR

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Introduction:

Conventional coronary angiogram is the mainstay of diagnosis of coronary obstruction and thus clinicians can take appropriate revascularization decisions. Although invasive coronary angiography is the gold standard investigation to assess severity and extent of CAD. Angiography is limited as it is a two-dimensional representation of a more complex three-dimensional structure. Foreshortening or overlay may the important reason to reliably assess the stenosis severity in highly eccentric lesions. Thus, visual estimation of stenosis severity lacks precision with up to 30% of angiographic assessments found to be erroneous and inter-observer variance in diameter stenosis of 15–45%.[1]

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Given these inherent difficulties in accurate stenosis assessment on angiography, patients often fall into a category termed intermediate stenosis severity. Intermediate stenosis is defined as an angiographic stenosis of between >40% to <70% and presents a distinct clinical challenge with uncertainty about optimal assessment and management strategy.[2] Although its prevalence in the general population is not well known, registry data suggests it may be present in up to 25% of patients undergoing coronary angiography.[3]

Angiographic intermediate coronary lesion is challenging to interventionist on decision making, how to treat this subset of coronary artery disease (CAD) as many may be the cause of acute myocardial infarction.[4] In our day-to-day Cath lab procedure, we often find many of the patient has angiographic intermediate coronary lesion at time of routine or emergency angiogram and difficult to judge at the time how to deal with these subsets of patient's population.

Invasive physiological assessment is recognized as the optimal method for assessment of intermediate angiographic lesions. Approximately 20 years ago, *Pijls N and De Bruyne B et al* introduced a coronary pressure wire derived index for interrogating intermediate coronary lesions termed as fractional flow reserve (FFR).[5-6] FFR is defined as the maximum flow down a vessel in the presence of a stenosis compared with the maximum flow in the hypothetical absence of stenosis.

This assessment is undertaken during the conditions of maximal hyperemia induced by administering adenosine, to ensure the effects of the coronary microcirculation are minimized. Pressure-wire derived FFR is the most utilized invasive tool and has been found to be superior to angiography when used to guide revascularization strategy.[7] Recent data also now support the role of hyperemic-free pressure derived indices, such as the instantaneous wave-free ratio (iFR).[8-9] Both physiological approaches are supported by current European Society of Cardiology (ESC) guidelines on myocardial revascularization suggesting that, when evidence of ischemia is not readily available, FFR or iFR should be utilized to assess the hemodynamic significance of intermediate grade coronary stenosis.[10] FFR is a Class 1a recommendation for guiding coronary revascularization in patients with stable angina, are widely adopted in the catheterization laboratory.[11-12] Diastolic Hyperemia-Free Ratio dFR are adenosine-free and is measured during the entire period of the diastole. (dFR) calculates a diastolic portion of the cardiac cycle at rest and uses a cutoff of 0.89.[13] dFR was found to show the numerical consistency to iFR.[14]

The role of pressure wires derived physiology study by using FFR/dFR in treating angiographic intermediate coronary lesion in our population is not well known. Therefore, we have carried out this observational study to see the impact of FFR/dFR guided PCI in these group of patients in our population. Thus, to prevent myocardial infarction by treating the pressure-wire derived significant intermediate coronary lesion and kept on medical treatment of insignificant intermediate lesion with the possible harmful effects of inappropriate stenting and cost savings.

Method:

Methods and Materials: Total 50 patients (Male patients 39, Female patients 11), were enrolled in this prospective cohort study. Coronary angiogram (CAG) done at our center and found to have approximately >40% to <70% lesion, were considered as intermediate coronary lesion, and planned for pre-PCI FFR/dFR study. Among them FFR/dFR significant patients were stented by drug eluting stents. Patient were routinely loaded with pre-procedural Ticagrelor 180mg or Clopidogrel 300mg and Aspirin 300mg with post procedural maintenance doses Ticagrelor 180mg bid Clopidogrel 150mg and Aspirin 150mg for those whose intermediate lesion turned significant by pressure-wire derived flow resistance significant to cause stenosis.

Coronary Angiogram

Diagnostic CAG were performed using 5-6 French Terumo Radial catheter through radial approaches. To avoid spasm and to achieve maximal epicardial vasodilatation, (0.1-0.3mg) nitroglycerine and mild sedation with 1mg IV midazolam was commonly given to enrolled patient with prior written consent for the procedures in detail. All stenosis was assessed visually. Patient with angiographic intermediate lesion of about >40% to <70% lesion was enrolled for pre-PCI FFR or dFR study.

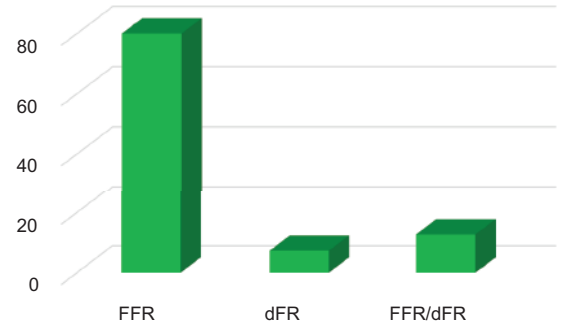
FFR and dFR:

FFR was defined as the ratio between mean distal coronary pressure and mean aortic pressure at maximal hyperemia. Intracoronary pressure wire

0.014-inch was introduced via a 6F guiding catheter, calibrated, advanced into the coronary artery, and placed >3cm distal to the assessed stenosis as described previously.[15] FFR was assessed after the administration of 200mcgm adenosine to achieve maximal hyperemia. Simultaneous recording of aortic and distal coronary pressure was performed. FFR was calculated as the ratio of the mean distal (trans-stenotic) coronary pressure measure by the pressure wire to the aortic pressure measured by the guiding catheter at maximal hyperemia. For dFR intracoronary adenosine not used, pressure-wired placed distal to stenosis and measurement recorded as significance <0.85. Generally, PCI was performed in patients with FFR <0.75 and dFR <0.85. Patients were loaded with Ticagrelor and Aspirin before the procedure. Written consent taken prior. Patient whose intermediate coronary lesion by angiography turns significant either by dFR or FFR, were received intra-procedural Integrillin loading and post-procedural IV infusion of 25 mg as our common protocol along with DAPT.

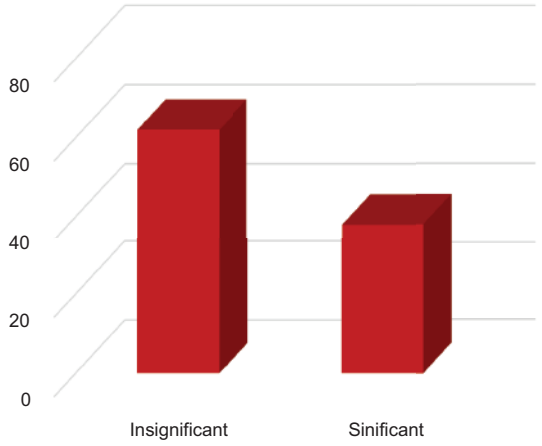
Result:

Total 50 patients (n=50; Male-39, Female-11) were enrolled in this observational non-randomized prospective cohort. Average age for male 58yrs vs Female 57 yrs. Total 55 vessel were studied by FFR/dFR prior intervention. Patient who had angiographically intermediate coronary lesion >40 to <70%, were planned for pre-PCI physiological study by FFR or dFR. Cut off value for FFR were clinically significant only if Pd/Pa ratio <0.75 and for dFR Pd/Pa ratio <0.85. Fig. 1 shows Percentage distribution of total FFR and or DFR or both FFR/dFR in the studied patients. FFR done in total 80% (n=44), dFR 7.3% (n=4) and FFR/dFR in 12.7% (n=7) patients. Fig. 2 shows Percentengae distribution of significant/insignificant intermediate lesion by FFR/dFR. FFR/dFR was nonsignificant in 62%(n=31), kept on medical treatment and significant in 38% (n=17) patients, were treated by drug eluting stents. Among the significant stenosis, all were male. Fig. 3 Shows the percentage distribution of coronaries in which FFR was done. Among the vessel wise FFR and or dFR distribution; in LAD 69% (n=38), RCA 16.3% (n=9), LCX 12.7% (n=7), and Ramus Intermedius 1.8% (n=1).



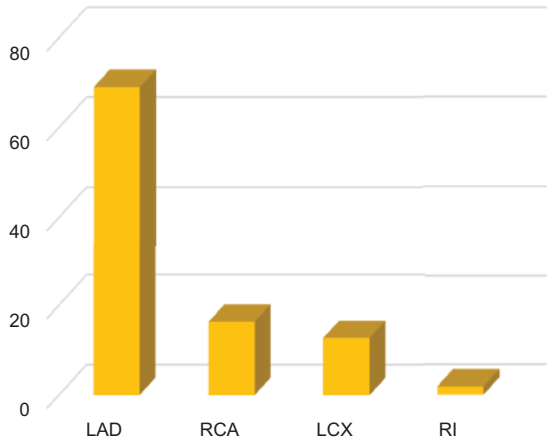
FFR 80%(n=44), dFR 7.3% (n=4) and FFR/dFR in 12.7% (n=7) patients

Fig.-1: *Percentage distribution of FFR/dFR or both in the studied patients (n=50).*



FFR/dFR was insignificant in 62%(n=31), kept on medical treatment and significant in 38% (n=19) patients, were treated by drug eluting stents. Among significant all were male.

Fig.-2: *Percentengae distribution of significant/insignificant intermediate lesion by FFR/dFR (n=50)*

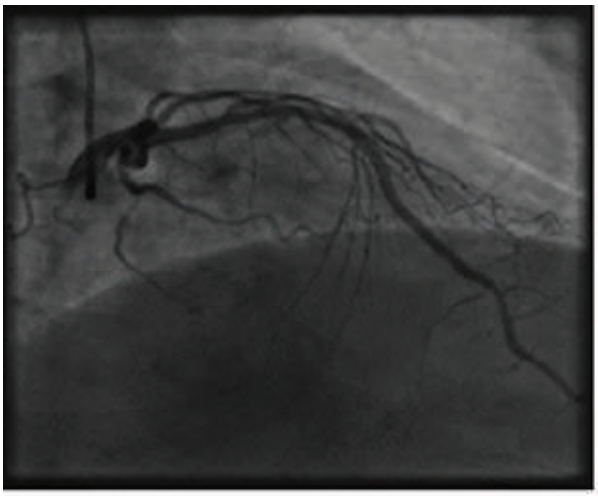


LAD 69% (n=38), LCX 12.7%(n=7), RCA 16.3%(n=9) and Ramus Intermedius 1.8% (n=1)

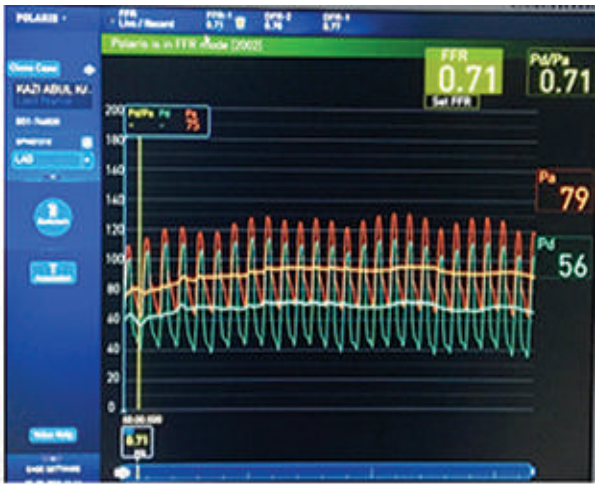
Fig.-3: *Shows the percentage distribution of coronaries in which FFR/dFR studied (n=50)*

Young Gentleman, presented with recent inferior myocardial infarction and treated pharmacologically in Covid era. EKG showed Q in inferior leads, echocardiography showed Hypokinetic distal Septum EF 50%. Planned CAG done, revealed Normal RCA and LCX. LAD is Type IV vessel, has got 50% plaque at the junction of mid and distal segment. Considering the h/o recent MI, we plan physiology study of the LAD lesion and found significant FFR 0.71. Lesion prepared well by pre-dilation and deployment of 2.5 x 18 mm DES. Post dilatation done and further optimization done by stent Boost and 3.0 x 10 mm NC balloon. Final Cine showed well apposed stent with TIMI III distal flow (Fig. 4).

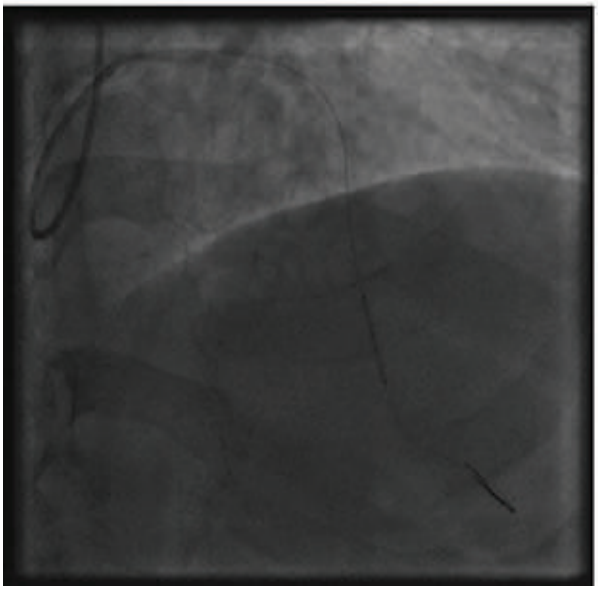
Patent admitted for CAG with background history of single episodic Angina III, H/o DM, Dyslipidemia, Echo showed no RWMA, LVEF 60%. CAG revealed 50% plaque in LAD proximally at the level of D-1. Imaging physiology shown FFR 0.86, found as non-significant and kept on medication (Fig. 5).



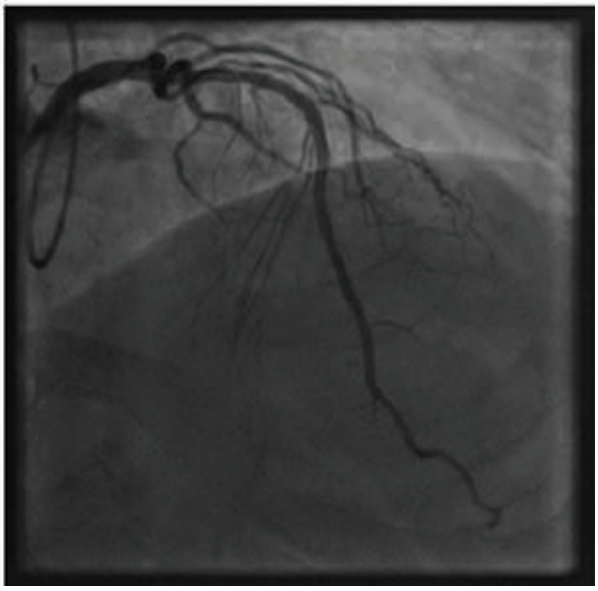
a. 50-60% lesion at junction of mid and distal LAD



b FFR 0.71



c 2.75 x 18 mm DES deployed at 12-14 ATM



d. Well apposed stent with TOIMI III distal flow

Fig.-4: *62 yr gentleman, recent Inferior MI, DM, HTN, Dyslipidemia, LVEF 50% with Hypokinetic distal IVS. CAG revealed 50-60% LAD lesion at the junction of mid and distal LAD. FFR 0.71, stented by deploying 2.75 x 18 mm DES at 12-14 ATM*

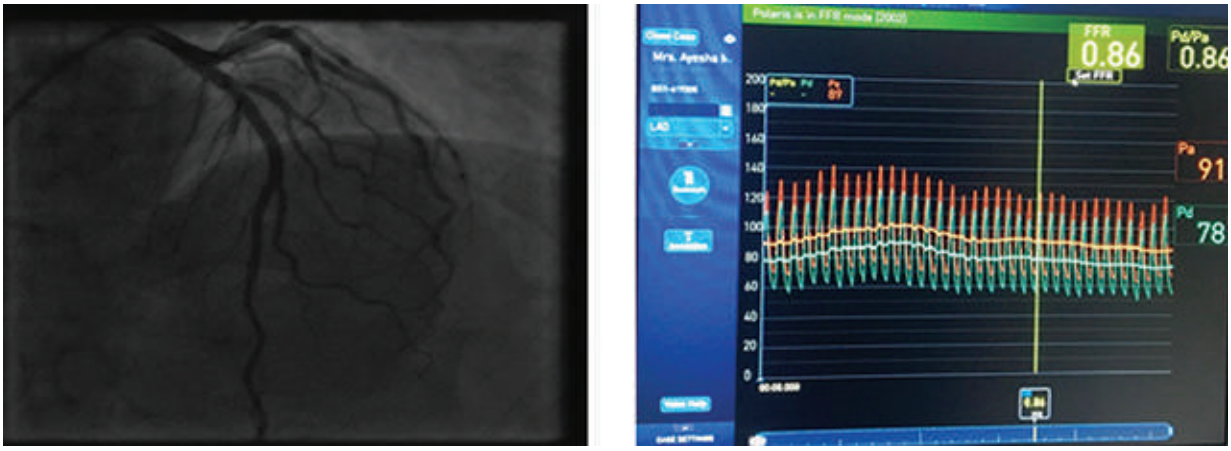


Fig.-5: *49 year old Female patient with Angina III, DM on OHG. CAG revealed 50% plaque in proximal LAD at the level of D1. Pressure wire guided physilogy study (FFR) found 0.86 as insignificant to stent, kept on medical management*

