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SEVERITY IN STENOSIS OF CULPRIT CORONARY ARTERY IN PATIENT WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT

Background: ST elevation myocardial infarction (STEMI) is a life-threatening condition that is often due to a thrombotic occlusion of an offending coronary artery. Measuring the degree of stenosis is important for defining risk and choosing a revascularization strategy.
Objective: To establish the prevalence and extent of stenosis of culprit coronary arteries in patients with acute STEMI who are treated with primary PCI.
Methods: The current study is a cross-sectional descriptive study carried out at the Department of Inpatients of Cardiology, NICVD Karachi, from October 2019 to April 2020. A total of 181 patients with STEMI undergoing primary PCI were included. Quantitative coronary analysis parameters of vessel diameter, lesion length, area stenosis, and diameter stenosis were measured through angiography.
Results: Severe Stenosis (>70%) was noted among 60.2% of the patients, moderate stenosis among 26.5% of patients, and minimal stenosis among 13.3%. Severe stenosis was significantly correlated with lower TIMI flow grades and higher thrombus burden.
Conclusion: Culprit arteries of STEMI have a predominant angiographic finding of severe stenosis requiring early recognition and intervention.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is one of the leading causes of morbidity and mortality in the world. It happens when a coronary artery becomes blocked by a blood clot on a weak spot (plaque) of cholesterol and fat. New technology has brought new tools with artificial intelligence and deep learning to determine the source of the blockage. These tools can help doctors to give more resources in emergency situations (1). However, the specific lesion is often difficult to identify, particularly in patients with non-ST elevation syndromes or more than one blocked vessel. The combination of the clinical, angiographic, and functional information makes the decision-making difficult (2). The distribution of coronary atherosclerosis is very different between STEMI and non-STEMI patients. Recent observational studies have shown that patients with STEMI have a more severe disease as reflected in higher scores in nonculprit lesions (3). The plaque is also distinct in both cohorts. Optical coherence tomography (OCT) revealed an excess of thin-cap fibroatheroma, plaque ruptures, and red thrombosis in STEMI. This comprised an improved selection of extraordinary load lesions and a greater prominence of the culprit lesion (4).

Secondly, the underlying vessel implications arise in broad direct association with risks of reperfusion injury. An atheromatous lesion with short segment length conveys a relatively worse chance of recoverability (5). The treatment component of STEMI is to treat the culprit lesion or refrain from treating the non-culprit lesion. Background clinical guidelines had been controversial between culprit-only treatment versus complete revascularization, emphasizing the importance of careful evaluation of lesion severity to determine management (6). Advanced imaging, including strain echocardiography, has been used to better characterize the culprit vessel in the non-STEMI patient as an exploratory tool to specify culprit vessel identification, and that may feature flow (7). Occasionally, coronary arteries have birth defects and are known to cause blockage (8). Non-occlusion myocardial infarction (STEMI patients) becomes more complicated due to the non-infarct cause. Comparing invasive studies with cardiac imaging during electrophysiological coronary priming prior to interventional testing, it was demonstrated that many of the angiographic side branches with small perfusion defects represent hemodynamically significant regions documented by a technique sensitive to obstruction (9).

This new discovery affects treatment. For instance, the complete trial showed that complete revascularization has a much greater benefit on angina-related quality of life versus culprit lesion revascularization alone. This shows the need for lesion severity assessment for long-term management (10). This also emphasizes the need for good evaluation of the culprit lesion in the clinical setting (11). In addition, lesions in a different characteristic coronary artery (e.g., left circumflex originating from the right cusp) were rarely found to be culprit vessels in STEMI, an indication of the heterogeneity of lesions (12). The potential connection of new biomarkers (specific indicators) with vulnerability to the severity of STEMI is also being investigated. In particular, the uric acid/creatinine ratio in serum is noted as a potential outcome predictor for patients with multivascular disease and indicates some of the generalized renal and metabolic effects of coronary disease (13). It is important to emphasize that the angiographic assessment of culprit lesions goes on to potentially include at least the infarcted heart attack and its true extent of clinically signaled potentially infarcted areas at the start of STEMI. The reason is that atherothrombosis in its acute stage signifies an early event, including vasoconstriction and inflammation, and it offers inaccurate misinformation on stenosis (14). Given this potential, some of these concerns have led to evaluating stenosed arteries with functional measures

