Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Isolated ST Elevation in Lead aVR Revealing Left Main Equivalent Ischemia in a Diabetic Patient Presenting Without Chest Pain

Aakash Acharya
Prime West Consortium, Centinela Hospital Medical Center
Himal Bikram Bhattarai
Prime West Consortium, Centinela Hospital Medical Center
Justin Besharat
Prime West Consortium, Centinela Hospital Medical Center
Raman Goit
goitraman2025@gmail.com

Dhulikhel Hospital
Ali Morshedi-Meibodi
Prime West Consortium, Centinela Hospital Medical Center

Case Report

Keywords:

Posted Date: June 10th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9588485/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background: ST-segment elevation in lead aVR with diffuse ST depression represents global subendocardial ischemia and is strongly associated with left main or multivessel coronary artery disease. Recognition of this high-risk electrocardiographic pattern is essential, particularly in patients with atypical acute coronary syndrome presentations.

Case Presentation: A 67-year-old male with type 2 diabetes mellitus (HbA1c 5.7%) presented with progressive dyspnea without associated chest pain. He tested positive for influenzae A. laboratory evaluation demonstrated mild leukocytosis and elevated serum lactate consistent with tissue hypoperfusion. EKG demonstrated isolated ST elevation in lead aVR with diffuse ST depression in leads I, aVL, and V4-V6. Initial high-sensitivity troponin I was 3,480 ng/L (3-53 ng/L) and peaked at 69,935 ng/L. Transthoracic echocardiography showed reduced left ventricular ejection fraction (35-40%) with global hypokinesis. Coronary angiography revealed multivessel obstructive coronary artery disease involving mid LAD (90-95% Stenosis), left circumflex bifurcation (second obtuse marginal), and diffuse right coronary artery stenosis.

Conclusion: This case illustrates the clinical significance of isolated ST elevation in aVR as a left main equivalent, particularly in diabetic patients with atypical presentations and concurrent systemic infection.

Introduction

Lead aVR has emerged as a critical yet often underappreciated component of electrocardiographic analysis in acute coronary syndromes. ST-segment elevation in lead aVR accompanied by widespread ST depression reflects diffuse subendocardial ischemia and is strongly predictive of left main coronary artery stenosis, proximal left anterior descending artery disease, or severe multivessel coronary artery disease [1–3]. This pattern carries high short-term mortality and is frequently considered a STEMI equivalent requiring urgent invasive evaluation [4].

Diabetes mellitus predisposes patients to silent myocardial ischemia due to autonomic dysfunction, resulting in atypical presentations such as dyspnea without chest pain [5]. Furthermore, influenza infection has been associated with increased cardiovascular events via systemic inflammation, plaque destabilization, endothelial dysfunction, and prothrombotic activation [6–8]. We report a case in which influenza A infection precipitated acute hypoxic respiratory failure and unmasked severe multivessel coronary artery disease in a patient presenting without chest pain but with a high-risk aVR electrocardiographic pattern.

Case Presentation

A 67-year-old man with a history of type 2 Diabetes Mellitus presented with progressively worsening shortness of breath. Three days prior to admission, he developed upper respiratory symptoms including

sore throat, rhinorrhea, fever, and dry cough, and tested positive for influenza A. He denied chest pain, palpitations, syncope, or any prior history of coronary artery disease. On arrival, he was tachypneic and in moderate respiratory distress, with a heart rate of 127 beats per minute, respiratory rate of 32 breaths per minute, blood pressure of 160/86 mmHg, and oxygen saturation of 96% while receiving noninvasive positive pressure ventilation.

laboratory evaluation demonstrated mild leukocytosis and elevated serum lactate consistent with tissue hypoperfusion. (Table 1) Initial EKG with sinus tachycardia with HR of 127 beats/min which one hour later demonstrated ST elevation in lead aVR with diffuse ST depression in leads I, aVL, and V4–V6: raising concern for left main coronary artery involvement or severe multivessel ischemia; and resolution of EKG changes post cardiac cath (Fig. 1, 2, 3) Initial high-sensitivity troponin I was 3,480 ng/L (3–53 ng/L) and peaked at 69,935 ng/L. (Fig. 4) He tested positive for influenzae A and chest radiography revealed bilateral patchy infiltrates. Chest CTA/Thorax without any evidence of pulmonary embolism. Transthoracic echocardiography with LVEF of 40–45% with severe aortic valve stenosis with Vmax 4.8m/s(Fig. 5)