Discussion:

Intermediate coronary lesion is defined by quantitative coronary angiography as luminal narrowing with diameter stenosis >40 to <70%. Assessment and revascularization for intermediate coronary stenosis has been still under debate due to the limitations of coronary angiography itself. Coronary angiography, which evaluates lesions based on projection images, often under- and overestimates a lesion's severity because of contrast-dependent coronary dilation, high interobserver variability and poor ability to analyze plaque characteristics, especially in intermediate lesion.[16-17] Current guidelines recommend the use of fractional flow reserve (FFR) and intravascular ultrasound (IVUS) to guide and optimize revascularization in intermediate coronary lesion.[18-19]

FFR measurement is generally used as index for the functional severity of coronary artery stenosis, and well known as modality to decide whether to proceed with percutaneous coronary intervention (PCI) or defer in stable coronary disease.[20-21] On the other hand, IVUS has been widely used to optimize for PCI and assess accurate stenotic lesion and stent implantation.[22] The use of FFR and IVUS has been associated with improved clinical and procedural outcomes.[23-24]

In our present study of 50 patients with angiographic coronary intermediate lesion (>40% to <70%), we found that 62% (n=31) patient's lesions are insignificant by FFR or dFR and kept on medical treatment and 38% (n=19) patient had significance FFR/dFR and PCI done by drug eluting stents. Thus, in context to our patient population, FFR/dFR may aid in decision making of angiographic intermediate coronary lesion whether to stent or not to. Our results, very much consistent with our previous study of FFR role in determining lesion severity and subsequent PCI.[25]

In our present financial perspective where health expenditure is not covered by insurances, performing FFR/dFR might be an extra burden to patient and their family. In Korea, FFR measurement has been covered by the national insurance system under some limited cases like an intermediate lesion.[26] However, it was not well known about contemporary patterns of FFR and IVUS among patients undergoing PCI in Korea. They aimed to investigate the status of intervention using FFR and IVUS for intermediate stenotic lesions from Korean percutaneous coronary intervention (K-PCI) registry The rate of FFR measurement in intermediate lesions among PCI cases in K-PCI registry,[26] even though it did not have deferred-PCI cases, was quite low (11.5%). Despite a significant increase in the use of FFR and IVUS according to the national database and data from the registry,[27] FFR was only performed in 3.7% of patients (2.5% in ACS patients, 7.2% in non-ACS patients) according to the original report of the 2014 K-PCI registry.[26] Prolonged procedure time due to catheter reinsertion, the use of drug like adenosine, high cost of catheter, and poor reimbursement could be considered as factors that can be considered as causes for the low rates of FFR uses.[28] The low rates of FFR use was similarly observed in other country,

6.1% in US (among 61,874 attempted PCI for intermediate lesion between April 2009 and September 2010) and 8.2% in Japan.[29] There might have been more obstacles in using the FFR for intermediate coronary lesions due to strict national insurance coverage than in Japan, where application for national insurance coverage is relatively easy.

The DEFER (Deferral Versus Performance of PTCA in Patients Without Documented Ischemia) trial *Bech et al*,[30] demonstrated the safety of a deferral strategy in stenoses with an FFR >0.75, and PCI of FFR value >0.75 did not improve clinical outcome in comparison with patients deferred to medical treatment. Whereas the FAME (Fractional Flow Reserve Versus Angiography for Multivessel Evaluation) and FAME II (Fractional Flow Reserve Versus Angiography for Multivessel Evaluation 2) studies used an 0.80 FFR cutoff to investigate its value in guiding angioplasty.[6,20] This different choice of dichotomous cutoffs has its origin in early validation studies, which demonstrated that an FFR 0.75 has 100% specificity to identify stenoses with inducible ischemia in noninvasive tests, whereas an FFR of 0.80 has a sensitivity of more than 90% to exclude stenoses that generate myocardial ischemia.[31] Because both studies yielded positive results, the most reasonable explanation is that the main determinant of improved patient outcome in the physiology-guided arms was the substitution of angiography by FFR in the decision-making process, not the strict application of either 0.75 or 0.80 FFR cutoffs. Yet, in the post-FAME era, this diagnostic FFR gray zone is not currently incorporated in clinical guidelines.[32]

In a series of 265 patients assessing diagnostic performance of QFR, there was strong correlation with both FFR (r=0.863, P<0.001) and iFR (r=0.740, P<0.001) producing a diagnostic accuracy of 90.8% and 81.3% (P<0.001), respectively.[33] Despite these initial encouraging results, the uptake of QFR remains limited due to time constraints of calculating results in real-time, along with cost issues relating to software and licensing. However, pressure wire assessment of intermediate lesions is not without its limitations and/or contraindications. Certain patient and lesion specific factors may decrease the efficacy of FFR and/or iFR and therefore physiological assessment may not be suitable in all patients.

Patient specific characteristics may include patient contraindication to adenosine (e.g., profound bradycardia), concurrent or recent ischemia, significant left ventricular hypertrophy, severe aortic valve stenosis and situations where maximal hyperemia (in the case of FFR) cannot be achieved due to significant microvascular impairment. In these scenarios, FFR may over or underestimate lesion-specific ischemia and may not be as reliable in guiding revascularization.[34]

Lesion specific features may also hamper physiological assessment including aorto-ostial, left main and tandem lesions along with segments of diffuse disease.[35] These specific patient/lesion subsets may benefit from assessment with the use of intravascular imaging, chiefly intravascular ultrasound (IVUS) or optical coherence tomography (OCT). An intravascular ultrasound (IVUS)– derived minimal lumen area (MLA) < 4.0 mm^2, or minimal lumen diameter <1.8 mm have been shown to correlate with a FFR < 0.75,[36] and deferring intervention in intermediate coronary lesions based on MLA >4.0 mm^2 results in favorable clinical outcomes.[37] *Nam et al.*[38] found IVUS-guidance (MLA cut-off <4 mm2) resulted in a greater number of interventions compared to FFR-guidance (≤0.8) in 167 patients with intermediate coronary lesions, with no significant difference in MACE at 1 year. They also calculated that reducing the MLA cut-off to <3 mm^2 equalized the number of interventions between groups, concluding that both FFR and IVUS were suitable for use in guiding PCI in patients with intermediate lesions.

Eye-ball estimation of coronary angiographic lesion severity may be difficult to judge it as a cause to induce significant stenosis and clinical symptoms. Sometimes, experienced angiographers are unable to reliably assess lesion severity. *Lindstaedt et al*,[39] performed angiography and FFR in 51 patients with intermediate left main (40%–80% diameter narrowing) stenoses. Lesions were graded as significant or insignificant by four experienced interventional physicians blinded to the FFR results. Using FFR as the gold standard for significant lesions, the angiographers correctly classified the lesions in less than 50% of cases (regardless of FFR threshold of 0.75 or 0.8). Observer variability was high, resulting in only 29% of cases achieving unanimous appropriate classification. Diagnostic accuracy of angiographic left main assessment for detecting physiologically significant stenoses (FFR < 0.8) in only 69% patient. An updated data set with 213 patients from this group with 73 bypass-treated and 136 medically treated

patients had identical survival rates over 5 years of follow-up. In this population, a threshold of 0.8 was applied.[40]

In a very similar series of 38 consecutive patients with intermediate left main lesions (44% ± 10%). Of these, 20 (53%) had an FFR > 0.75 and were treated medically. One patient had surgery due to progression of the left main stenosis, and one experienced a myocardial infarction unrelated to the left main lesion. Of note, two of the 18 patients with an FFR < 0.75, in whom surgery was performed, died.[41]

Most recently, *Courtis et al*[42] studied 142 patients with mean 42% ± 13% left main stenosis. They used a threshold value of > 0.8 to assign a patient to medical therapy and < 0.75 to assign a patient to surgical therapy. Patients with an FFR between 0.75 and 0.8 were assigned based on FFR and other clinical findings. At 14 ± 11 months of follow-up, the 82 patients (58%) assigned to medical therapy had a major adverse cardiovascular event (MACE) rate of 13% and a cardiac death and infarction rate of 6%. In the 60 patients (42%) assigned to surgical therapy, MACE was 7% (P = 0.27) and cardiac death and infarction was 7% (P =0.70). Importantly, these investigators found that in the medically treated group, the incidence of MACE was higher in patients who had FFR measured using lower adenosine bolus doses (86 ± 57 µg) versus those in whom FFR was measured using higher adenosine doses (176 ± 102 µg). Patients with MACE more commonly had diabetes (55%) than those without MACE (21%).[42]

The FFR guided PCI reduces the need for revascularization of many of these lesions, some of the intermediate lesion may progress and might induced myocardial infarction. Some argue that for this reason, PCI of intermediate lesion might be beneficial.[43] Study showed PCI of intermediate lesion without physiological significance, does not improve major outcome such as the risk of death, myocardial infarction, or other major cardiovascular events.[20] Optimum medical management might result similar outcome.[44]

Very recently, *Puymirat E et al*,[45] has published FLOWER-MI trial that in patients with STEMI undergoing complete revascularization, an FFR-guided strategy did not have a significant benefit over an angiography-guided strategy with respect to the risk of death, myocardial infarction, or urgent revascularization at 1 year. However, given the wide confidence intervals for the estimate of effect, the findings do not allow for a conclusive interpretation.

Fractional flow reserve measurements were first introduced at the Mayo clinic in 1999, now considered as the gold standard for the detection of myocardial ischemia and is a valuable tool to guide percutaneous revascularization. The safety of functional evaluation is attributed to identification of ischemia-causing coronary stenosis, and its contribution to judicious decision-making revascularization.[20] Although, some of the patient with FFR 0.75-0.76 had PCI based on this clinically significant angina. FAME study showed that FFR guided PCI resulted in significant cost saving by reducing stent use of inappropriate stenting, avoidance hospitalization and MACE.[46]

Our present study supported the rationale for the use of FFR and or dFR in routine practice, even though the cost burden is an issue as our healthcare is not covered by health insurance. In this present non-randomized observational study, we were able to justify that 62% patients with approximate >40% to <70% angiographically intermediate lesion by eyeball estimation, were not inducing significant stenosis either by FFR or dFR and kept on medical treatment. Thus, thus these group of intermediate patients might has prevented from inappropriate stenting and subsequent consequences in future. Total 38% (n=19) patients of all angiographic intermediate lesion were found significant by physiological study (FFR/DFR) and underwent for PCI, if we left these patients on medical treatment, they might have developed life threatening MI and subsequent consequences.

Thus, FFR /dFR provides important physiologic information on the hemodynamic severity of intermediate coronary lesion and is helpful in the cardiac catheterization laboratory to determine whether PCI should be performed without stopping the procedure and sending the patient for a noninvasive stress test. Both FFR/dFR provides lesion specific index of functional severity of coronary stenosis that correlates with non-invasive tests of myocardial ischemia in patients with intermediate lesion.[47]

We also found, that among the significant group, all were male, indicating, male patients with intermediate coronary lesion are more prone to sustain MI if left untreated. Therefore, we recommend mandatory and routine FFR/dFR for male patients. Although sample number is not sufficient to conclude, but from our findings, we may conclude that not all intermediate

coronary stenosis needs to be stented unless it is proved by FFR or dFR.

Conclusion:

Angiographic assessment of intermediate lesions is unreliable and represents one quarter of all angiographies. Decision in treating these lesions by interventional procedures is difficult to judge. Therefore, pressure wire derived FFR or dFR are the key important tools in assessing the severity of myocardial ischemia in intermediate coronary lesions. Deferring intervention of intermediate coronary lesion with a FFR >0.75 or dFR > 0.85 may be associated with more favorable clinical outcome. we justified the needs of revascularization in significant FFR/dFR patients and kept on medical management of insignificant ones.

Thus, we recommend, FFR/dFR guided PCI for intermediate coronary lesion assessment prior intervention. Thereby, to reduce the needs for revascularization or inappropriate stenting of many of intermediate lesion and subsequent stent induced complications. Also, can avoid potential life-threatening MI and subsequent consequences of those intermediate lesion whose FFR/dFR were significant for PCI.

Limitation:

We already documented in our present study that FFR and dFR both are important tool to evaluate the clinical significance of ischemia, especially in a patient with angiographically intermediate coronary lesion. Major limitation is the cost of FFR/dFR, if it is significant then, in addition to stent cost, cost of FFR or dFR will put extra burden to patient and family as our health system is not covered by health insurance.

Future Perspective

We emphasize the uses of both FFR/dFR along with IVUS in assessing the significance of intermediate coronary lesion, and thus to guide which lesion may need PCI or keep on medical therapy. No doubt, this may provide better favorable outcomes in our patient perspectives of angiographic intermediate coronary lesion.

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Demographic profile of Covid-19 patients admitted in a Tertiary Care Hospital in the Northern Part of Bangladesh

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Abstract

Background: On the 8th March 2020 the first Covid-19 patient was detected in Bangladesh. Till then the number of Covid-19 patients is being increasing. As it is a newer pandemic, the demographic profile of Covid-19 patients may be helpful in the treatment arena of corona virus patients. Like other countries in Bangladesh, we have very limited research-oriented data regarding the social, demographic and/or clinical status of Covid-19 patients.

Aim of the study: The aim of this study was to fulfill a demographic profile of Covid-19 patients of the northern part of Bangladesh.

Methods: This was a cross-sectional study conducted at the Department of Cardiology in Rangpur Medical College Hospital, Rangpur, Bangladesh during the period from July 2020 to December 2020. In total 37 RT-PCR (Reverse Transcription Polymerase Chain Reaction) confirmed COVID-19 patients attended the mentioned hospital from 3rd July to 16th September, 2020 were selected as the study population. Data were collected from patients or their attendants through direct and/or telephonic interview by using a pre-designed structured questionnaire, after having appropriate consent. The statistical analysis was done by the Statistical Package for the Social Sciences (SPSS) version 22.0.

Result: In this study 70% participants were male whereas 30% were female the male-female ratio was 2.36:1. The mean (±SD) age of the participants was 56.57±15.96 years. The highest number of participants were from 51-60 years' age group which was 27.3%. Then 2.70%, 18.92%, 16.22%, 13.51%, 21.62% participants were from 21-30, 31-40, 41-50, 61-70 and > 70 years' age groups respectively. The majority portion participants were with normal body-weight (BMI: 18.5-24.9) which was in 57% and 43% were with overweight (BMI: 25.0-29.9). More than half of the patients were from Rangpur district which was 54%. The highest number of patients were with cough as a major symptom, which was among 51.35%. On the other hand, the highest number of participants were with IHD as a major comorbidity, which was in 64.86%.

Conclusion: As per the findings of this study, we can conclude that, middle aged people are mostly being affected by Covid-19 in Bangladesh. The affected number of male people are higher than that of female. Body weight or BMI is not associated with the possibilities of being affected by Covid-19. Cough shortness of breath and hypoxemia may be considered as the most potential clinical symptoms and IHD, HTN, DM and COPD may be considered as the most potential comorbidities to be considered for a Covid-19 patient.

Keywords: Demographic profile, Comorbidity, Covid-19 patients.

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Introduction:

On the 8th March 2020 the first Covid-19 patient was detected in Bangladesh. Till then the number of Covid-19 patients is being increasing. As it is a newer pandemic, the demographic profile of Covid-19 patients may be helpful in the treatment arena of corona virus patients. Like other countries in Bangladesh, we have very limited research-oriented data regarding the

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social, demographic and/or clinical status of Covid-19 patients. Several papers have been published regarding arthroscopically assisted. The novel coronavirus disease (COVID-19) is by far the most concerning outbreak of atypical pneumonia since the far less detrimental 2003 outbreak of severe acute respiratory syndrome (SARS)[1]. The COVID-19 pandemic has been declared an international public health emergency by the World Health Organization (WHO)[2]. As of July 1st 2020, the COVID-19 pandemic has infected over ten million people across the world, causing more than 5,00,000 deaths[3]. Experts are still uncertain of the trajectory of the COVID-19 pandemic, the projected number of cases and deaths, or to what extent quarantine measures will disrupt daily life[5]. The unpredictable nature of this situation and uncertainty regarding COVID- 19 can often trigger psychological distress and mental illness, including depression, anxiety, and traumatic stress[4]. Day by day the Covid situation in Bangladesh is worsening. COVID-19 has reached all 64 administrative districts in Bangladesh by July 1st 2020, causing over 145,000 cases and 1,874 deaths thus far[5]. High density of population, poor hygiene practices and poor economic conditions make the majority of the Bangladeshi population particularly vulnerable to this virus (Covid 19). Severe acute respiratory illness with fever and respiratory symptoms, such as cough and shortness of breath, comprise the main clinical presentations.[6] But unusual manifestations, such as patients without respiratory symptoms or only very mild symptoms are rising worldwide[7]. Understanding regional features are always important. A number of studies elaborating local epidemiological and clinical features have been published.[8] But we have very few statistical data regarding the demographic information of Covid patients. So, to fulfill a demographic profile of Covid-19 patients this study was conducted.