like quantitative flow ratio (QFR) and fractional flow reserve (FFR), moving forward in the flow of established measurement to evaluate stenosed arteries in a feed-forward, dutiful way in iterative decision making in the interventions (15). This phenomenon occurs as a result of simultaneous occlusion of both coronary arteries, described similarly to a common STEMI, but with a much higher risk. This complicates the identification of culprit lesions (16).

Diseases also improve alongside treatments. For example, a decrease in low-density lipoprotein cholesterol through treatment with a PCSK9 inhibitor resulted in a decrease in overall lipids and may also improve nonculprit lesions, thereby acknowledging a potential pathogenic treatment to mitigate future risks (17,18). Electrocardiography (ECG) - the mainstay of diagnostics for STEMI continues to be a key investigative tool. Research has demonstrated a good correlation between ECG results and angiographic results. Despite advances in technology, the ECG continues to supply important, real-time information as to which artery may be blocked and allows for prompt treatment (19). Overall, current evidence provides impetus to consider the severity of blockages in culprit coronary arteries during the acute STEMI. While older studies had shown that mild to moderate blockages could still cause heart attacks because of plaque rupture, a more recent body of research calls attention to the fact that severe blockages are common in culprit lesions.

Objective: To determine the frequency and severity of stenosis in culprit coronary arteries amongst patients with acute ST elevation myocardial infarction undergoing primary percutaneous coronary intervention at NICVD Karachi.

MATERIALS AND METHODS

Study Design: Descriptive Cross-sectional Study.

Study Setting: Cardiology department at the National Institute of Cardiovascular Diseases (NICVD), Karachi.

Duration of the Study: The study carried out over the duration of six months from October 4, 2019 to April 3, 2020.

Inclusion Criterion: Patients aged 35 to 70 years of either sex, presenting with chest pain for greater than 30 minutes, had ST elevation myocardial infarction (STEMI) and had emergent percutaneous coronary intervention (PCI) procedures can be included in the study. Pre-selected patients met the clinical and angiographic criteria to ensure patient uniformity.

Exclusion Criteria: Patients with a history of myocardial infarction, previous PCI or coronary artery bypass grafting, electrolyte abnormalities, visible thrombus after thrombus aspiration, failed guidewire crossing, conditions that affect the ST-signal (i.e., myocarditis, pericarditis, hypothermia, amiodarone therapy) were excluded.

Methods: All eligible patients who met the inclusion criteria were enrolled after giving informed consent. Each patient with STEMI had an emergency percutaneous coronary intervention (PCI) following standard protocols. They received pretreatment with aspirin (300 mg) and clopidogrel (300 mg) before PCI. The study performed coronary angiography through the radial or femoral artery using 6F or 7F sheaths. After sheath insertion, administered intravenous heparin boluses (60–100 U/kg). The use of glycoprotein IIb/IIIa inhibitors was at the operator's discretion. The study carried out manual thrombus aspiration in patients with TIMI flow grade 0–1 or visible thrombus using aspiration catheters. The study assessed coronary flow based on TIMI criteria. It analyzed angiographic images with a quantitative coronary analysis system (QAngio XA 7.3, Medis, Netherlands) to determine the reference vessel diameter, minimal luminal diameter, lesion length, area stenosis, and diameter stenosis.

Results

A total of 181 patients took part in this study to find out how often severe stenosis occurs in the culprit coronary arteries of patients with acute ST elevation myocardial infarction (STEMI) who are undergoing primary PCI. The average age of the patients was 52.7 years, with a standard deviation of 10.3 years, and their ages ranged from 35 to 70 years. When looking at gender, 122 patients (67.4%) were male and 59 patients (32.6%) were female.