Coronary angiography demonstrated severe multivessel coronary artery disease, characterized by critical mid–left anterior descending (LAD) artery stenosis (90–95%), significant left circumflex (LCx) bifurcation disease involving the obtuse marginal branches, and diffuse right coronary artery (RCA) disease with notable involvement of the posterior descending artery (PDA) (Fig. 6). The patient subsequently underwent coronary artery bypass grafting (CABG) along with aortic valve replacement.

Table 1
Initial lab parameters per ED recording

Parameter	Value	Reference range
High sensitivity troponin I	3480◊69935ng/L(Peak)	3-53ng/L
BNP	822pg/mL	< 100pg/mL
Lactate	6.2mmol/L	0.5-2.0mmol/L
WBC	$12.4x10^9$/L	$4.0\text{-}11.0x10^9$/L
Hgb	13.5g/dL	13.0-17.0g/dL
K+	3.4mmol/L	3.5-5.0mmol/L
HCO3	19mmol/L	22-28mmol/L
Glucose	318mg/dL	70-140mg/dL
Creatinine Kinase	1818IU/L(Peak)	46-171IU/L
HbA1c	5.7%	< 6.5%

Discussion

ST-segment elevation in lead aVR accompanied by diffuse ST-segment depression represents a high-risk electrocardiographic pattern reflecting global subendocardial ischemia. This pattern has been consistently associated with left main coronary artery (LMCA) disease, proximal left anterior descending (LAD) artery stenosis, or severe multivessel coronary artery disease (CAD) [1–3]. The underlying mechanism involves circumferential subendocardial ischemia resulting in a reciprocal injury vector directed toward the right shoulder, manifesting as ST elevation in aVR [1, 4]. Several studies have demonstrated that ST elevation ≥ 1 mm in aVR in the setting of widespread ST depression predicts significant LMCA or three-vessel disease and is associated with increased in-hospital mortality [2, 3, 5].

Although not traditionally classified as a classic ST-elevation myocardial infarction (STEMI), this electrocardiographic pattern is increasingly regarded as a STEMI equivalent due to its strong association with critical coronary anatomy and adverse outcomes [4, 6]. Contemporary guidelines emphasize that patients with diffuse ST depression and concomitant ST elevation in aVR should undergo urgent invasive evaluation because of the high probability of left main or multivessel ischemia [6, 7]. In our patient, the presence of isolated aVR ST elevation with diffuse ST depression appropriately prompted urgent coronary angiography, which confirmed extensive multivessel obstructive CAD with critical LAD involvement.

Diabetes mellitus significantly increases the risk of silent or atypical myocardial ischemia. Autonomic neuropathy blunts anginal perception, leading to presentations characterized by dyspnea, fatigue, or heart failure rather than chest pain [8, 9]. Silent ischemia is more prevalent among diabetic patients and carries a worse prognosis due to delayed recognition and treatment [8]. In this case, the absence of chest pain despite profound troponin elevation and high-risk ECG findings underscores the importance of maintaining a high index of suspicion for acute coronary syndromes (ACS) in diabetic individuals presenting with unexplained dyspnea.

Additionally, concurrent influenza A infection likely contributed to plaque destabilization and myocardial injury. Viral infections are recognized triggers for acute cardiovascular events through multiple mechanisms, including systemic inflammation, cytokine-mediated endothelial dysfunction, increased metabolic demand, prothrombotic activation, and sympathetic surge [10–12]. Epidemiologic data have demonstrated a markedly increased risk of myocardial infarction in the first week following laboratory-confirmed influenza infection [10]. Influenza-associated myocardial stress may therefore have acted as a precipitating factor in this patient with underlying severe multivessel CAD.