Objectives

General Objective

To assess the demographic profile of Covid patients in the northern part of Bangladesh.

Specific Objective

To collect information regarding the clinical symptoms and comorbidities of Covid patients.

Methods:

This was a cross-sectional study which was conducted at the Department of Cardiology in Rangpur Medical College Hospital, Rangpur, Bangladesh during the period from July 2020 to December 2020. In total 37 RT-PCR (Reverse Transcription Polymerase Chain Reaction) confirmed COVID-19 patients attended the mentioned hospital from 3rd July to 16th September , 2020 were selected as the study population. Data were collected from patients or their attendants through direct and/or telephonic interview and hospital register by using a pre-designed structured questionnaire, after having appropriate consent. Proper consents were obtained from every patient or from legal guardian by reading out the written informed consent according the revised Declaration of Helsinki. The protocol was approved by the ethical committee of the mentioned hospital. We collected demographic data (age, sex, address, contact history, etc.) and clinical data (symptoms on admission, comorbidities and their durations, etc.) The statistical analysis was carried out using the Statistical Package for Social Sciences version 22.0 for Windows (SPSS Inc., Chicago, Illinois, USA). Qualitative variables such as fever, cough etc. were expressed as frequency and percentage. Quantitative variables like age, durations, etc. were expressed as mean ± standard deviation and median. Test of significance was performed by unpaired t-test for quantitative variable and Chi square test for qualitative variables compared separately in different clinical presentation. In addition, multivariate logistic regression analysis of possible risk factors was done to determine the association with mortality by calculating odds ratio with 95% confidence intervals. A “p” value <0.05 was be considered as significant.

Result:

In this study among all the participants, 70% were male and the rest 30% were female. So, male participants were dominating in number and the male-female ratio was 2.36:1. The mean (±SD) age of the participants was 56.57±15.96 years. The highest number of participants of this study were from 51-60 years’ age group which was 27.3%. Then 2.70%, 18.92%, 16.22%, 13.51%, 21.62% participants were from 21-30, 31-40, 41-50, 61-70 and > 70 years’ age groups respectively. According to the BMI status of the participants, we observed, majority portion participants were with normal body-weight (BMI: 18.5-24.9) which was in 57% and the rest 43% were with overweight (BMI: 25.0-29.9). Among 8 district of Rangpur division, the division situated at the northern part of Bangladesh, our study people were found from 7 districts. The highest number, more than half of the

patients were from Rangpur district which was 20 in number (54%). On the other hand, the last one district, Panchagorh was free from Covid patient during the period of this study. In this study, in analyzing the clinical symptoms of the participants, we observed, the highest number of patients were with cough which was among 51.35%. Besides this, shortness of breath, hypoxemia/oxygen use, lower limb swelling and bleeding were found among 43.24%, 29.73%, 13.51% and 8.11% patients respectively. In this study, in analyzing the comorbidities among the participants we observed, the highest number of participants were with IHD which was in 64.86%. Besides this HTN, DM, COPD and CKD were found among 48.65%, 40.54%, 29.73% and 13.51% patients respectively. In this study, not a single case of death was occurred.

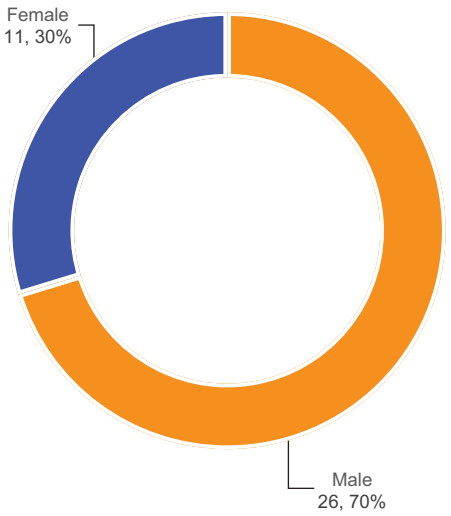


Fig.-1 : *Gender distribution of the participants (N=37)*

Table-I

Age distribution of participants (N=37)

Age (Years)	n	%
21 – 30	1	2.70
31 – 40	7	18.92
41 - 50	6	16.22
51 – 60	10	27.03
61 - 70	5	13.51
> 70	8	21.62

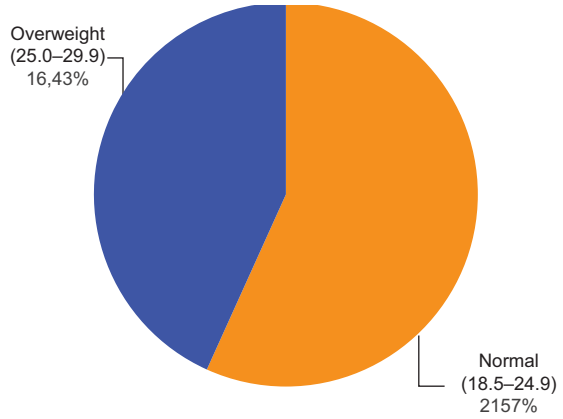


Fig.-2: *BMI distribution of the participants (N=37)*

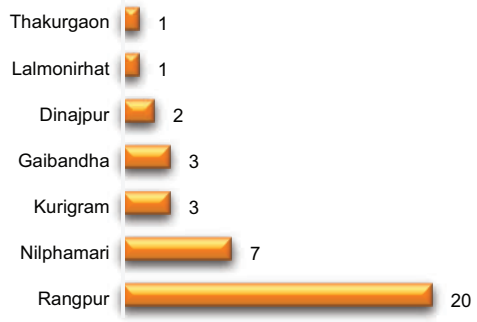


Fig.-3: *Living zone or district of the participants (N=37)*

Table-II

Symptoms distribution of participants (N=37)

Symptoms	n	%
Cough	19	51.35
Shortness of breath	16	43.24
Hypoxemia/Oxygen use	11	29.73
Lower Limb Swelling	5	13.51
Bleeding	3	8.11

Table-III

Comorbidities distribution among participants (N=37)

Comorbidities	n	%
IHD	24	64.86
HTN	18	48.65
DM	15	40.54
COPD	11	29.73
CKD	5	13.51

Discussion:

The aim of this study was to fulfill a demographic profile of Covid-19 patients of the northern part of Bangladesh. In total 37 RT-PCR (Reverse Transcription Polymerase Chain Reaction) confirmed COVID-19 patients attended the mentioned hospital between July 3 and September 16, 2020 were selected as the study people. Data were collected from patients or their attendants through direct and/or telephonic interview and hospital register by using a pre-designed structured questionnaire, after having appropriate consent. First COVID-19 cases were declared by Bangladesh in Dhaka city on 8 March, 2020[9], highest number of cases have been detected in Dhaka[10] and thus it is considered as the core of the disease transmission in Bangladesh. Hossain I, *et al*. have shown that most of the confirmed cases (about 48.9%) of Bangladesh reported that they lived in or had come to Dhaka within 14 days before the onset of illness or had been in close contact with any Dhaka resident.[11] In our study, among all the participants, 70% were male and the rest 30% were female. So, male participants were dominating in number and the male-female ratio was 2.36:1. The mean (±SD) age of the participants was 56.57±15.96 years. The highest number of participants of this study were from 51-60 years' age group which was 27.3%. Then 2.70%, 18.92%, 16.22%, 13.51%, 21.62% participants were from 21-30, 31-40, 41-50, 61-70 and > 70 years' age groups respectively. According to the BMI status of the participants of this study, we observed, majority portion participants were with normal body-weight (BMI: 18.5-24.9) which was in 57% and the rest 43% were with overweight (BMI: 25.0-29.9). Many of these findings matched that of Asia, e.g. China[6] (median age: 47 years; 41.9% female), India[12] (mean age 40.3 years, 66.7% male) and other reports from Bangladesh[13] (43% were in the age range of 21 to 40 years, female: male ratio 1:2.33). But studies from America[14] (median age, 63 years) and Europe[15] (Median age, 67.5 years) showed higher age of patients but same male preponderance. In this study, in analyzing the clinical symptoms of the participants, we observed, the highest number of patients were with cough which was among 51.35%. Besides this, shortness of breath, hypoxemia/oxygen use, lower limb swelling and bleeding were found among 43.24%, 29.73%, 13.51% and 8.11% patients respectively. The earliest reports from China described fever, dry cough, breathing difficulties (dyspnoea), headache and pneumonia as the typical clinical symptoms of COVID-19.[8]

Limitations of the study

This was a single centered study with a small sized sample. So, the findings of this study may not reflect the exact scenario of the whole country.

Conclusion:

As per the findings of this study, we can conclude that, middle aged people are mostly being affected by Covid-19 in Bangladesh. The affected number of male people are higher than that of female. Body weight or BMI is not associated with the possibilities of being affected by Covid-19. Cough shortness of breath and hypoxemia may be considered as the most potential clinical symptoms and IHD, HTN, DM and COPD may be considered as the most potential comorbidities to be considered for a Covid-19 patient. Among 8 districts of the northern division of Bangladesh more than 50% Covid-19 patients were found in Rangpur area as a single district.

Recommendations

For getting more reliable information, we suggest for conducting more studies in several places with larger sample size.

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Conflict of Interest: None Declared

Approval: From the respective Department

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Association of low Serum Magnesium level with in-hospital adverse outcome in patients with Acute Myocardial Infarction

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Abstract

Background: Acute Myocardial Infarction is the leading cause of morbidity and mortality throughout the world. Its prevalence among developing countries has increased significantly over the past two decades. Acute myocardial infarction is associated with electrolyte imbalance most commonly hypomagnesemia, which leads to adverse in-hospital outcome.

Objectives: To find out association between hypomagnesemia and in-hospital adverse outcome in patients with acute myocardial infarction.

Methods: Patients with acute myocardial infarction admitted in the department of Cardiology, DMCH, within the study period and who fulfilled the inclusion and exclusion criteria were taken as study sample. Informed consent was taken from all patients and then the patients were evaluated by detailed history, clinical examination and relevant investigations. Serum magnesium level was measured after admission. The sample population was Grouped into Group A(Acute myocardial infarction with normal serum magnesium) and Group B(Acute myocardial infarction with hypomagnesemia). Patients were followed up regularly till discharge or death for evidence of in-hospital mortality. Then the obtained data was analysed with SPSS 22.0.

Results: Among 110 patients of Acute MI, 44 patients were in Group A who had plasma magnesium level e"0.7 mmol/l and 66 patients were in Group B who had plasma magnesium level <0.7 mmol/l. Incidence of hypoagnesemia was 60% and more common in male. Male vs female percentage of hypomagnesemia were 61% vs 39%. Mean age was 54.16±11.72 yrs vs 57.52±10.59 yrs in group A vs group B. On admission serum magnesium level was 0.9218 vs 0.523 mmol/L(group A vs group B). The study showed that group B patients were more haemodynamically unstable and mean SBP and DBP were found 89.39±19.93 and 60.67±11.56 mm-Hg respectively. Troponin I was markedly increased in group B than A (i.e 4.7±1.79 vs 14.6±4.3 vs ng/ml). Adverse cardiac events such as cardiogenic shock (group A vs group B = 11.36% vs 28.27%) and ventricular arrhythmias(group A vs group B = 34% vs 72.73%) were also higher in group B than group A. Mean hospital stay for group B patient was higher than group A(6.78±0.85 vs 5.31±0.35 days). The study result showed that ventricular arrhythmia is negatively correlated with serum magnesium and the correlation coefficient was -0.541. It also showed that serum Magnesium is positively correlated with Potassium($r= 0.831$, $p<0.01$) and Calcium($r= 0.902$, $p<0.001$). Multiple logistic regression analysis showed that hypomagnesemia is an independent risk factor for ventricular arrhythmia. Its also shown that AMI patient with hypomagnesemia has adverse prognostic outcome and also increased mortality and morbidity.

Conclusions: This study showed that in patients with acute myocardial infarction, hypomagnesemia has adverse in-hospital outcome in comparison with acute myocardial infarction with normal serum magnesium.

Keywords: Hypomagnesemia,In-Hospital adverse prognostic outcome, Acute myocardial infarction.

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Introduction:

Myocardial infarction (MI) can be recognised by clinical features, including electrocardiographic (ECG) findings, elevated values of biochemical markers (biomarkers) of myocardial necrosis, and by imaging, or may be defined by pathology. It is a major cause of death and disability worldwide[1]. New ST elevation at J point in two contiguous leads with cut-points: e"1mm in all leads other than leads V2-V3 where the following cut points apply: ≥2mm in men ≥40 years; ≥2.5mm in men <40 years or ≥1.5 mm in women[1].

Acute myocardial infarction is associated with electrolyte imbalance most commonly hypomagnesemia and hypokalaemia. Both are associated with ventricular arrhythmia which can lead to increase hospital mortality and morbidity[2].

Magnesium is the 2nd most common intracellular cation after potassium and 4th abundant cation in the body .The total magnesium content in the body of an average adult is around 25 Gm or 1000 mmol. About 60% of the body reserve of magnesium is found in the skeletal bone mass, about 20% is in muscle and another 20% is in soft tissues and liver. Normal plasma Magnesium concentration is from 1.7 to 2.5 mg/dl[3].

Hypomagnesaemia is present in acute myocardial infarction (AMI) as shift of magnesium from extra cellular to intracellular compartments occur as it is taken up by adipocytes after catecholamine induced lipolysis and combined with soaps formed by free fatty acids[4,5]. Although the total body Mg contents may not change with the onset of AMI, extra cellular Mg declines markedly, especially over the first 24 to 48 hours after the onset of AMI[5,7]. Hypomagnesaemia in the initial phase of post AMI period is very critical, as ventricular tachyarrhythmia sudden cardiac death and re-infarction are the usual outcome.[4,5,6] Magnesium has been suggested as a possible intervention to be used in AMI since the early 1960s mainly because it was thought to be an antiarrhythmic agent, although no studies have conclusively shown this to be the mechanism of action for magnesium in reducing mortality.[6]

Magnesium, a divalent cation, is a physiologic calcium antagonist that inhibits calcium entry into vascular smooth muscle cells.[12,13] Furthermore, magnesium promotes coronary artery vasodilation and peripheral systemic arterial vasodilation, thereby increasing coronary blood flow and reducing afterload.[14,15] Magnesium may reduce ischemia and decrease sinus node and atrioventricular conduction.[16] Because of its ability to inhibit myocardial cell sodium and calcium influx as well as potassium egress, magnesium may diminish infarct related reperfusion injury and myocardial stunning, thereby limiting infarct size.[17]

Methods:

Prospective cross sectional study was conducted in the Department of Cardiology, DMCH, Dhaka, Bangladesh from January 2017 to Dec 2017. All the patients with Acute Myocardial infarction with or without hypomagnesaemia admitted in the department of Cardiology, DMCH, within the study period and who fulfilled the other inclusion and exclusion criteria was taken as sample.

Inclusion criteria:

All the Acute Myocardial infarction patients with or without hypomagnesemia presenting to CCU in DMCH within the study period. Acute coronary syndrome was included STEMI, non-STMI.

Exclusion criteria:

- Patients with history of any previous MI.
- Patients with history of PCI or CABG.
- Patients with any valvular heart disease, congenital heart disease, primary myocardial or pericardial disease, acute on chronic heart failure
- Patients with other co-morbid .conditions like COPD, end stage renal disease, nephrotic syndrome, liver disease, chest infection and bronchogenic carcinoma, stroke, hypothyroidism
- Post diarrhea MI(within last 3 days)
- Patients on Total parenteral nutrition
- Patients unwilling to give consent.

Ethical Consideration:

Prior to the commencement of this study, the research protocol was approved by the Research Review Committee of Department of Cardiology and the Ethical Review Committee of DMCH, Dhaka. The aims and objective of the study along with its procedure, alternative diagnostic methods, risk and benefits was explained to the patients in easily understandable local language and then informed consent will be taken from each patient. It was assured that all records was kept confidential and the procedure will be helpful for both the physician and patients in making rational approach regarding management of the case.

Statistical Analysis

All statistical analysis was performed using the statistical package for social science (SPSS) program, version 22 for Windows. Continuous parameters was expressed as mean ±SD and categorical parameters as frequency and percentage. Comparisons between groups (continuous parameters) was done by Student's t test. Categorical parameters was compared by Chi-Square test. Correlation analyses was done by Pearson correlation coefficient. The significance of the results as determined in 95.0% confidence interval and a value of p <0.05 was considerd statistically significant.