Table 1: Distribution of Patients by Age and Gender

Variable	Mean ± SD / Frequency (%)
Age (years)	52.7 ± 10.3
Male	122 (67.4%)
Female	59 (32.6%)

The angiographic assessment showed important quantitative results. The mean reference vessel diameter was 3.3 ± 0.5 mm. The minimal lumen diameter measured 1.1 ± 0.3 mm.

Table 2: Angiographic Quantitative Measurements

Parameter	Mean ± SD
Reference Vessel Diameter	3.3 ± 0.5 mm
Minimal Lumen Diameter	1.1 ± 0.3 mm
Lesion Length	21.1 ± 9.5 mm
Area Stenosis	77.6 ± 6.6 %
Diameter Stenosis	94.6 ± 2.7 %

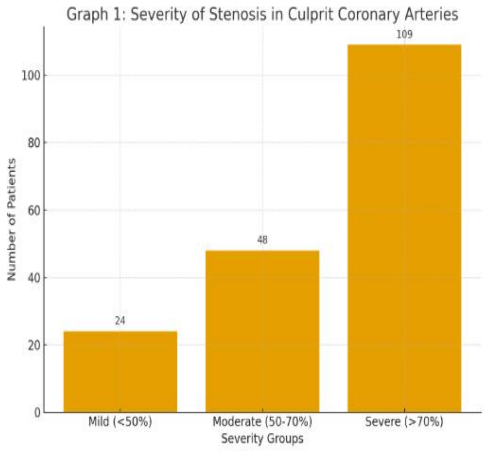
Before PCI, 40 patients (22.1%) had TIMI grade 0 flow. 71 patients (39.3%) had TIMI grade 1. 20 patients (11.0%) showed TIMI grade 2. Finally, 50 patients (27.6%) had TIMI grade 3 flow. Thrombus burden grading gave more insight into the severity of the lesions. Most patients had higher thrombus loads.

Table 3: Distribution of TIMI Flow Grades

TIMI Flow Grade	Frequency (%)
Grade 0	40 (22.1%)
Grade 1	71 (39.3%)
Grade 2	20 (11.0%)
Grade 3	50 (27.6%)

When patients were grouped by the severity of stenosis, 109 patients (60.2%) were classified as having severe stenosis (>70%). Another 48 patients (26.5%) had moderate stenosis (50–70%), and 24 patients (13.3%) had mild stenosis (<50%). These findings show that most STEMI patients undergoing PCI experienced significant narrowing of the culprit artery. To further illustrate this distribution, a bar graph was created to display the relative frequencies of the stenosis severity categories.

Graph 1: Severity of Stenosis in Culprit Coronary Arteries



Stratification by demographic and angiographic characteristics revealed that severe stenosis occurred in both genders and all age groups, although there were some differences. Among males, 69.7% had severe stenosis compared to 45.8% of

females. When looking at age, patients older than 50 years were more often found in the severe stenosis group than younger patients. Further subgroup analysis indicated that patients with TIMI grades 0 and 1 were mostly found among those with severe stenosis, while patients with higher TIMI grades (2 and 3) were more often linked to moderate or mild stenosis. Likewise, thrombus burden grades 4-5 were strongly connected to severe stenosis, highlighting the high clot load in culprit lesions.

Discussion

The aim of the study was to determine the severity of stenosis of the proximal coronary arteries in patients with acute ST elevation myocardial infarction (STEMI). The results indicated that most of the patients had severe stenosis followed by moderate stenosis and mild stenosis. These results provide a contribution to the long-standing discussion as to what degree of coronary stenosis is required to cause STEMI, and support the idea that significant stenosis contributes to the development of such cases. In the early years, myocardial infarction was considered to have plaque rupture primarily at locations of mild-to-moderate coronary stenosis. However, in recent years, technology has progressed, and patient data has become better, causing this OP to have to change its outlook, showing that when severe stenosis occurs, it is more common in the regions involved. Achieving better diagnosis of culprit lesions in STEMI using deep learning and artificial intelligence. Studies of non-ST elevation syndromes have further documented the challenge of detecting culprit lesions in multivessel disease cases and highlighted the necessity of appropriate angiographic and physiological assessment for differentiating between ischemic and non-ischemic lesions (2).