The coexistence of reduced left ventricular ejection fraction and low-flow, low-gradient severe aortic stenosis further complicated the clinical picture. Severe aortic stenosis increases myocardial oxygen demand while limiting coronary perfusion reserve, thereby predisposing to subendocardial ischemia, particularly in the setting of tachycardia or systemic infection [13, 14]. The combined hemodynamic burden of multivessel CAD, aortic stenosis, and systemic inflammatory stress likely amplified global ischemia, explaining the dramatic troponin rise and high-risk ECG pattern.

Conclusion

Isolated ST-segment elevation in lead aVR with diffuse ST depression represents a high-risk electrocardiographic marker of global subendocardial ischemia and frequently reflects left main or multivessel coronary artery disease. In diabetic patients, atypical presentations without chest pain may delay diagnosis unless clinicians recognize this critical ECG pattern. Concurrent systemic infections such as influenza may further precipitate acute coronary events through inflammatory and prothrombotic mechanisms. Prompt identification of this STEMI-equivalent pattern and urgent coronary angiography are crucial for timely revascularization and improved clinical outcomes.

Declarations

Acknowledgements:

Not applicable

Funding:

Not applicable.

Data availability:

The data sets are available from the corresponding author on request.

Ethics approval and consent to participate:

Ethical approval was not required for this case report in accordance with local institutional policies. Written informed consent was obtained from the patient for publication of this case report.

Consent for publication:

Written consent was obtained from the patient for publication and any accompanying images.

Competing interests:

The authors declare that they have no competing interests.

Authors' contributions:

A.A., H.B.B., and J.B. contributed to the conception of the case report, patient data collection, and initial drafting of the manuscript. R.G. performed literature review, data analysis, and contributed to drafting and revising the manuscript. A.M.M. provided critical supervision, contributed to manuscript editing, and reviewed the case for important intellectual content. All authors read and approved the final manuscript.

References

1. Yamaji H, Iwasaki K, Kusachi S, Murakami T, Hirami R, Hamamoto H, et al. Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography. J Am Coll Cardiol. 2001;38(5):1348–54. 10.1016/s0735-1097(01)01563-7.
2. Barrabés JA, Figueras J, Moure C, Cortadellas J, Soler-Soler J. Prognostic value of lead aVR in patients with a first non–ST-segment elevation acute myocardial infarction. Circulation. 2003;108(7):814–9. 10.1161/01.CIR.0000084553.92734.83.
3. Kosuge M, Kimura K, Ishikawa T, Ebina T, Hibi K, Tsukahara K, et al. ST-segment elevation in lead aVR predicts left main or three-vessel disease in patients with non–ST-segment elevation acute coronary syndrome. Am J Cardiol. 2005;95(6):712–4. 10.1016/j.amjcard.2005.01.085.
4. Knotts RJ, Wilson JM, Kim E, Huang HD, Birnbaum Y. Electrocardiographic criteria to differentiate acute left main coronary artery occlusion from proximal left anterior descending artery occlusion. Am J Cardiol. 2013;111(1):1–6. 10.1016/j.ihj.2016.03.025.
5. Nikus KC, Eskola MJ, Virtanen VK, Harju J, Huhtala H, Sclarovsky S, et al. ST-depression with negative T waves in leads V4–V5: a marker of left main coronary artery disease in non-ST elevation ACS. Ann Med. 2010;42(4):268–75. 10.1111/j.1542-474X.2004.93545.x.
6. Collet JP, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt DL, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021;42(14):1289–367. 10.1093/eurheartj/ehaa575.
7. Gulati M, Levy PD, Mukherjee D, Amsterdam E, Bhatt DL, Birtcher KK, et al. 2021 AHA/ACC chest pain guideline. Circulation. 2021;144(22):e368–454. 10.1161/CIR.0000000000001029.
8. Valensi P, Pariès J, Brulport-Cerisier V, Torremocha F, Sachs RN, Vanzetto G, et al. Predictive value of silent myocardial ischemia for cardiac events in diabetic patients. Diabetes Care. 2005;28(11):2722–7. 10.2337/diacare.28.11.2722.
9. Nesto RW, Phillips RT, Kett KG, Hill T, Perper E, Young E, et al. Angina and exertional myocardial ischemia in diabetic and nondiabetic patients. Circulation. 1988;78(6):1157–63. 10.7326/0003-4819-108-2-170.
10. Kwong JC, Schwartz KL, Campitelli MA, Chung H, Crowcroft NS, Karnauchow T, et al. Acute myocardial infarction after laboratory-confirmed influenza infection. N Engl J Med. 2018;378(4):345–53. 10.1056/NEJMoa1702090.
11. Madjid M, Solomon SD, Vardeny O. Influenza and cardiovascular disease: a new opportunity for prevention and the need for further studies. Circulation. 2020;142(10):962–5. https://doi.org/10.1161/01.CIR.0000102380.47012.92.
12. Musher DM, Abers MS, Corrales-Medina VF. Acute infection and myocardial infarction. N Engl J Med. 2019;380(2):171–6. 10.1056/NEJMra1808137.
13. Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease. Circulation. 2021;143(5):e72–227. 10.1161/CIR.0000000000000932.