Methods of Data Collection:

All patients of Acute Myocardial infarction admitted in the department of cardiology, Dhaka Medical college hospital within the study period who fulfilled the inclusion and exclusion criteria was taken as sample. Information from the patients and relatives were collected through preformed proforma. Patients were evaluated based upon History, Clinical examination and investigations. After Admission Serum magnesium level was done concurrently Serum calcium, Serum potassium was done to exclude other confounding variables, Cardiac biomarkers were done to differentiate between STEMI, NSTEMI. Occurrence of Ventricular arrhythmia were counted on repeated regular ECG tracing on twice daily when admission in Hospital. Then occurrence of Ventricular arrhythmia of Acute Myocardial infarction patients with hypomagnesaemia and those without hypomagnesaemia were compared and analyzed. After compilation of collected data collection from all patients, statistical analysis were performed using the statistical package for social science(SPSS) program, version 22 for Windows. The confidentiality of the patient was maintained properly when observations were compiled by the investigator. Informed written consent were taken from each patient. Continuous parameters was expressed as mean±SD and categorical parameters as frequency and percentage. Study subjects were grouped into 2 groups, patients with hypomagnesemia (Plasma magnesium level <0.7 mmol/L as Group B and patients with normal plasma magnesium level ≥0.7 mmol/L as Group A. Comparisons between groups (continuous parameters) was done by Student's t test. Categorical parameters was compared by Chi-Square test. Correlation analyses was done by Pearson correlation coefficient. The significance of the results as determined in 95.0% confidence interval and a value of p <0.05 was considered statistically significant.

Results :

This prospective observational study was conducted in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka, over a period of one year from Jan 2017 to Dec 2017. The main objective of the study was to find out the association between low serum magnesium and ventricular arrythmia in patients with acute myocardial infarction. A total of 110 adult patients with acute myocardial infarction(both STEMI and NSTEMI) admitted in the department of cardiology, DMCH within the study period were included in the study. The study population was divided into two groups: Group A included AMI with normal serum magnesium level (≥0.7 mmol/dl) and Group B included AMI with low serum magnesium level (<0.7 mmol/dl). These patients were followed up during hospital stay to evaluate the prelevance of different types of ventricular arrhythmia(Frequent PVC, Idioventricular rhythm, Sustained VT, non sustained VT, VF). Appropriate statistical techniques were used for data analysis. Results were presented with tables and graphs where required.

Table-I

Distribution of study subjects by age (n=110)

Age in years	Group A(n=44)		Group B(n=66)		P value
	No.	%	No.	%	
31-40	9	20.45	7	10.61	
41-50	7	16	10	15.15	
51-60	15	34	15	22.73	
61-70	8	18.18	27	40.91	
>70	4	11.37	7	10.6	
Total	44	100.0	66	100.0	
Mean±SD (Range mgm)	54.16±11.70 (42-66)		58.45±9.47 (46-68)		<0.06^{ns}

P value reached from unpaired t-test.

Group A : Acute myocardial infarction patients with normal serum magnesium

Group B : Acute myocardial infarction patients with hypomagnesemia

ns : not significant

Above table shows the age distribution of study subjects. It shows that maximum patients of Group A is in the age group of 51-60 yrs 15(34%) whereas maximum (27 or 40.91%) in Group B is in 61-70 yrs. The mean age is 54.16±11.70(yrs) vs 58.45±9.47(yrs) in Group A vs B. The mean difference between two Groups were statistically not significant (p<0.05).

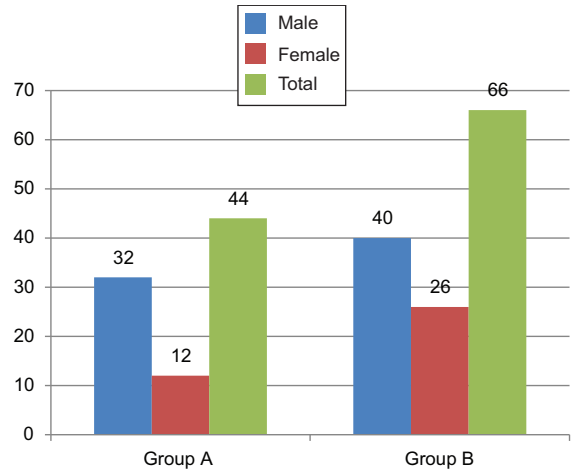


Fig.1: *Bar diagram showing distribution of the sex between two groups (n=110)*

Fig.1: Bar diagram showing distribution of the sex between two groups. It shows Male is 32(72.7%) vs 40(60.6%) respectively in Group A vs Group B, Similarly Female is 12(27.3%) vs 26(39.3%) respectively in Group A and Group B. It shows, the difference between this two groups is not statistically significant (p<0.05)

Above table shows that mean troponin I is 4.7±1.79 vs 14.87±4.3(ng/dl) respectively in group A vs group B. Serum creatinine is 0.807±0.168 vs 0.92±0.185 respectively in group A vs group B. Serum Potassium is 3.84±0.16 vs 2.54±0.41 respectively in group A vs group B. Serum Calcium is 8.71±0.3 vs 6.52±0.55 respectively in group A vs group B. The mean difference of troponin I, serum Potassium, Calcium between group A and group B is statistically significant (p<0.05) but mean serum creatinine between this two groups is not statistically significant (P>0.05).

Table shows the distribution of adverse cardiac events namely cardiogenic shock, asystole, ventricular arrhythmia, post MI angina and Death. It shows that Ventricular arrhythmia and Cardiogenic shock between group A and B is statistically significant (P<0.05) whereas Post MI angina, asystole and Death between group A and B are not statistically significant (p>0.05).

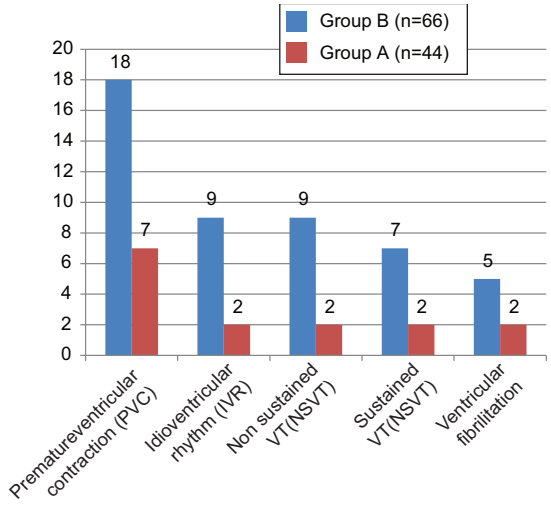


Fig.-2: *Bar Graph Showing Frequency distribution of Ventricular Arrythmia in Between Group(n=110)s*

Table-II

Comparison of biochemical parameters between two groups (n=110)

Laboratory parameters	Group A(n=44) Mean±SD	Group B(n=66) Mean±SD	P value
Troponin- I (ng/ml)	4.7±1.79	14.87±4.3	0.001^{s}
Serum creatinine (mg/dl)	0.807±0.168	0.92±0.185	0.511^{ns}
Serum Potassium(mmol/l)	3.84±0.16	2.54±0.41	0.001^{s}
Serum Calcium(mg/dl)	8.71±0.3	6.52±0.55	0.001^{s}

Data were expressed as mean±SD
P value reached from unpaired t-test.
Group A : Acute myocardial infarction with normal Serum magnesium
Group B : Acute myocardial infarction with hypomagnesemia
ns : not significant
s : significant
n : number of patients

The figure shows that premature ventricular contraction(PVC) is most frequent arrhythmia in both group - 7 (46.66%) vs 18(39%) respectively in group A vs Group B. Group B has more incidence of Ventricular Arrhythmia than group A, respectively 48(72.72%) vs 15(34.09%). This difference between two groups is statistically significant (p<0.05).

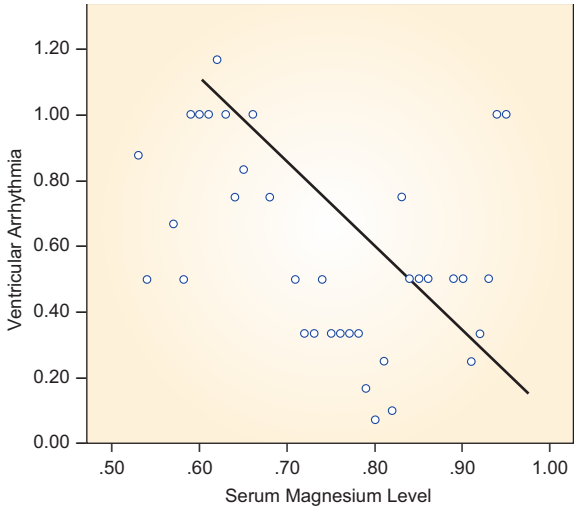


Fig.-3: *Showing correlation between serum magnesium level with occurrence of ventricular arrhythmia in Group B(n=66)*

The scattered diagram shows corerelation between ventricular arrhythmia and serum magnesium level. It shows serum magnesium level is negatively correlated with ventricular arrhythmia with correlation co efficient ®-.541

This table shows the mean duration of hospital stay which is 3.31±0.51 vs 6.18±0.95 days respectively in group A and group B. The mean difference between two groups is statistically significant (p<0.05).

This table shows that there is a significant negative correlation between ventricular arrhythmia with serum magnesium (r= -0.541, p<0.001), serum potassium (r= -.436, p<0.01), serum calcium (r= -0.530, p<0.001). It also shows Serum magnesium is negatively correlated with serum calcium and serum potassium.

Table V: shows logistic regression analysis of Ventricular arrhythmia with independent risk factors. Out of 10 variables, only hypomagnesemia was observed as independent risk factor (OR-136,p-0.001), Other 9 variables such as Obesity, Systolic BP, Diastolic BP, Cardiogenic shock, Troponin I, Hospital stay, Serum potassium, Serum calcium and Heart Failure, none was observed as independent predictors for developing ventricular arrhythmia (OR-1,p-0.275; 2.07,0.493; 263,0.1; 2,0.06; 0.7,0.73; 0.9,0.14; 8.38,0.3; 1,0.456).

Table-III

Comparison of in-hospital adverse cardiac events between the study groups (n=110)

Adverse cardiac events	Group A (n=44)		Group B (n=66)		Pvalue
	N	%	N	%	
Cardiogenic shock					
Yes	05	11.36	18	27.27	0.044^{s}
No	39	88.66	48	72.73	
Asystole					
Yes	04	9.09	08	12.13	0.6195^{NS}
No	40	90.91	58	87.87	
Ventricular Arrhythmia					
Yes	15	34.1	48	72.73	0.001^{s}
No	29	65.90	18	27.27	
Post MI angina					
Yes	10	22.72	10	15.15	0.3129^{ns}
No	34	77.28	56	84.85	
Death					
Yes	01	2.27	06	9.09	0.1512^{ns}
No	43	97.73	66	90.91	

P value reached from unpaired t-test.
Group A : Acute myocardial infarction with normal Serum magnesium
Group B : Acute myocardial infarction with hypomagnesemia
s=significant;
ns=not significant

Table-IV

Comparison of study patients by hospital stay (N=110)

Hospital stay (days)	Group A(n=44) Mean±SD	Group B(n=66) Mean±SD	P value
Duration of hospital stay (Range)	3.31±0.51 (3-4) days	6.18±0.95 (5-7) days	<0.002[s]

Data were expressed as mean±SD
P value reached from unpaired t-test.
Group A : Acute myocardial infarction with normal serum magnesium
Group B : Acute myocardial infarction with hypomagnesemia
s : significant
n : number of patients

Table-V

Correlation between Ventricular arrhythmia and other Biochemical parameters

	R	p-value
S. Magnesium	-0.541	<0.001[s]
S. Potassium	-0.436	0.010[s]
S. Calcium	-0.530	<0.001[s]

r =Pearson's correlation co-efficient.
s= significant (p< 0.05).

Table-VI

Multivariate Logistic regression analysis of Ventricular arrhythmia with independent risk factors in patients with Acute Myocardial Infarction.

Independent risk factors	'	S.E.	P value	Odd ratio	95% C.I. for EXP(B)	
					Lower	Upper
Obesity	-0.140	0.128	0.275	1.00	-0.485	0.007
Sytolic BP	-0.16	0.03	2.07	0.493	-0.011	0.01
Diastolic BP -0.02	-0.09	0.06		1	0.0543	-0.003
Heart failure	-0.159	0.05	0.06	2	-0.281	-0.064
Cardiogenic shock	0.02	0.106	0.79	0.723	-0.174	0.25
Troponin I ng/ml	0.023	0.015	0.14	0.971	.932	1.010
Hospital stay	-0.067	0.044	0.87	1.066	.974	1.167
S. Potassium	-0.020	0.196	0.307	8.388	1.268	55.48
S. Calcium	-0.2	0.5	0.456	1	-0.33	0.2
S. Magnesium	16.380	5.90	0.001	136	136	1381

Discussion:

This prospective comparative observational study was conducted in the coronary care unit (CCU) of Department of Cardiology, Dhaka Medical College Hospital, Dhaka, over a period of one year from Jan 2017 to Dec 2017. The main objective of the study was to find out the association of ventricular arrhythmia with low serum magnesium in patient with acute myocardial infarction admitted in Dhaka Medical College Hospital during this time period.

For this purpose 110 newly diagnosed acute myocardial infarction patients were included according to exclusion and inclusion criteria. The study population was divided into two groups based on serum magnesium level. Those with Acute Myocardial infarction with normal magneseium was in Group A

which includes 44 patients. In group B, 66 patient were included those with acute myocardial infarction with hypomagnesemia.

It was found that hypomagnesemia occurs in 86.66% in acute myocardial infarction[18], similar study found Hiralal Murmu[19]and it was 80% whereas our study showed it was around 60%(66). In our study male were more common in both group. It observed that age(years) incidence was 54.16±11.70 yrs and 58.45±9.47 yrs in between group A and group B respectively .

Our study showed male have higher incidence of hypomagnesemia 40(60.60%) vs 26 (39.40%), similar result observed by Jaffery, et al. study[20], the M:F ratio was were 2.66:1 (34 vs 16). i.e male have higher incidence than female. In our study it also showed that total male, female was 76 vs 34. Sex incidence was also higher in Male than female. In different study conducted by Akila, et al.[21]showed that male are predominance to hypomagnesemia than female and it male female ratio was 5.25:1. which supports our study.

Our study have shown serum magnesium in group A and group B was 0.9218±0.1 and 0.523±0.08 mmol/L respectively. The difference between two groups is statistically significant(<0.05). Other study conducted by Hiralal Murmu[19], showed the similar result, it was 2.2 meq/L(1.1±0.175 mmol/l) for control group and 1.01(0.55±0.07) for AMI group. Jaffery, et al.[20] also showed that mean serum magnesium level was 1.24±0.48 but no significant difference in between sex, which supports our study. Whereas Nasim B, et al.[8], showed mean serum magnesium was 1.7-2.5 meq/L(0.85-1.25 mmol/L) on the other hand Chowdhury, et al.[18] showed it was 0.7±0.13 mmol/L. In different study conducted by Akila et al[21] and it was found serum magnesium level in hypomagnesemia group was 1.65±0.26 mg/dl(0.82±0.16 mmol/dl).

In our study It has shown that group B patient had significant low DBP(mm-Hg) and it was 56.67±11.56(DBP). No such evidence found in any articles regarding this issue.

Akila et, al.[21] showed smoking, DM, HTN, CVD, Obesity, Dyslipidaemia are risk factors for cardiovascular disease whereas our study shown the similar result which supports our study. In another study conducted by Jaffrey, et al.[20] showed similar risk factors HTN. Obesity, CVD, DM, Smoking, Dyslipidaemia and Impaired Glucose tolerance test.

Our Study showed raised serum troponin in both group, yet that group B has triple fold rise of serum troponin than that of group A. The serum troponin level was 4.7±0.79 ng/ml and 14.87±4.3ng/ml in group A and group B respectively. This difference between two groups is statistically significant (p<0.05). It was also observed that raised troponin also associated with severity of hypomagnesemia.

Similarly serum Potassium between two group A and B was 3.84±0.16 vs 2.54±0.41 mmol/dl, The difference between two group was statistically significant (p<0.05). The difference of serum calcium level between two group was statistically significant.

Our study also showed that cardiogenic shock 05(11%) in group A and 18(27%) in group B. Group B patient has more shock event than group A. The difference between group is statistically significant (p<0.05). Our study also showed ventricular arrhythmia more in group B than group A respectively 15(34%) vs 48(73%). The Difference between two groups is statistically significant (p<0.05). Other adverse cardiac events such as asystole, post MI angina, Death were not statistically significant in between group. It was observed in our study that hypomagnesemia is positively correlated with ventricular arrhythmia. The study also showed hypokalaemia ,hypocalcaemia and hypomagnesemia also positively correlated with each other.

Our study shows that group B has three times more chance of occurrence of ventricular arrythmia which is 15 vs 48 in between group A vs group B. The difference is statistically significant(p<0.05). Similar study found by Hiralal Murmu[19]. He showed that hypomagnesemia group has 2-3 times more chances of ventricular arrythmia than control group which supports our study.

Ceremuynski L[22], showed that the mean plasma magnesium concentration was 1.83mgl/dl(0.76mmol/L) in patient with no abnormal rhythm, 1.68mg/dl(0.7 mmol/L) in those with multifocal premature complex and 1.5 mg/dl(90.65mmol/l) in those with unstained ventricular tachycardia. That is hypomagnesemia is positively correlated with occurrence of ventricular arrythmia. Similar results shown by our study which is hypomagnesemia is negatively correlated(r=- 0.543, p<0.001) with occurrence of Ventricular arrythmia.