Our results are consistent with reports that patients presenting for STEMI often have more coronary atherosclerosis than non-STEMI patients. This is demonstrated by improvement in Gensini and SYNTAX but not in SYNTAX in non-culprit vessels. (3) Assessments carried out with the optical coherence tomography (OCT) also confirm this difference. They demonstrate that STEMI frequently is associated with thin-cap fibroatheromas and red thrombi, which are susceptible, and authors indicate that this indicates an high level of vulnerability and blocking. In this study, the high degree of severe stenosis adds to the notion that severe atherosclerotic narrowing of the arteries, particularly with unstable plaque, is a central cause to the etiology of STEMI. The size of the vessel is also important in determining the outcome. Although this study did not directly analyse vessel size in our population, the prevalence of severe stenosis links to previous evidence between narrowing and reduced flow and subsequent heart damage.

The treatment of culprit and non-culprit lesions is still debated. Complete revascularization strategies are now considered on a greater basis than interventions that address only the culprit lesion (6). This is particularly important in patients with multi-vessel disease, where decision-making on the treatment for additional lesions besides the culprit artery can have enormous contributions to the outcome. Imaging studies using non-invasive modalities have provided potential in identifying the culprits with the use of strain echocardiography by assessing the myocardial deformation. This offers a helpful approach for patients with unclear angiographic results (7). Additionally, congenital coronary anomalies like unusual right or left circumflex arteries may act as potential culprit vessels in STEMI, even though they are rare (8, 12). This study excluded patients with such anomalies, but these cases highlight the variety of anatomical differences that can lead to myocardial infarction.

The evaluation of residual non-infarct-related stenoses has become more important. Studies that compare myocardial perfusion with invasive hemodynamic measures show

that the severity observed through angiography does not always match its physiological significance (9). Nevertheless, our results suggest that culprit lesions in STEMI are mostly severe, which aligns with findings from the COMPLETE trial. In that trial, complete revascularization led to better quality of life relating to angina compared to strategies focused solely on the culprit lesions (10). Local studies from Pakistan have reported similar findings, showing that angiographic measures like TIMI flow and lesion characteristics strongly relate to severity, emphasizing the need to evaluate culprit lesion stenosis in regional populations (11). In our study, a high thrombus burden and TIMI flow grades of 0–1 were most often linked with severe stenosis, consistent with earlier findings. This is crucial in clinical practice since such patients may need more aggressive treatment. The study have also explored biochemical markers for their link to STEMI severity. The serum uric acid-to-creatinine ratio has recently been noted as a prognostic factor in patients with multivessel disease.

This suggests that overall metabolic health may influence plaque instability and the severity of stenosis (13). Additionally, angiographic studies show that non-culprit lesions can appear more severe during acute infarction because of temporary vasoconstriction and inflammation. This can lead to overestimating stenosis (14). Rare cases of double coronary occlusion presenting as STEMI have been reported, emphasizing the challenges in identifying culprit lesions and the necessity for detailed angiographic analysis (16). In terms of prevention, medications like PCSK9 inhibitors have shown promise for not only lowering lipids but also improving non-culprit lesions. This could reduce the chances of future severe stenosis (17). Electrocardiography remains a key diagnostic tool. Several studies confirm the relationship between ECG localization of infarction and the angiographic identification of culprit vessels (19). In our population, ECG findings guided the initial diagnosis and targeting of lesions before confirming them angiographically. This highlights the need to combine clinical, electrocardiographic, and angiographic data to effectively assess the severity of culprit lesions. Overall, this study shows that severe stenosis is very common among patients with STEMI. This contrasts with earlier beliefs that mild or moderate stenosis was the most frequent issue.