14. Rajappan K. Mechanisms of coronary microcirculatory dysfunction in aortic stenosis. J Am Coll Cardiol. 2003;41(3):423–9. 10.1161/hc0402.102931.

Figures

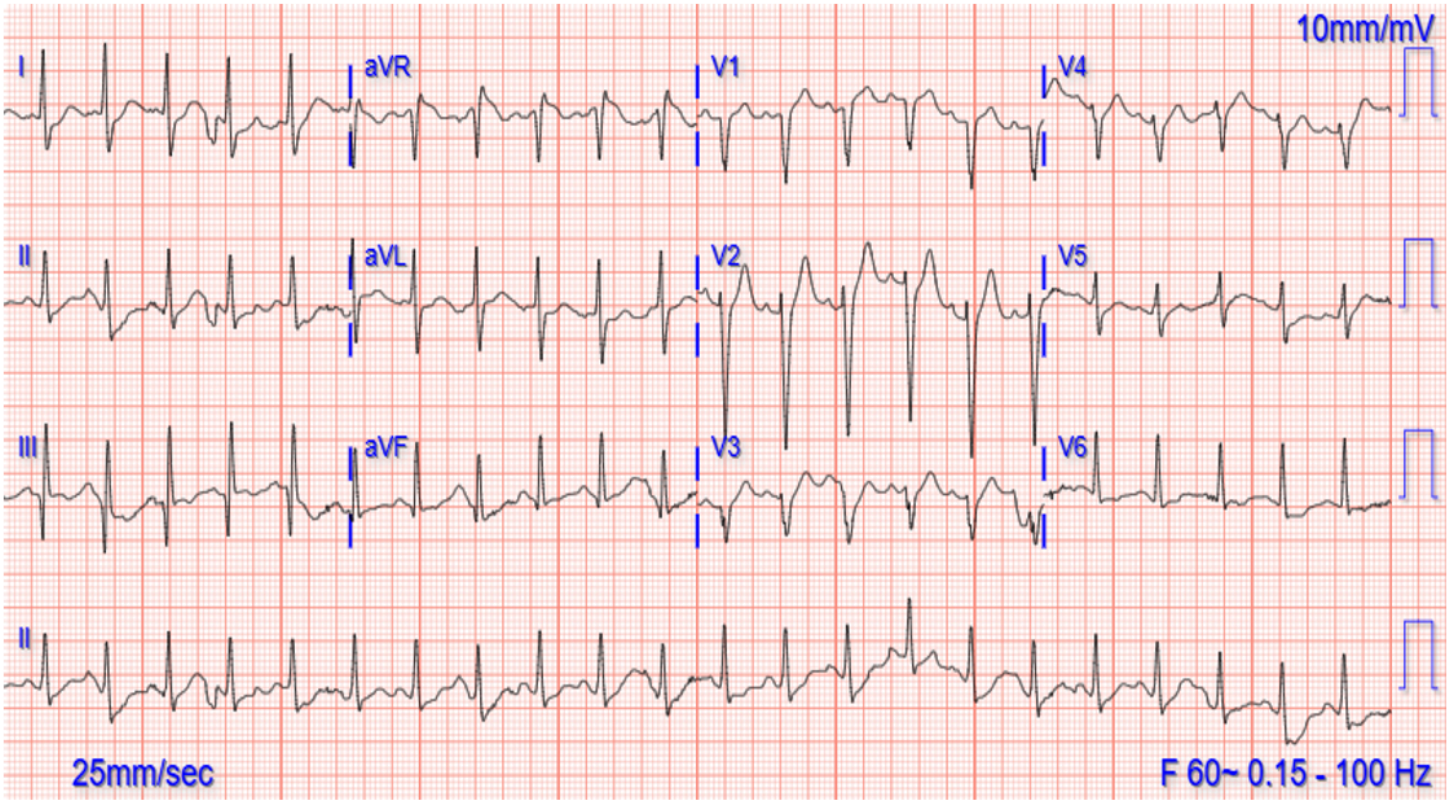