Hiralal Murmu[19], showed that after Acute myocardial infarction hypomagnesemia occurs within 48hrs in most(80%) cases and which is the causes of ventricular arrythmia. Woods et al[24], showed in different study that this ventricular arrythmia improves after IV magnesium infusion. Multivariate logistic regression analysis showed that out of nine variable such as Obesity, SBP, DBP, Heart failure, Cardiogenic shock, Troponin I, Hospital stay, Serum potassium and Serum calcium , none was observed as idependent risk factor for ventricular arrhythmia.

Our study also showed that hypomagnesemia is also associated with hypokalaemia(r=0.831,p<0.01), Multivariate regression analysis strongly disagree hypokalaemia is not independent risk factor for Ventricular arrythmia. Similar study was found by Chowdhury, et al.[18]. In Chowdhury et al[18] showed that mean level of Mg(mmol/L) was 0.59±0.09 in group I and 0.67±0.07 in group II. The mean level of serum K(mmol/L) in group I and group II were 3.28±0.45 and 3.63±0.43 respectively. Our study showed the similar data. The serum potassium(mml/L) level in between group A and B respectively 3.84±0.16 vs 2.54±0.41. This mean difference between group is statistically significant(p<0.005). Similar result supported by Dyckner, et al.[24] and Kafka, et al.[25]. But no mechanism is yet explained why hypomagnesemia and hypokalaemia occurs concurrently Chowdhury, et al.[18]

Our study showed hypomagnesemia is strongly positively correlated with ventricular arrhythmia and the association was statistically significant.It also showed that patients with hypomagnesemia have adverse in-hospital outcome.

Conclusion:

This study showed that there is association between hypomagnesemia and adverse in-hospital outcome in patient with acute myocardial infarction in comparison to normal serum magnesium. It is also showed hypomagnesemia increase the risk of mortality and morbidity. So, its treatment with IV magnesium is necessary to reduce mortality and morbidity. Hypomagnesemia patient has adverse in-hospital prognostic outcome in comparison to normal serum magnesium.

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Spontaneous Coronary Artery Dissection (SCAD): Case Series with Review of Literature

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Key words: *Spontaneous Coronary Artery Dissection, Medical management, Percutaneous Coronary Intervention.*

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SCAD is defined as a separation of the layers of an epicardial coronary-artery wall by intramural hemorrhage, with or without an intimal tear. This condition is not associated with atherosclerosis, iatrogenic injury, or trauma.[1] It was first described by Pretty in 1931[2]. SCAD is underdiagnosed and sometimes mismanaged as atherosclerotic ACS. Accurate diagnosis is important to provide early supportive care and ensuring that invasive strategy is used only for a selected group of patients. PCI has been associated with more complications and has suboptimal outcome in SCAD. With conservative management most of the SCAD lesion heals with time.[3,4]

The true incidence of SCAD is unknown, largely because most patients fall into the lowest risk categories for conventional atherosclerotic disease leading to underdiagnosis. Accurate angiographic diagnosis can be challenging and reported rates of SCAD diagnosis from angiographic registries (0.2%–0.7% of angiograms; 2.0% of ACS angiograms) are variable. Recent series have shown that SCAD may be a cause of up to 4% of overall ACS cases, up to 35% of MIs in women ≤50 years of age,' and is the most common cause of pregnancy-associated MI (43%).[5,6]

Two models have been de-scribed for the development of SCAD. One proposes that the primary pathological event is a disruption in the vessel wall (intimal tear, "inside out model"), which allows blood from the true lumen to enter and generate a false lumen. Another theory proposes that the primary event is a spontaneous hemorrhage arising from the rupture of vasa vasorum within the vessel wall ("outside in model").[7,8] Fibromuscular dysplasia (FMD) has been described in several histopathological case reports of SCAD.[9] SCAD is thought to be arising by interplay of underlying arteriopathies, genetic factors, hormonal influences, or systemic inflammatory diseases often precipitated by stressors including covid infection.[10-12]

Anatomically, SCAD has a preference for mid to distal coronary segments, and left anterior descending is the most commonly vessel involved, followed by circumflex and right coronary arteries. Multivessel affection is rare, in only <10% of cases are the proximal segment of artery is affected.[13,14]

Here we present a series of 5 cases of SCAD followed by literature review.

Case 1

25 years lady married for 2 years with history of irregular menstruation taking hormonal therapy from last 4 months presented with anterior wall STEMI of 10 hours duration. She was taken for primary PCI. Her CAG revealed complete occlusion of Proximal LAD. POBA was done with LAD. Final Result showed slow flow with complete occlusion of distal segment. Her hospital course was uncomplicated. Repeat CAG after 3 months shows normal flow in LAD suggesting the invasive strategy have suboptimal result and most SCAD heals with time.

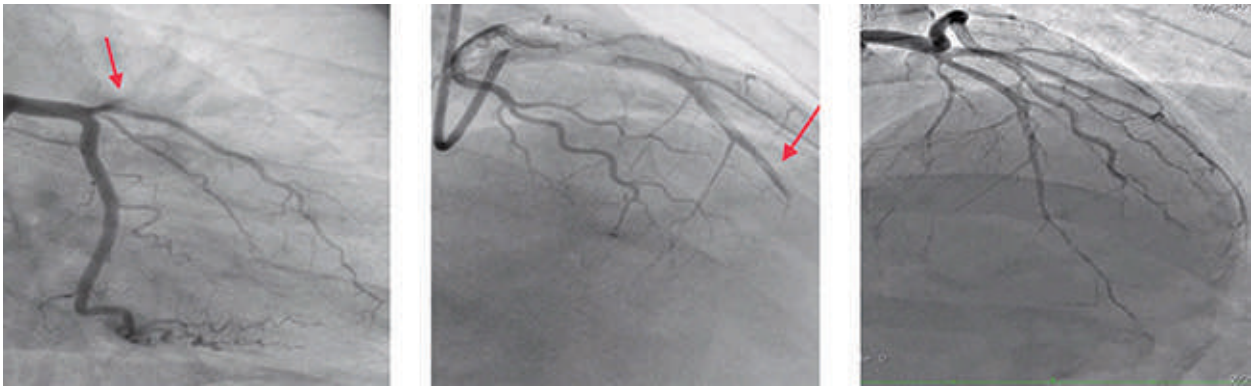
Case 2.

39 years lady with history of superior sagittal sinus venous sinus thrombosis presents with recurrent epigastric pain. On evaluation she was found to have anterior wall MI. Her CAG revealed SCAD of proximal LAD and First diagonal branch. PCI was done with

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RAO caudal view showing complete occlusion of proximal LAD(stump is visible in figure A) After PCI, slow flow is clearly visible with complete occlusion on distal LAD (arrow on figure B), Repeat angiogram after 3 months demonstrate healing of SCAD lesion normal flow on LAD.

DES from ostial LAD. (Figure 2). Further workup showed positive AntiPhospholipid antibody and was under treatment for same. After 2 years of check angiogram was done which revealed completely occluded LAD stent along with narrowed ostium of LCX and high OM1. This is Probably due to high predisposition for Stent thrombosis in SCAD.

Case 3

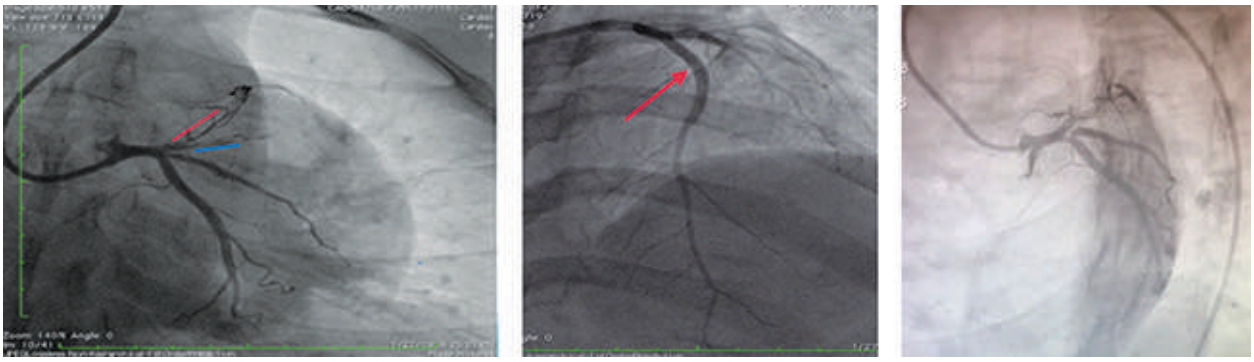
29 years lady who was on inj. Depo provera(Progesterone) for 10 yrs and switched to combined oral contraceptive since one month presented with anterior myocardial infarction of 7 hours duration. she does not have other atherosclerotic risk factors. CAG revealed Type 1 SCAD on distal LAD. (Figure 3). She was managed conservatively with DAPT and beta blocker. She had uneventful hospital stay and was discharged 5 days later without any complications.

Case 4

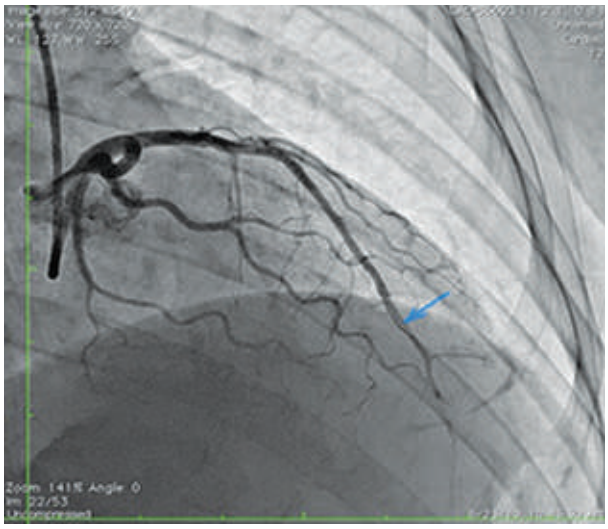
56 years old obese male presented with Shortness of breath without chest pain after taking bath at mansarovar (4500 metres). He has no other conventional atherosclerotic risk factors. He was diagnosed to have anterior wall MI. CAG revealed type 1 SCAD in LAD and Diagonal branch with complete occlusion of mid LAD. PCI (POBA)was done on LAD .He had uneventful recovery and was discharged.(figure 4)

Case 5

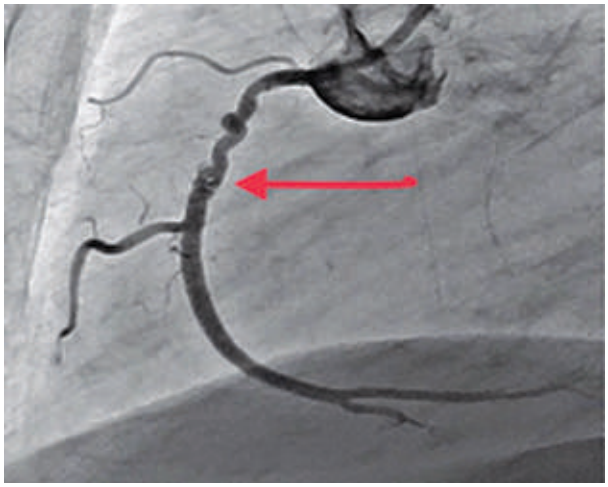
36 years female without conventional atherosclerotic risk factor presented with acute inferior wall STEMI. Her CAG revealed tortuous proximal RCA with SCAD. She was managed conservatively without revascularisation. She improved clinically and was discharged on 5th day.(figure 5)



AP caudal image shows there is multiple lumen with decreased flow in LAD (red line) and diagonal branch (green line) of figure A, the proximal LAD was stented with DES with achievement of good forward flow i(figure B). After 2 years of follow up in check angiogram was done which revealed stent thrombosis along with partial occlusion in ostium of LCX and high OM1 .







Literature review

The clinical presentations and severities of SCAD is wide; ranging from asymptomatic to cardiac arrest and sudden death. patients who survive and present in hospital almost always experience ACS and increased levels of cardiac enzymes (26% to 87% of with STEMI, and 13% to 69% present with NSTEMI in differernt series). About 5% of patients present in cardiogenic shock. Ventricular arrhythmias or sudden cardiac death account for 3% to 11% SCAD presentation. Symptoms are similar to atherosclerotic ACS, with chest pain being the most common. [14,15]

Patients with SCAD are at risk of misdiagnoses and underdiagnosis. There are chances that patients may be discharged from emergency department because most of them are younger female without conventional atherosclerotic risk factors even in the presence of typical symptoms. Therefore there should be high clinical index of suspicion for SCAD especially when younger women (especially <50 yrs or peripartum state) with no or minimal conventional atherosclerotic risk factor present with typical chest pain or other cardinal symptoms especially after a recent intense exercise or emotional stress. They may have relevant connective tissue disorder, FMD or systemic inflammatory disorders.

Natural history of SCAD:

Majority of SCAD lesions heals angiographically after conservative management (95% in 30 days).[26] A minority of patients had persistent dissection. Among conservatively managed patients, recurrent MI may develop in 5-10%, mostly related to extension of dissection within 7 days after an acute episode. The majority of these patients require emergency revascularization. So, inpatient monitoring for an extended period is typically recommended.[25]

Diagnosis

Once SCAD is suspected, coronary angiography should be performed as early as possible, especially in the setting of STEMI. On angiography there may be no evidence of typical atherosclerotic lesion. Accurate diagnosis of SCAD at presentation is important because management and investigation are different from those for atherosclerotic forms of coronary artery disease.[3,15]

Traditionally SCAD was diagnosed on the basis of the presence of multiple radiolucent lumens and extraluminal contrast staining or intraluminal ûlling defects. However, this “pathognomonic” appearance is present in only a small proportion (30%) of SCAD cases. Newer angiographic classification for SCAD has been commonly adopted.[16]

There are 3 main types. Type 1 is the typical appearance that is characterized by multiple coronary artery lumens associated with the longitudinal filling defect. Type 2 is the most commonly encountered entity where patients develop diffuse (>20mm) vessel stenosis which may or may not extend upto vessel end.Type 3 disease is usually marked with focal vessel stenosis. Both type 2 and type 3 show elusive changes in the caliber, signifying a hematoma in the wall of the involved vessel. Intravascular imaging is considered mandatory to confirm type 3 SCAD as it cannot be differentiated from atherosclerosis as conventional angiography is basically a lumenography and does not speciûcally image the arterial[16].

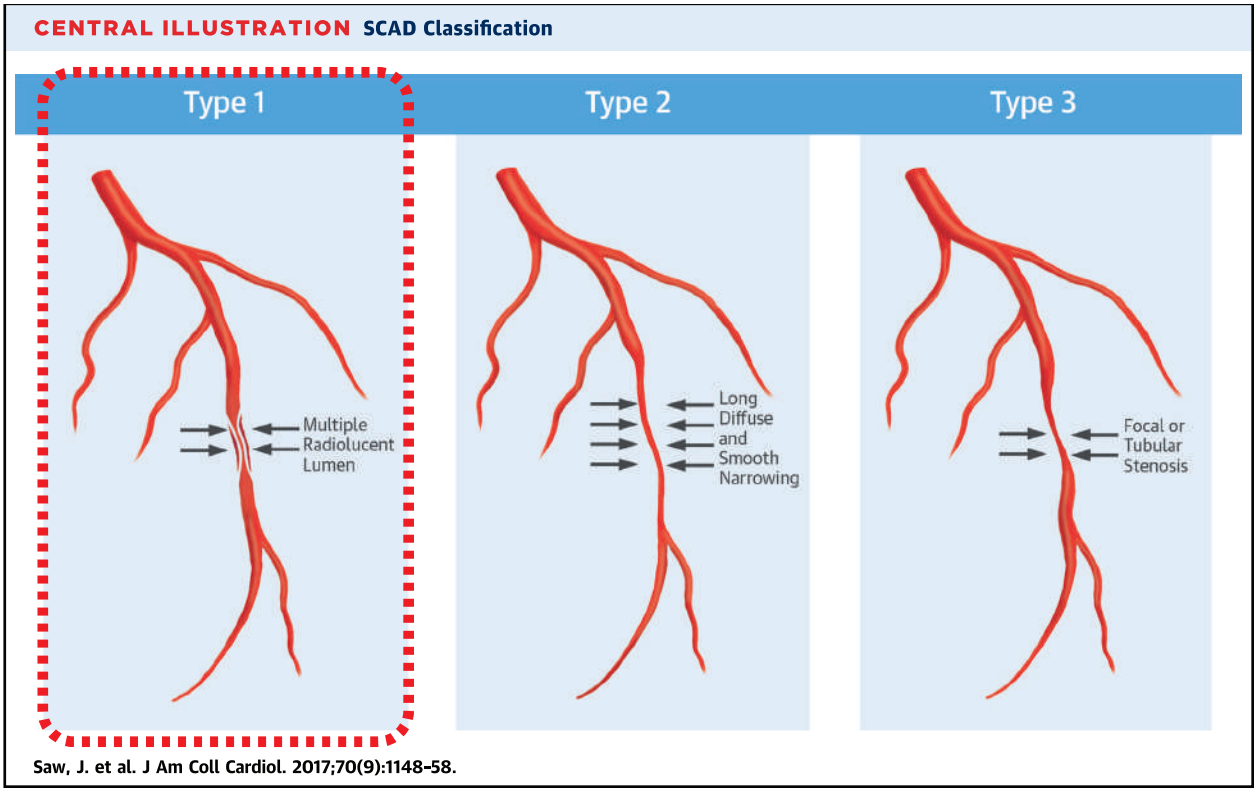
IVUS offers good spatial resolution of ≈150 μm to image the coronary arterial wall. It can detect intimal tear, false lumen, IMH, and intraluminal thrombi, although its resolution may not be adequate to clearly identify all such abnormalities related to SCAD.[18] It has the advantage of deep penetration to assess the depth and extent of IMH. OCT is a more advanced technology with even sharper resolution of 10-20 μm, which has revolutionized the diagnosis of SCAD.[19] Diagnosis of SCAD, requires the presence of IMH or double lumen.