Conclusion

The study showed that significant stenosis (narrowing) of the main coronary artery was most common in patients with acute ST elevation myocardial infarction (STEMI) who received primary percutaneous coronary intervention. Moderate and mild narrowing were less frequent, showing that massive narrowing is still the dominating cause of acute coronary narrowing. Miles Kimiel, MD, MPH, and AIEC, and colleagues noted that 3% (severe narrowing) and 13% (low TIMI flow grades) had an increased thrombus load, underscoring its critical importance in the disease biology of STEMI. These findings were repeated in both international and regional studies, which seem to support the view that acute infarction is mainly caused by combinations of severe narrowing and unstable plaque. The findings also emphasize the need for early identification and appropriate treatment, as well as the importance of including all the obstructed vessels in patients suffering from polyvessel disease. Intracardiac evaluation of the myocardium by using a combination of angiographic, physiologic, and clinical evaluation may help clinicians evaluate the risk and tailor individual treatment strategies, thereby improving the survival and quality of life of STEMI patients.

References

1. Tseng LM, Chuang CY, Chua SK, Tseng VS. Identification of coronary culprit lesion in ST elevation myocardial infarction by using deep learning. IEEE Journal of

Translational Engineering in Health and Medicine. 2022 Dec 8;11:70-9.

2. Balbi MM, Scarparo P, Tovar MN, Masdjedi K, Daemen J, Den Dekker W, Ligthart J, Witberg K, Cummins P, Wilschut J, Zijlstra F. Culprit lesion detection in patients presenting with non-ST elevation acute coronary syndrome and multivessel disease. Cardiovascular Revascularization Medicine. 2022 Feb 1;35:110-8.

3. Tanaka T, Miki K, Akahori H, Imanaka T, Yoshihara N, Kimura T, Yanaka K, Asakura M, Ishihara M. Comparison of coronary atherosclerotic disease burden between ST-elevation myocardial infarction and non-ST-elevation myocardial infarction: non-culprit Gensini score and non-culprit SYNTAX score. Clinical Cardiology. 2021 Feb;44(2):238-43.

4. Mani A, Ojha V, Sivadasanpillai H, Sasidharan B, Ganapathi S, Valaparambil AK. Culprit Lesion Morphology on Optical Coherence Tomography in ST-elevation Myocardial Infarction vs Non ST-elevation Myocardial Infarction–A Systematic Review of 7526 Patients. Journal of the Saudi Heart Association. 2023 Mar 9;35(1):40.

5. Lechner I, Reindl M, Tiller C, Holzknecht M, Oberhollenzer F, Mayr A, Bauer A, Metzler B, Reinstadler SJ. Culprit Lesion Vessel Size and Risk of Reperfusion Injury in ST-Segment Elevation Myocardial Infarction: A Cardiac Magnetic Resonance Imaging Study. Journal of the American Heart Association. 2024 Feb 6;13(3):e033102.

6. Gerculy R, Mitra N, Szabó E, Opincariu D, Chițu M, Benedek I. Management of culprit and non-culprit lesions in acute coronary syndrome. Journal of Interdisciplinary Medicine. 2021;6(1):32-6.

7. Guaricci AI, Chiarello G, Gherbesi E, Fusini L, Soldato N, Siena P, Ursi R, Ruggieri R, Guglielmo M, Muscogiuri G, Baggiano A. Coronary-specific quantification of myocardial deformation by strain echocardiography may disclose the culprit vessel in patients with non-ST-segment elevation acute coronary syndrome. European Heart Journal Open. 2022 Mar 1;2(2):oeac010.

8. Kersey CB, Oyetunji S, Don CW. Anomalous Right Coronary Artery: Culprit or Innocent Bystander?. Case Reports in Cardiology. 2025;2025(1):1450803.