Potential risks of IVUS / OCT in SCAD includes extension of the coronary dissection with wire or imaging catheter, guide-catheter iatrogenic dissection. As because conservative management is treatment of choice in most of the cases, generally imaging can be limited to the proximal segment of dissection.[14]

Coronary CT angiogram (CCTA) is not recommended as the first line investigation for suspected acute SCAD.[20] Moreover, normal results on CCTA do not exclude SCAD. It has lower spatial and temporal resolution than coronary angiography[21] It may be useful for noninvasive follow up of patients with SCAD, particularly in those with dissections in proximal or large-caliber coronary arteries.[22]

Initial Management

Current guidelines for management of ACS advocate an early invasive strategy with revascularization of culprit lesions over conservative therapy alone.[23,24] This stent-based treatment reduces the risk of recurrent occlusion in atherosclerotic MI, but there have been no randomized studies for ACS caused by SCAD. These are critical because the mechanism of vessel obstruction, the acute response to balloon dilation, and the natural history of conservatively managed lesions are significantly different in SCAD compared with atherosclerotic ACS.[14]



SCAD may be a first manifestation of an underlying systemic arteriopathy, and the diagnosis and treatment of extracoronary vascular abnormalities are important components in the comprehensive care of patients with SCAD. [1]

Percutaneous Coronary Intervention

Conservative therapy may not be appropriate in high risk patients with ongoing ischemia, left main artery dissection, or hemodynamic or electrical instability. In such cases, urgent intervention with PCI or coronary artery bypass grafting (CABG) should be considered, considering the coronary anatomy and the expertise of the operators or centers. A proposed algorithm to guide conservative versus invasive management is outlined flowchart.[26]

Hassan S, Saw J. et al. Natural History of Spontaneous Coronary Artery Dissection With Spontaneous Angiographic Healing. JACC Cardiovasc Interv. 2019 Mar 25;12(6):518-527.

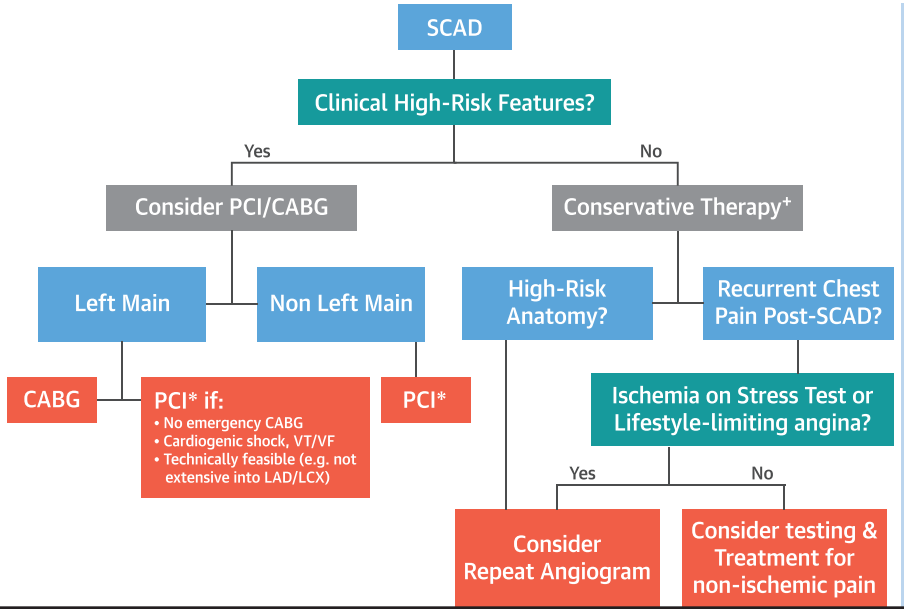
PCI is associated with an increased risk of complications and suboptimal outcomes in treatment of SCAD as seen in different observational studies.[8] This might be due to underlying arteriopa-thies, which make them more vulnerable to iatrogenic dissections, propagation of hematoma and loss of vessel flow with PCI. There are technical difficulties in negotiating the guidewire into the true lumen and increased risk of side branch occlusion. Balloon dilation and stent placement can increase risks of propagating IMH both proximally and distally, causing worsening of vessel obstruction. Dissection lengths are often extensive and hence complete PCI may require long coronary stents, with increased risk of subsequent in-stent restenosis and stent thrombosis. Moreover most IMH naturally resorbs over time, which can result in subacute and late stent strut malapposition, potentially predisposing to future risk of stent thrombosis.[28] These technical complications have been associated with adverse clinical outcomes in several series including technical failure of upto 53% and need of emergency CABG upto 13%.[3]

Strategies during PCI

While performing PCI in SCAD patients, certain things should be avoided as far as possible like deep catheter engagement, noncoaxial positioning of catheter tip, catheter dampening, and strong contrast agent injection. In addition, femoral access is preferable or one should be extra cautious with radial access as there is a 3-fold higher risk for catheter-induced iat-rogenic dissection when radial access.[14]

Multiple strategies have been described for PCI in for SCAD lesions. Examples with successful outcomes include the following: (1) implantation of long DES covering in ex-cess of 5 to 10 mm on both proximal and distal edges of the IMH so as to accommodate propagation of the IMH when compressed by the stent;

CENTRAL ILLUSTRATION **Suggested Algorithm for Management and Repeat Angiography**



(2) direct stenting without balloon predilation to avoid additional risks of extension of the IMH; (3) balloon angioplasty alone to restore coronary flow without stenting; (4) cutting balloon fenestration of the IMH to allow decompression of the false lumen into the true lumen with or without additional stenting (there is theoretical risk of coronary rupture, cutting balloon inflation should be performed cautiously with an under-sized balloon); (5) multistent approach by sealing distal and proximal ends ûrst with stents, before stenting the middle, to minimize IMH propagation; and (6) use of bioresorbable stents to provide a temporary scaffold because complete resorption of struts occurs within 2 years. Finally, after successful PCI, dual-antiplatelet therapy should be administered according to the stents implanted [14]

CABG has been described as a treatment strategy for SCAD in patients with left main stem or proximal dissections, after technical failure of attempted PCI, when there is a complica-tion of attempted PCI, and when ischemia is refractory despite attempted conservative therapy.[17]

Conservative management

Although rates of in-hospital mortality is low, up to 14% of patients require urgent in-hospital revascularization, usually because of extension of dissection. Recognized complications of SCAD depend on the initial treatment strategy.[1] Patients with acute MI should be admitted for a minimum of 48 hours. A prolonged period of in-hospital monitoring (3–5 days) is justified to observe for dissection extension or new recurrent SCAD. If there is clinical worsening despite conservative management (worsening of symptoms together with evidence of ischemia by electrocardiography or significant arrhythmia), repeat angiography should be done and emergency revascularization undertaken to relieve ischemia if feasible. In SCAD patients, Chest pain does not necessarily mean active myocardial ischemia. Pain is also a manifestation of vessel wall dissection itself. Thus, chest pain alone should not be an indication to pursue emergency revascularization, especially if there is no evidence of ischemia or if the vessel flow is normal. [14]

Pharmacotherapy

Thrombolytics should be avoided. Diagnostic angiogram is recommended, but PCI is associated with higher complication rates and suboptimal outcomes, including risk of iatrogenic dissection or propagation of hematoma. If there is minimal ongoing ischemia with distal coronary involvement or preserved coronary flow, instrumentation is avoided because 95% of conservatively treated patients with SCAD will heal within 30 days[14].

Early heparin use may be beneficial in atherothrombotic ACS by reducing thrombus burden. However in SCAD there are theoretical concerns of increasing the risk of bleeding into the IMH or extension of dissection. Therefore, even if systemic anticoagulation is started at presentation; one should consider stopping once SCAD is diagnosed, (if there is no other indications for systemic anticoagulation).[30]

Patients with SCAD who undergo coronary revascularization should receive the standard guideline-based antiplatelet therapy after PCI. There is no clear evidence supporting the use of DAPT in pa-tients with SCAD who do not undergo coronary inter-vention. Theoretically DAPT protects from thrombosis in the intimal dissection site, however many experts avoid it because of increased bleeding risk (menorrhagia in premenopausal women) and no evidence of benefit. Some experts recommend DAPT for at least 1 year, followed by lifelong aspirin.[23] Others for no or limited DAPT use (1–3 months) followed by longterm aspirin therapy. individualised decision for DAPT and aspirin is indicated. [25,31]

β-Blockers should be considered in patients with SCAD who have LV dysfunction or arrhythmias and for man-agement of hypertension. Some advocate for their routine use for SCAD on the basis of its beneûts in atherosclerotic MI or aortic dissection, whereas others recommend that they should be used selectively out of concern for exacerbating vasospasm or symptomatic hypotension.[1]

After SCAD, ACE or ARBs should be used when MI is complicated by LV systolic dysfunction, according to guidelines for the management of patients after MI. Hypertension should be rigorously controlled. Female of reproductive age must be warned of the teratogenicity. [16]

Statin is not recommended routinely after SCAD and is reserved for patients meeting other guideline-based indications [17]

The antianginal therapy is mostly needed managing post-SCAD chest pain syndromes. chest pain during hospitalisation may also warrant antianginal, but

currently, there is no role for routine use. Nitrates, calcium channel blockers, or ranolazine can be used for reliving of isch-emia and symptoms.[14]

Coronary Artery Assessment after SCAD

After initial management of SCAD, repeat imaging may be needed in certain situations like in patients with recurrent symptoms, abnormal functional tests, high-risk anatomy (eg, left main, proximal, or multivessel dissection, in which residual dissections may beneût from revascularization for high ischemic burden) or unclear cause of coronary stenosis (to assess for angiographic healing to help differentiate SCAD from other coronary disease).[14]

Scad Prognosis and Recurrence Risk

Besides major adverse cardiac events such as recurrent MI, recurrent SCAD, unanticipated revascularization, congestive heart failure, stroke, and death; SCAD patients can develop chest pain, anxiety and depression after hospital discharge. At follow up of 2 to 3 years, major adverse cardiac events were reported in 10% to 30% of cases.[32]

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REVIEW ARTICLE

Post COVID Cardiovascular Manifestation: An Emerging Issue to Consider

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J Inv Clin Cardiol 2021; 3(2): 80-83

Introduction:

The cardiovascular consequences of COVID-19, the disease stemming from infection with the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is evolving rapidly. Cardiologists are taking a closer look at the possible long-term cardiovascular effects on COVID long-hauler patients who still show symptoms long after they should be recovered from the virus. With nearly two years of experience with the COVID-19 (SARS-CoV-2) virus, it has been found that some post-COVID patients experience long-term lingering coughs, cognitive issues and other complains months after they recover from the virus. These long-haulers are also appearing to be having the heart issues as well. Determining the long-term impact of coronavirus will certainly be one of the top cardiology issues discussed in 2021. Here, we review our current understanding of the implications of COVID-19 on the cardiovascular system in resolved infection (considerations those with "long COVID").

Why there is long term Cardiovascular impact of COVID

COVID patient cadaver studies also raised questions on the long-term damage to the heart after it was found COVID kills cardio myocytes.[1,2] But the question has been what is the long-term impact of this cardiac injury. Early on in the COVID pandemic, it became quite clear that hospitalized patients, particularly those that were sick enough to make it to the ICU, a significant minority of them, anywhere from 20 to 30%, had evidence of heart injury. And that heart injury was really related to two different types of pathophysiology. The first was a response to the high levels of circulating stress hormones. And the second was direct viral invasion myocarditis. And that became an important therapeutic target for us early on in the course of this disease. But the question asking now is what would the long-term implications of this be, and perhaps more importantly.

Qualifying COVID Cardiovascular Damage and the Role of Comorbidities in Mortality

One study during the first wave of COVID found cardiac injury occurred in 19.7% of hospitalized COVID patients, and it was an independent risk factor for increased in-hospital mortality.[3] A more recent late-breaking study at the American Heart Association (AHA) meeting in November reported data from 14,000 U.S. COVID patients. It showed in-hospital mortality due to cardiovascular comorbidities ranged from 28.5 percent for patients with hypertension to 28.6 percent for those with diabetes, 25.5 percent for those with coronary artery disease, and 38.4 percent for those with heart failure.[4] But for the patients who do recover, it is not yet known if there is a lasting impact.

A German MRI study of 100 post-COVID patients revealed cardiac involvement in 78 patients (78%) and ongoing myocardial inflammation in 60 patients (60%).[5] This was independent of pre-existing conditions, severity and overall course of the acute illness, and time from the original diagnosis. These findings indicate the need for ongoing investigation of the long-term cardiovascular consequences of COVID-19.

With the myocarditis, this is something that has been seeing in some of the sick COVID patients, but it's sounding like there might be signs of myocarditis even

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less sick patients, About 25% to a third of patients who are severely ill with COVID have evidence of troponin being elevated, indicating heart damage, but lots of different things can cause this. A more sensitive test would be an MRI of the heart, and there have been research reports of MRI abnormalities even in young athletes who had COVID.

While the acute inflammation and injury to the heart are seen in some COVID patients, the long-term effects of healed myocarditis is not completely unknown. Most infected patients experience mild, self-limiting symptoms, but are not undergoing regular ECG or cardiac imaging to monitor them.[6] Some of these patients may be at risk for subsequent arrhythmias.

Evidence for pericarditis and myocarditis was also found in a Spanish study of 139 healthcare workers who had COVID.[7] About 10 weeks after infection, the patients underwent ECG, lab tests and cardiac magnetic resonance (CMR) imaging. The prevalence of pericarditis or myocarditis was found in up to 40% of cases. Pericarditis coexisted with some degree of concurrent myocardial inflammation in 11% of cases.

Chest pain, dyspnoea or palpitations were observed in 58 (42%) participants. ECG abnormalities were found in 69 (50%) of patients. CMR abnormalities were found in 104 (75%). Isolated pericarditis was diagnosed in 4 (3%) participants, myopericarditis in 15 (11%) and isolated myocarditis in 36 (26%). NT-pro-BNP, a non-active prohormone that is released in response to changes in pressure inside the heart usually related to heart failure or other cardiac issues, was elevated in 11 (8%) of patients. Troponin was elevated in 1 patient.[7]

CDC Recognizes COVID Long-Haulers

Thousands of patients have reported symptoms ranging from shortness of breath, chronic fatigue, brain fog that causes difficulty concentrating, anxiety and stress. The Centers for Disease Control and Prevention (CDC) started reporting these issues last July.[8] In the study the CDC posted, more than a third of people had not returned to their usual state of health two to three weeks after testing positive.

Physicians began suspect there may be long-term damage not just in lungs, but in the heart, immune system, brain and other organs. Evidence from previous coronavirus outbreaks, especially the 2002 severe acute respiratory syndrome (SARS) epidemic, suggested these effects may last years. [9]

COVID Clinics to Monitor Long Term Effects

To get a better handle on long-hauler patients, it is now mandatory in creating Post-COVID monitoring clinics, even dedicated cardiac post-COVID clinic for long-haulers.

After initial resolution of COVID, infection, either asymptomatically or with persistent symptoms, particularly among those that have disabling persistent symptoms, the basic assessment would be an echocardiogram, a 12, lead ECG, blood testing for cardiac troponin to rule out active myocarditis and then some form of ambulatory monitoring to screen for the heart rhythm disturbances that we know would increase risk of something bad happening like sudden death."

Athlete COVID Long-hauler Cardiac Research

In January 2021, the AHA and the American Medical Society for Sports Medicine (AMSSM) joining forces to accelerate a critical new research initiative studying cardiac conditions in athletes who had COVID-19. Both organizations hope to speed up research to find new insights on the impact to the cardiovascular system of college athletes and to understand when it is safety for them to return to play.

The Outcomes Registry for Cardiac Conditions in Athletes (ORCCA) has already collected data from more than 3,000 athletes. The AHA will use its Precision Medicine Platform (PMP), a secure cloud-computing platform hosted by the Association's Institute for Precision Cardiovascular Medicine to facilitate the research.

Long COVID and Cardiovascular manifestations

The cardiovascular consequences of COVID-19 extend beyond acute infection. Many continue to report lingering cardiopulmonary and neurologic symptoms, namely chronic fatigue, dyspnea, chest pain, and dysautonomia – colloquially known as "long COVID". Of discharged patients, 60-80% reported at least one residual symptom 50 days after initial COVID-19 diagnosis. [10,11] Even in non-hospitalized individuals, 35% of those surveyed by the Centers for Disease Control (CDC) reported symptoms 14-21 days after initial diagnosis, even young patients without comorbidities.[12]

Of particular relevance is a Postural Orthostatic Tachycardia Syndrome (POTS)-like syndrome emerging among COVID-19 survivors. The association between preceding viral illness and POTS has been

previously established.[13] With ACE2 found on neurons, alteration to the autonomic nervous system has been implicated in COVID-19.[14] An increased incidence in POTS will require clinicians to be familiar with current principles in management, which largely center on education, volume and salt optimization, and agents to maintain vascular tone (e.g., Midodrine) and manage palpitations (e.g., Beta blockers, Ivabradine).[15,16]

Other COVID Surveillance Registries

There were also several COVID registries and studies created to track the cardiac impacts of COVID. Here are a few they will likely yield new information later in 2021.

The AHA created its COVID-19 Cardiovascular Disease (CVD) Registry in April 2020 to capture data on patient clinical characteristics, medications, treatments, labs, vitals, biomarkers and outcomes for adult patients hospitalized with COVID-19. This includes those without a history of cardiovascular disease or stroke. To date, more than 22,000 de-identified patients' health measures and data points are available in the registry. The Society of Cardiovascular Angiography and Interventions (SCAI) also launched the North American COVID-19 Myocardial Infarction (NACMI) Registry enrolls any COVID-19 positive patients with ST-segment elevation MI (STEMI) or new-onset left bundle branch block with a clinical correlate of myocardial ischemia. The data will be compared to an age and gender-matched control population from the existing Midwest STEMI Consortium.

The American College of Cardiology collects COVID-19 data through the NCDR Chest Pain – MI Registry and the Cath PCI Registry. The goal is to better understand the relationship between COVID-19 and heart disease and maximize data on the cardiac impact of the virus.

In the U.K., the British Heart Foundation has started seven COVID studies to better understand the effect of the virus on the heart and what it means for long-term impacts. Cardiac MRI and computed tomography (CT) will be used in two of the studies.

Doctors Need to Avoid Ignoring COVID Long-Haulers

There is a tendency by some physicians to write off many patient complains as being in their head, rather than being caused traceable medical reasons. This has occurred in patients who take statins, had stents impacted and continue to have chest pain, and in some medical conditions such as chronic fatigue and Lyme disease. Since this is a new disease, many experts are warning that physicians need to pay attention and data needs to be collected to determine if there is an explanation for complains by COVID long-haulers.

Conclusion:

COVID-19 impacts the cardiovascular system in both the short and long term. We reviewed here our understanding to date of chronic cardiovascular considerations of infection. The wealth of basic and clinical research spurred by the global pandemic will continue to refine our understanding of the interplay between COVID-19 and the heart in the years to come.

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Two Stent Strategy in Primary Angioplasty

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Seema Nepal[4], Pravin Kumar Yadar[4]

Abstract:

A bifurcation coronary lesion is a lesion occurring at or adjacent to , a significant division of major epicardial coronary artery .[1] The prevalence of bifurcation in acute ST elevation Myocardial Infarction (STEMI) is 10 to 23 %. Although One-stent Provisional stenting technique is preferred approach, sometimes it is impossible or dangerous to stent only the main vessel branch.[2] Here we present a case of 72 year old male with acute extensive anterior wall STEMI who have undergone successful primary percutaneous coronary intervention (PPCI) for Left anterior descending (LAD) and diagonal bifurcation stenting with Inverted T- stenting and small protrusion (TAP) Technique showing that two stent strategy in PPCI is safe and feasible whenever indicated.

Key Word: STEMI, PPCI, Bifurcation Stenting.

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Introduction:

Bifurcation lesions occurs in 1 in 10 STEMI and one-stent provisional technique is preferred approach.[2] However, sometimes it is necessary for bifurcation stenting with two stent strategy as recrossing to the critical side branch may be difficult which usually occurs in left main bifurcation lesions and for large LAD diagonal bifurcations during primary angioplasty.

Case Summary:

A 73 year old male presented to emergency department of Sahid Gangalal National Heart Center, Kathmandu, Nepal with complain of acute onset central chest pain for 4hrs duration. He was an active smoker but without any other cardiovascular risk factors. He was haemodynamicaly stable and was in Killip class II at presentation and his door to balloon time was 40 minutes. His ECG showed diffuse ST Elevation from Lead V1 to V6 and echocardiography revealed Hypokinetic LAD artery territories. He was diagnosed as acute extensive anterior wall STEMI and was taken up for PPCI.

On coronary angiography showed a normal right coronary artery (RCA) and left circumflex artery (LCX). The left injection showed that the left main coronary artery was normal and bifurcated into LAD and LCX. The LAD showed bifurcation lesion in main branch proximal LAD and side branch diagonal 1 (D1) (Fig 1). The proximal LAD showed 100% thrombotic occlusion and D1 was 80 % occluded .The TIMI flow In proximal LAD was 0 and TIMI flow in D1 was 2 before predilation. We planned for provisional stent strategy for the bifurcation lesion.

The LAD occlusion was crossed with a balance middle weight (BMW) wire and predilated with 2x15 mm splinter balloon at 14 atmosphere . The D1 occlusion was crossed with BMW wire. The LAD was stented with 3x28 mm drug eluting stent (DES) (Xience Expedition) with jailed guide wire in D1(Fig 2.) After stenting the LAD, the D1 had only TIMI 1 flow. As the flow in D1 was compromised, the plan was changed for two stent strategy. Kissing ballon dilatation was done with 2X15 mm splinter balloon at D1, and with 3X8 mm NC balloon at LAD. The D1 was stented with 2.5x 18 DES (Xience Expedition) at 12 atmosphere stent positioning was adjusted to ensure full coverage of the ostium by placing the proximal stent at the level of proximal ostium border and uninflated balloon left in LAD (Fig 3). The D1 stent balloon was slightly pulled upward with the LAD to perform kissing balloon inflation and final kissing balloon inflation of the bifurcation with the D1 stent ballon and LAD ballon was performed at 12 atmosphere (Fig. 4). Proximal Optimisation technique (POT) was done in LAD using 3.5 X 8 mm balloon at 14 atmosphere (Fig. 5) with the final result of TIMI III flow in both LAD and D1 (Fig.6) . Intravascular ultrasound could not be done due to unavailability at our institution during that time.

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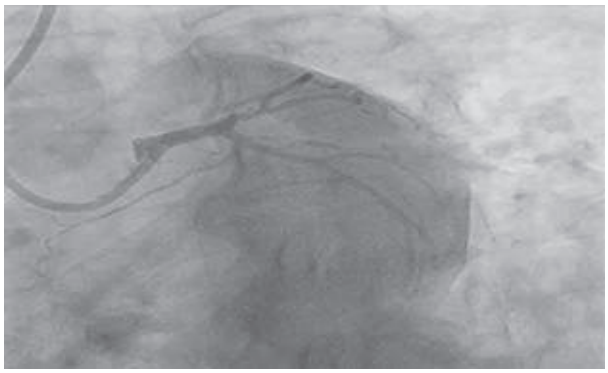


Fig.-1: *Bifurcation lesion in proximal LAD and D1*

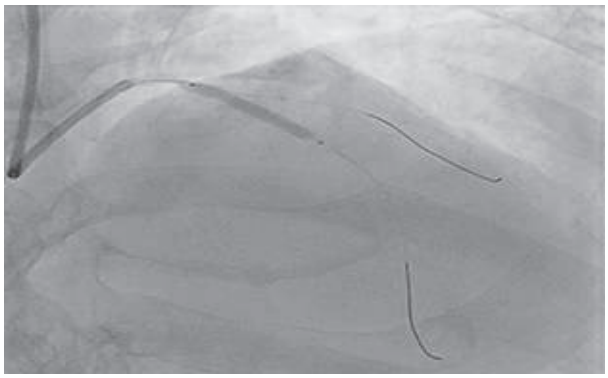


Fig.-2: *LAD was stented with 3x28 mm drug eluting stent (DES) (Xience Expedition) with jailed guide wire in D1.*

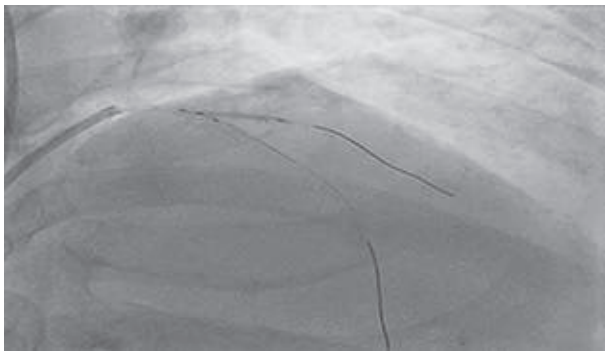


Fig.-3: *The D1 was stent with 2.5x 18 mm DES (Xience Expedition) and uninflated balloon in LAD.*

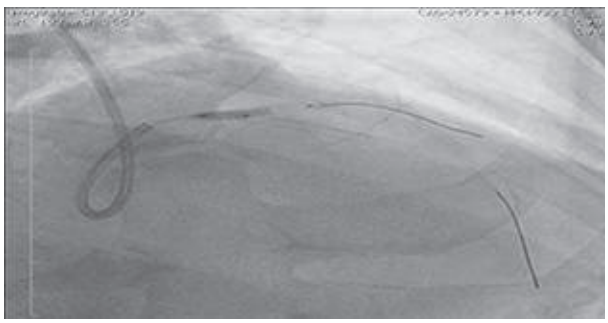


Fig.-4: *Kissing balloon inflation of the bifurcation with the D1 stent balloon and LAD balloon*

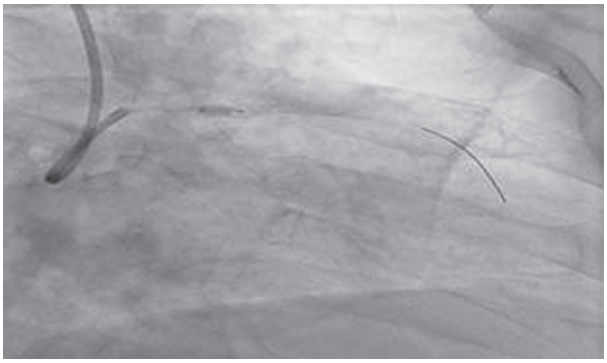


Fig.-5: *Proximal Optimisation technique using 3.5 x 8 mm Balloon in LAD*

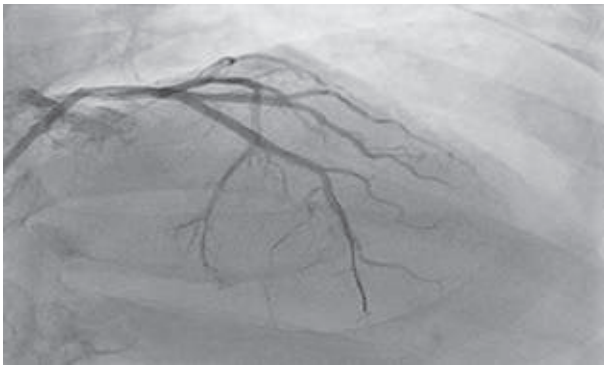


Fig.-6: *Final result TIMI III flow in both LAD and D1*

Discussion:

There are very few studies focus bifurcation stenting in STEMI with various issue. Despite being challenging lesions, bifurcation lesions in STEMI are associated with similar time to reperfusion and procedural success but led to significantly greater contrast use and prolonged procedural time compared with non-bifurcation lesions .[3] A retrospective case control study showed during acute STEMI, bifurcation intervention has excellent acute procedural outcomes, but significantly increased incidence of target lesion revascularization , stent thrombosis and mortality at 12 months.[4] There may criticism for a strategy of direct two stent technique in spite of the current policy of provisional stenting to the side branch . Kwan TW et. al in a randomized control trial in provisional stenting versus the DK crush stenting techniques demonstrated the bifurcation stenting in patients with STEMI is safe and feasible with comparable immediate and midterm clinical outcomes.[5] Herrera-nogueira R et al ,reported the predictors of angiographic success were: bifurcation anatomy non-Medina (1,1,1), TIMI flow 2-3 after wire insertion in the MB and SB, SB predilatation and stent implantation in STEMI patients

undergoing primary or rescue PCI of a bifurcation culprit lesion.[6] In the HORIZONS-AMI trial showed although the PCI procedure involving bifurcation lesions was more complex, the acute results and late outcomes in patients with BTLs were comparable to those in patients without BTLs.[7] Recently Harbalioglu H et al reported , a complex stenting strategy provides better side branch protection than a simple stenting strategy for the treatment of culprit true bifurcation lesions with major side branch involvement in patients with acute myocardial infarction.[8] The proximal optimisation technique (POT) is a key part of treating large bifurcation lesions and will optimise results of both single and two stent strategies with an appropriately sized balloon should be positioned and inflated just up to the carina. When performed well, the enhanced lesion scaffolding, reduced strut malapposition and improved flow dynamics are likely to translate into improved clinical results as shown in our case also.[9]

No- reflow is encountered most frequently among patients undergoing PPCI and angiographic factors independently associated with no-reflow included longer lesion length, higher risk class C lesions, bifurcation lesions, and impaired pre procedure TIMI flow.[10] However, angiographic and procedural success are similar in bifurcation vs nonbifurcation lesions if performed with correct technique. [3, 11] Here we have described a patient presented with acute myocardial infarction who needed bifurcation stenting with successful two stent strategy during primary angioplasty

Conclusion:

Two stent strategy for bifurcation stenting is safe and feasible and should be performed when indicated in bifurcation stenting.

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Cracking Recalcitrant Calcium in a Tortuous RCA: A Case Report

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Abstract:

Increasingly more patients present with lesions with high coronary calcium (LHCC) in contemporary practice. Tackling calcified coronary arteries present challenges to the interventionalist, and require the deployment of multiple debulking strategies, in addition to ancillary technologies such as guide extensions coupled with intravascular imaging guidance. This case report highlights the challenges faced in the percutaneous revascularisation of a severely calcified tortuous right coronary artery in a 74-year-old male with multiple co-morbidities, the lesion of which was initially balloon-uncrossable. Multiple techniques including grenadoplasty, upscaling of non-compliant balloons, rotational atherectomy and the use of guide extension, temporary pacing wire and intravascular ultrasound (IVUS) were necessary for successful stent deployment.

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Introduction:

Lesions with high coronary calcium (LHCC) are encountered with increasing frequency in current practice. They are associated with large plaque burden and greater lesion complexity[1]. Percutaneous coronary intervention (PCI) of severely calcified lesions is associated with procedural complexity and consequently, higher complication rates and adverse clinical outcomes[2,3]. Stent underexpansion is not uncommon with calcified lesions, and as such, adequate lesion preparation is crucial[3, 4]. Despite a growing armamentarium of debulking technologies which include balloon-based strategies and ablative strategies, calcified lesions remain a challenge to the interventionalist[1,3,4]. The role of intravascular imaging is key to demonstrating the nature and anatomic distribution of calcium, and thereby deciding on choice of appropriate lesion-modification strategy [1,3,4]. We report the case of an elderly male presenting with intractable angina, in whom imaging-guided PCI to a severely calcified and tortuous right coronary artery (RCA) lesion was done. We outline the challenges of stenting in this initially balloon-uncrossable lesion and demonstrate the use of calcium debulking strategies as well as ancillary tools.

Case Report:

A 74-year-old male presented with Canadian Cardiovascular Society (CCS) class IV angina for 6 weeks while on optimum anti-anginal medications and guideline directed medical therapy. He had multiple co-morbidities was hypertensive, diabetic and dyslipidaemic. Electrocardiogram (ECG) showed ST-T changes in inferior leads. Echocardiogram showed a preserved left ventricular ejection fraction of 65%. Coronary angiography was done transradially via a 5 French Tiger catheter. Left system angiography showed a significantly diseased first diagonal and distal obtuse marginal (OM) branches. RCA showed severe proximal tortuosity with severe diffuse calcified lesion with 95-99% stenosis in its midpart (Figure 1).

As both the diagonal and OM branches were of smaller calibre, percutaneous coronary intervention (PCI) to the RCA was planned as an elective procedure in this case of complex high risk indicated PCI (CHIP). Given the severe diffuse calcification, intravascular imaging was considered to decide on further strategies for adequate lesion preparation after wiring. Adequately sized balloons and rotational atherectomy was considered.