9. Vacha J, Spacek M, Kaminek M, Hutyra M, Nykl R, Sluka M, Taborsky M. Comparison of myocardial perfusion study and invasive hemodynamic measurement of the significance of non-infarct-related residual stenoses in ST elevation myocardial infarction patients. Biomedical Papers. 2024 Jun 24.

10. Mehta SR, Wang J, Wood DA, Spertus JA, Cohen DJ, Mehran R, Storey RF, Steg PG, Pinilla-Echeverri N, Sheth T, Bainey KR. Complete Revascularization vs culprit lesion–only percutaneous coronary intervention for angina-related quality of life in patients with ST-segment elevation myocardial infarction: results from the COMPLETE randomized clinical trial. JAMA Cardiology. 2022 Nov 1;7(11):1091-9.

11. Sial JA, Ali N, Shehzad M. Angiographic characteristics of culprit lesions in infarct related artery and correlation of TIMI score with SYNTAX score to predict extent and severity of coronary artery disease in patients undergoing primary percutaneous coronary interventions. Pakistan Journal of Medical Sciences. 2024 Jan;40(1Part-I):20.

12. Vadher A, Malik N, Pannikottu K, Kondur A, Kambhatla S, Malik N. Anomalous Left Circumflex Artery Originating From Right Coronary Cusp as Culprit Vessel in ST-Elevation Myocardial

Infarction (STEMI). Cureus. 2024 Aug 5;16(8).

13. Gumusdag A, Kalyoncuoglu M, Oguz H, Apaydin Z, Kilinc AY, Karaca M, Uzman O, Yildirimturk O. Prognostic role of the serum uric acid-to-serum creatinine ratio in patients with st-elevation myocardial infarction and multivessel coronary artery disease. Cardiology Journal. 2025 Sep 5.

14. Othman F, Abid AR, AlQahtani A, Gehani A, Suwaidi JA, Arabi A. Exaggeration of non-culprit coronary artery stenosis in the setting of primary percutaneous coronary intervention: A single center observational study. Research Reports in Clinical Cardiology. 2021 Nov 15:67-73.

15. Barauskas M, Žiubrytė G, Jodka N, Unikas R. Quantitative flow ratio for assessment of non-culprit coronary artery lesions during percutaneous coronary intervention (PCI) in 79 patients diagnosed with ST-elevation myocardial infarction (STEMI): a study from a single center in Lithuania. Medical Science Monitor: International Medical Journal of Experimental and Clinical Research. 2023 Apr 18;29:e939360-1.

16. Marchi E, Muraca I, Cesarini D, Pennesi M, Valenti R. Double coronary artery occlusion presenting as inferior ST segment elevation myocardial infarction and Wellens syndrome type A: a case report. European Heart Journal-Case Reports. 2024 Aug;8(8):ytae394.

17. Mensink FB, Los J, Oemrawsingh RM, von Birgelen C, Ijsselmuiden A, Meuwissen M, Cheng JM, van Wijk DF, Smits PC, Paradies V, van der Heijden DJ. Functional and morphological improvement of significant non-culprit coronary artery stenosis by LDL-C reduction with a PCSK9 antibody: Rationale and design of the randomized FITTER trial. Heliyon. 2024 Oct 15;10(19).

18. Pustjens TF, Veenstra L, Camaro C, Ruiters AW, Lux Á, Ruzsa Z, Piroth Z, Ilhan M, Vainer J, Gho B, Winkler PJ. Fractional Flow Reserve–Guided Complete vs Culprit-Only Revascularization in Non–ST-Elevation Myocardial Infarction and Multivessel Disease: The SLIM Randomized Clinical Trial. JAMA. 2025 Aug 31.

19. Sharma P, Mahesh NK, Prasad V. Culprit Artery Localisation in Electrocardiography and its Correlation with Angiography in Acute ST Elevation Myocardial Infarction. European Journal of Cardiovascular Medicine. 2023 Jan 1;13(1).

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