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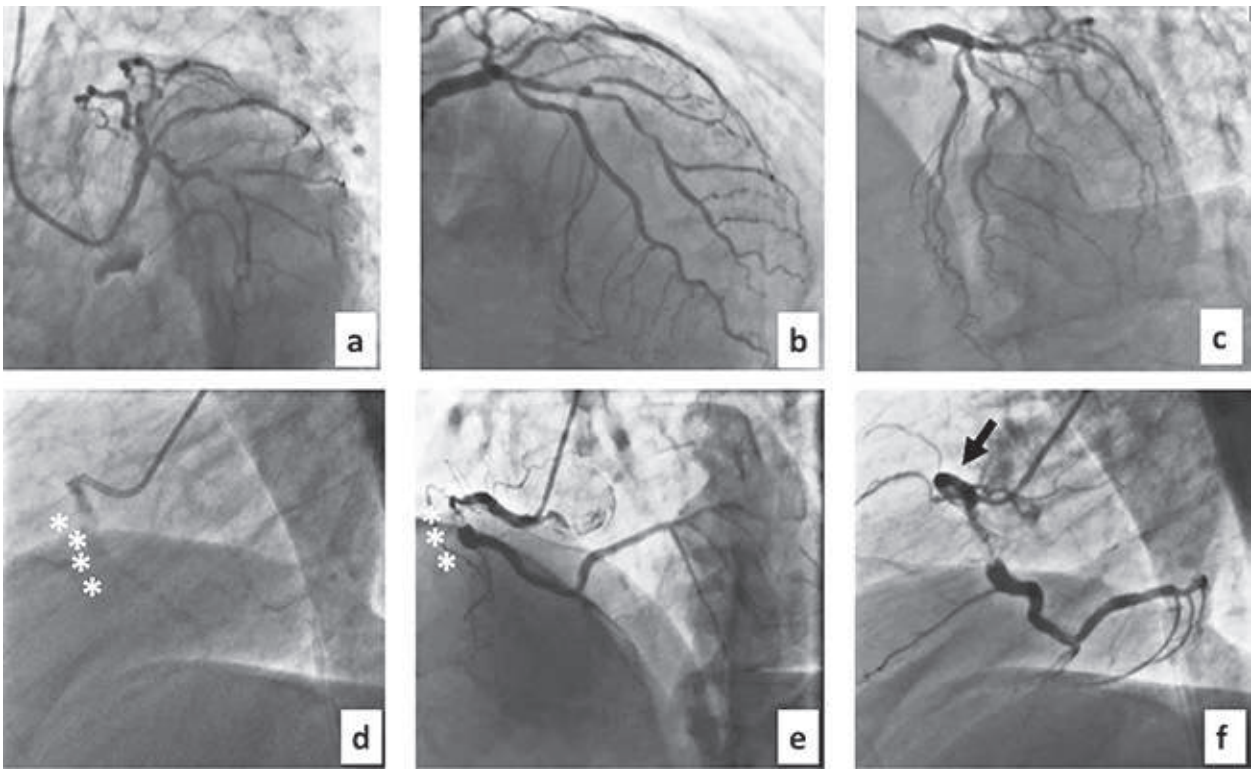


Fig.-1: *Coronary angiography. (a-c) Left system showing significantly diseased first diagonal and distal obtuse marginal (OM) branches. (d-f) Right system showing severe proximal tortuosity with severe diffuse calcified lesion with 95-99% stenosis in its mid part*

The RCA was engaged with a 6 French Judkin's Right (JR) 3.5 guide catheter. In anticipation of rotational atherectomy, a temporary pacing wire (TPW) was placed in advance, and wiring was attempted with *PT2* moderate support polymer tip hydrophilic wire (*Boston Scientific Corp, Natick, Massachusetts, USA*) with 1.25x5 m balloon support. The severe tortuosity and calcification presented difficulties in wire crossing, which ultimately crossed with the support of *Finecross* MG Coronary Micro-Guide Cathether (Terumo Interventional Systems) (Figure 2: a-e). However, although the wire crossed, the microcatheter was not crossed. Thus, the next challenge presented as a balloon uncrossable lesion at the mid part of the severely calcified RCA. The smallest profile 1.25x5 mm balloon partially crossed the lesion and grenadoplasty was done. The mid RCA was eventually traversed with the support of 6 French *Guidezilla* guide extension catheter wire (*Boston Scientific Corp, Natick, Massachusetts, USA*), with another 1.5x10 mm by which grenadoplasty was done. The balloons were subsequently upscaled for further lesion preparation and following multiple balloon inflations with 2.0x15 mm and 2.5x8 mm NC balloons, they gradually advanced across the calcified segment (Figure 2: f-j).

A 5 French *OptiCross* Intravascular ultrasound (IVUS) coronary imaging catheter (*Boston Scientific Corp, Natick, Massachusetts, USA*) could now be passed across the lesion, which showed severe concentric 360 degree "napkin ring" calcification (Figure 3). 4 runs of rotational atherectomy were performed with 1.5 mm burr at 160,000 rpm. The patient became entirely dependent on temporary pacing during the course of rotational atherectomy (Figure 4: a-b).

Distal embolization of debris immediately following rotablation led to no reflow as evident on the next

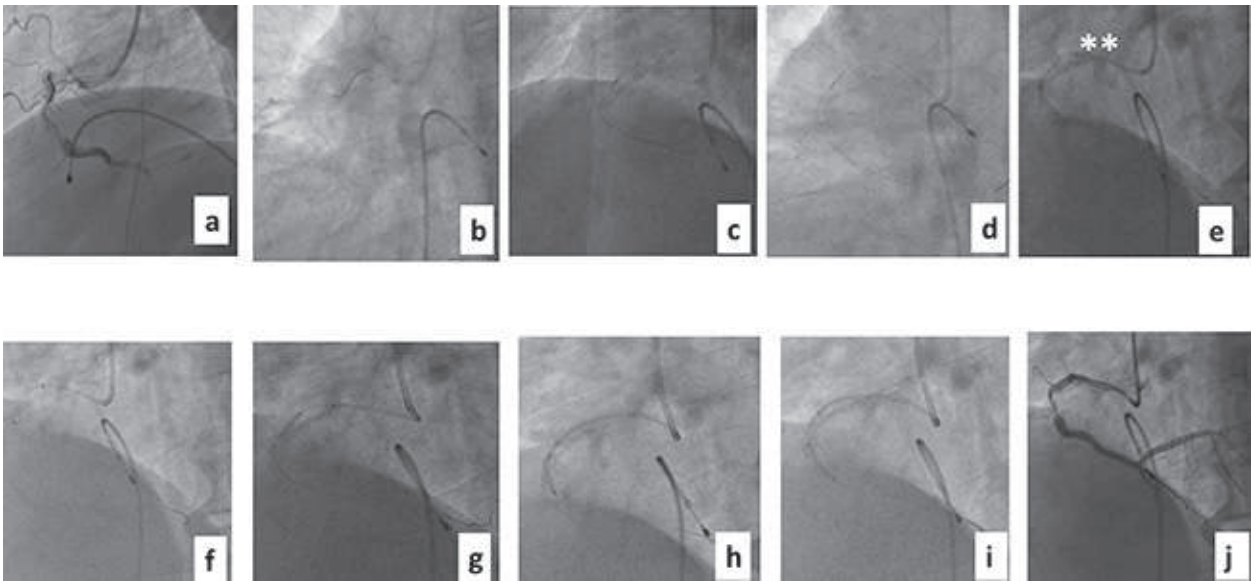


Fig.-2: *Navigating the balloon uncrossable lesion in calcified mid RCA. (a) Temporary pacing wire placed. (b) Began wiring with moderate support hydrophilic wire with small profile balloon which did not cross. (c) Wire crossed with microcatheter support, however microcatheter did not cross (d) Grenadoplasty done with 1.25x5 mm balloon (e) 1.5x10 m balloon navigated with guide extension catheter support (*). (f) Grenadoplasty done with 1.5x10 mm balloon (g-i) Multiple balloon inflations and upsizing with gradual advancement across the calcified segment. (j) Contrast injection after balloon crossed distally*

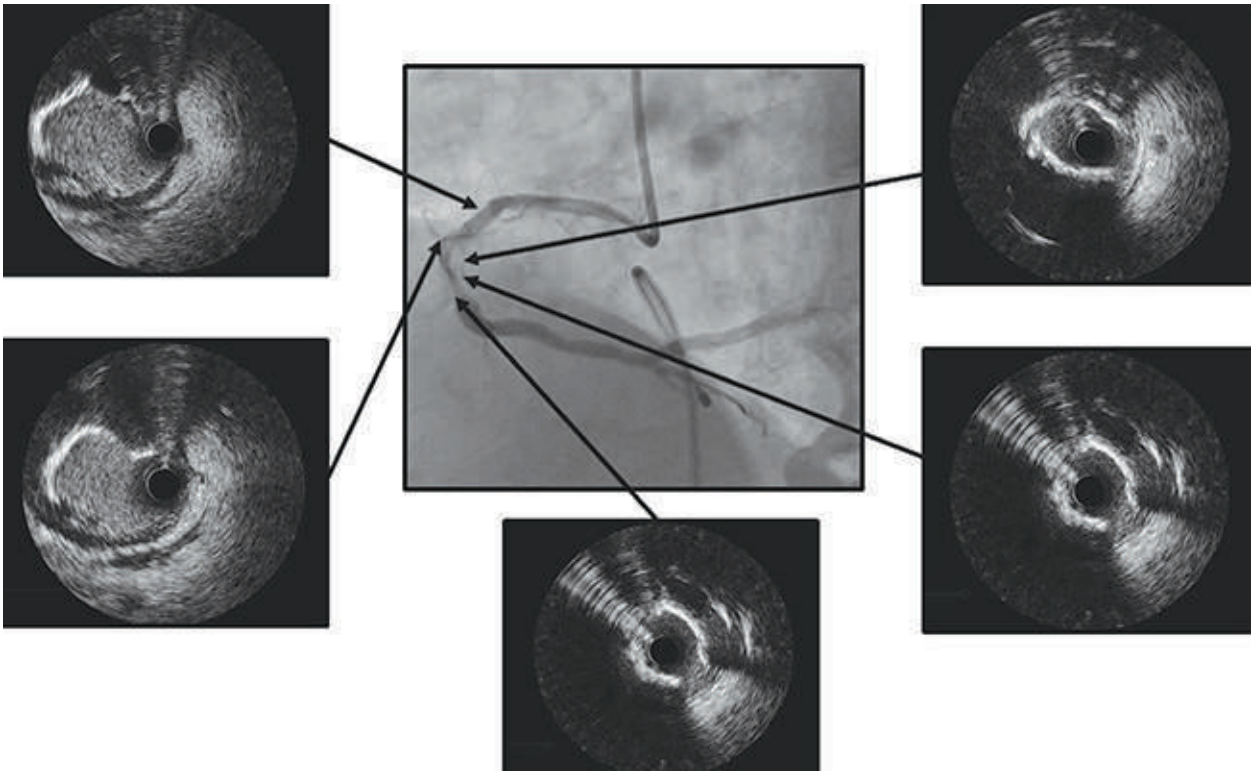


Fig.-3: *Pre-stenting IVUS. Images showing concentric 360 degree napkin ring calcium in the mid RCA*

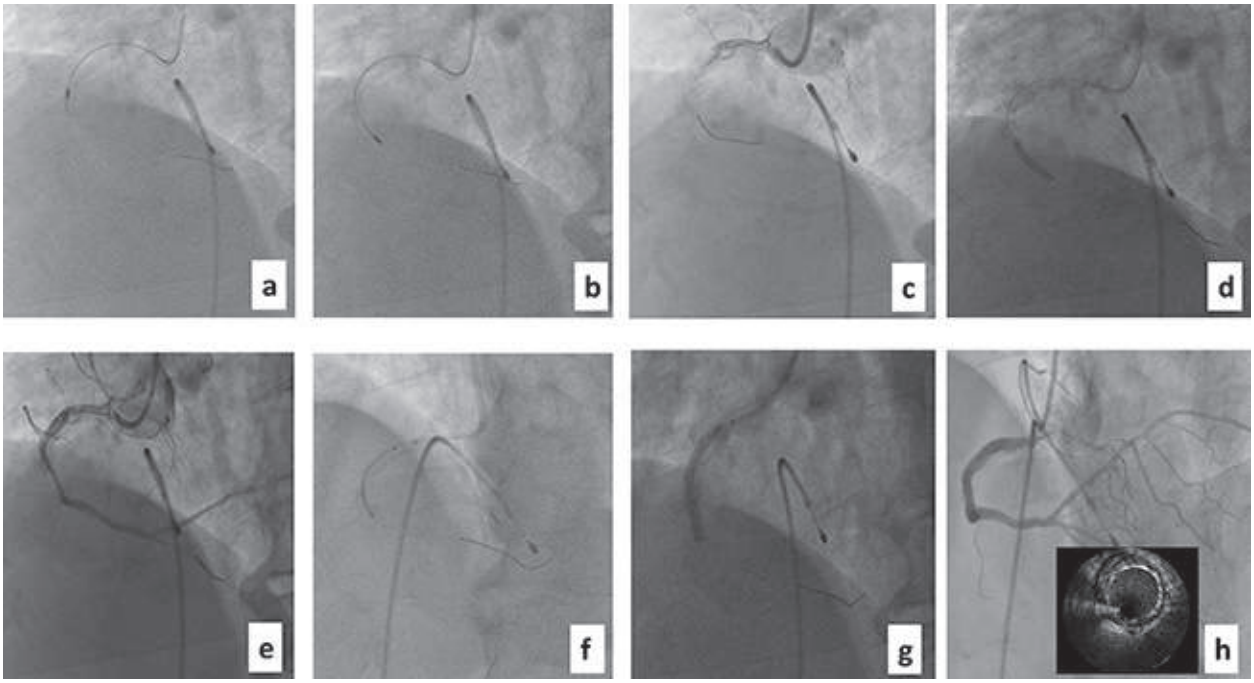


Fig.-4: *(a, b) Rotational atherectomy performed with 1. mm burr at 150,000 rpm. (c) Distal embolization of debris immediately following rotablation leading to no reflow. (d) Further lesion preparation by 2.5x10 mm NC balloon at 16 ATM. (e) Contrast injection after lesion preparation. (f-g) Mid RCA lesion stented by 3.5x38 mm Everolimus eluting stent (EES). (h) Final angiographic and IVUS (inset) result.*

contrast injection after rotational atherectomy (Figure 4: c). Further lesion preparation was done by 2.5x10 mm NC balloon at 16 ATM (Figure 4: d). The mid RCA lesion was stented by 3.5x38 mm Everolimus eluting stent (EES) at 16 atmospheres (Figure 4: f-g). Post-stenting angiography showed well-expanded and well-opposed stents and a satisfactory angiographic and imaging result (Figure 4: h). The patient was maintained on dual antiplatelet therapy comprising of aspirin 75 mg daily and ticagrelor 90mg BID for the initial month, with ticagrelor de-escalated to clopidogrel once daily thereafter.

Discussion:

There are multiple challenges posed by this case, each of which were successfully navigated with available resources. Firstly, the lesion was balloon-uncrossable, thus posing difficulties in navigating an IVUS catheter across the lesion. As the microcatheter also was not crossed, upfront rotational atherectomy was not possible. Uncrossable lesions are those that cannot be crossed with a balloon after successful guidewire crossing, and commonly encountered in tortuous and calcified arteries (5). First-line strategies to traverse balloon-uncrossable lesions include use of a small low-profile balloon <1.5mm in diameter, microcatheter, increased guide catheter support (large calibre), the use of guide extension, a combination of which resulted in successful crossing of the smallest profile balloon in our case (5). Guide catheter extensions allow for deep intubation and facilitate a smooth pathway to the lesion, traversing calcification and tortuosity in the proximal artery (5). Subsequently second line strategies of balloon-assisted microdissection (BAM) or the intentional rupture of balloon, also known as “grenadoplasty” was deployed for further crossing and dilatation of the lesion (5,6).

Adequate lesion preparation through calcium modification prior to stenting is crucial (3). Multiple algorithms for optimal lesion preparation and treatment of calcified lesions have been described in the literature (1,3). Central to these algorithms is the accurate detection and characterization of coronary artery calcium by intravascular imaging and optimal lesion preparation prior to stenting (1,3, 4). Contemporary technologies for the treatment and preparation of LHCC can be broadly categorized into 2 groups: balloon-based techniques and ablation techniques (1, 3). Balloon-based techniques (1,3) include non-compliant

balloons, cutting balloons, scoring balloons, chocolate balloon, super high pressure (OPN) balloons and lithoplasty balloons for shock wave intravascular lithotripsy (IVL), the latter two of which are not available in Bangladesh. In our case, the severely calcific lesion was not fully dilatable despite multiple non-compliant balloons. As such, further lesion preparation was done by rotational atheterectomy, which is the only available ablation technology in the country.

Rotational atherectomy (RA, *Rotablator [Boston Scientific*]), initially introduced the late 1980s, is a debulking device that facilitates the removal of atherosclerotic plaque and modification of superficial calcium by employing a mechanism of differential cutting (1,3,7). The use of RA is associated with a learning curve and technical expertise. Although use of RA has demonstrated significantly improved success rates in tackling LHCC, this procedure can be associated with potentially serious complications including slow flow, no-reflow, vessel perforation, dissection and burr entrapment. Embolisation of distal debris resulted in slow flow in our case. Furthermore, RA can also cause transient bradyarrhythmias due to vagal responses or localized adenosine release, necessitating a TPW (3). Anticipating this, we prophylactically placed a TPW in this patient, which proved extremely useful as the patent became entirely dependent on TPW during the entire course of RA.

In daily practice, most operators use a combination of devices and complementary strategies to achieve the desired outcomes in LHCC. The use of intravascular imaging is of paramount importance. Our case highlights the procedural complexities and challenges associated with treating balloon uncrossable LHCC in tortuous arteries, and the steps taken to overcome them as recommended in the calcium algorithms as a resource-tailoring approach.

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