Figure 1

Initial EKG with sinus tachycardia

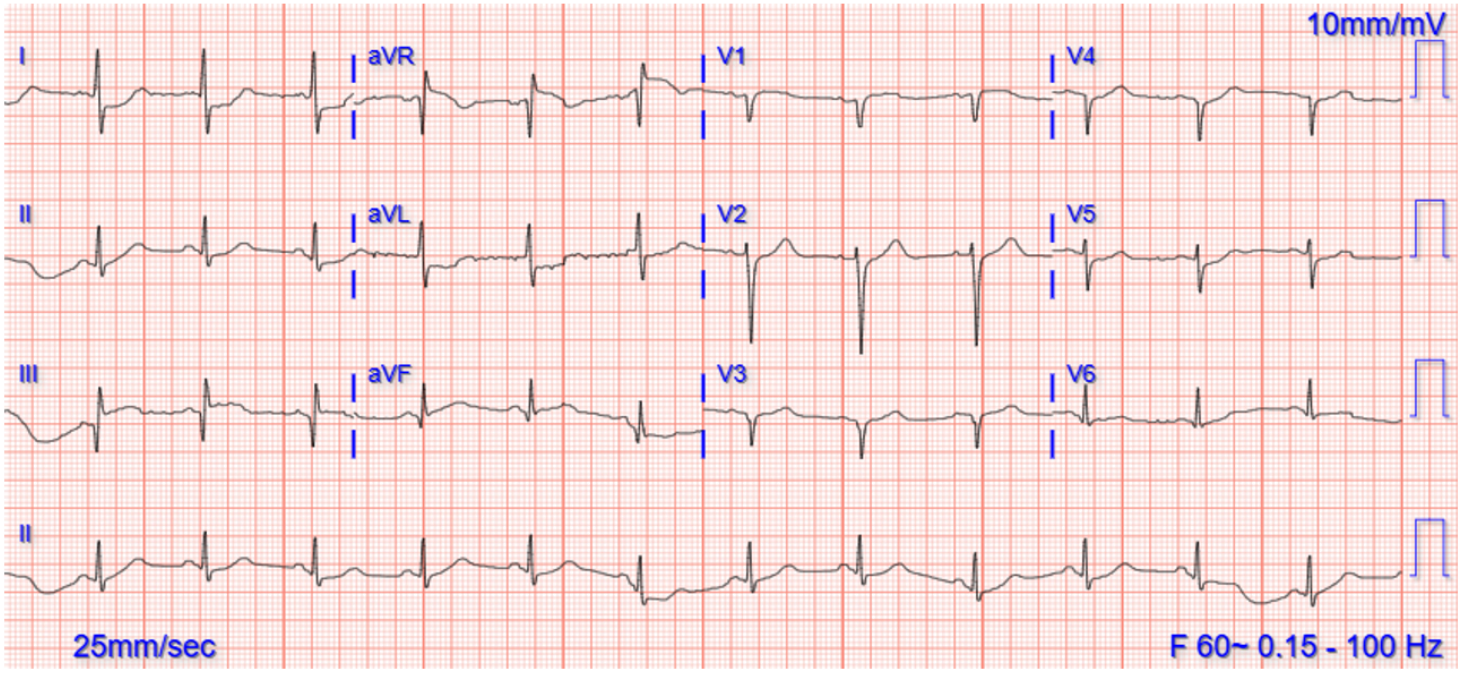


Figure 2

EKG after an hour of presentation with ST elevation on AVR with ST depression on I, avl, and V5

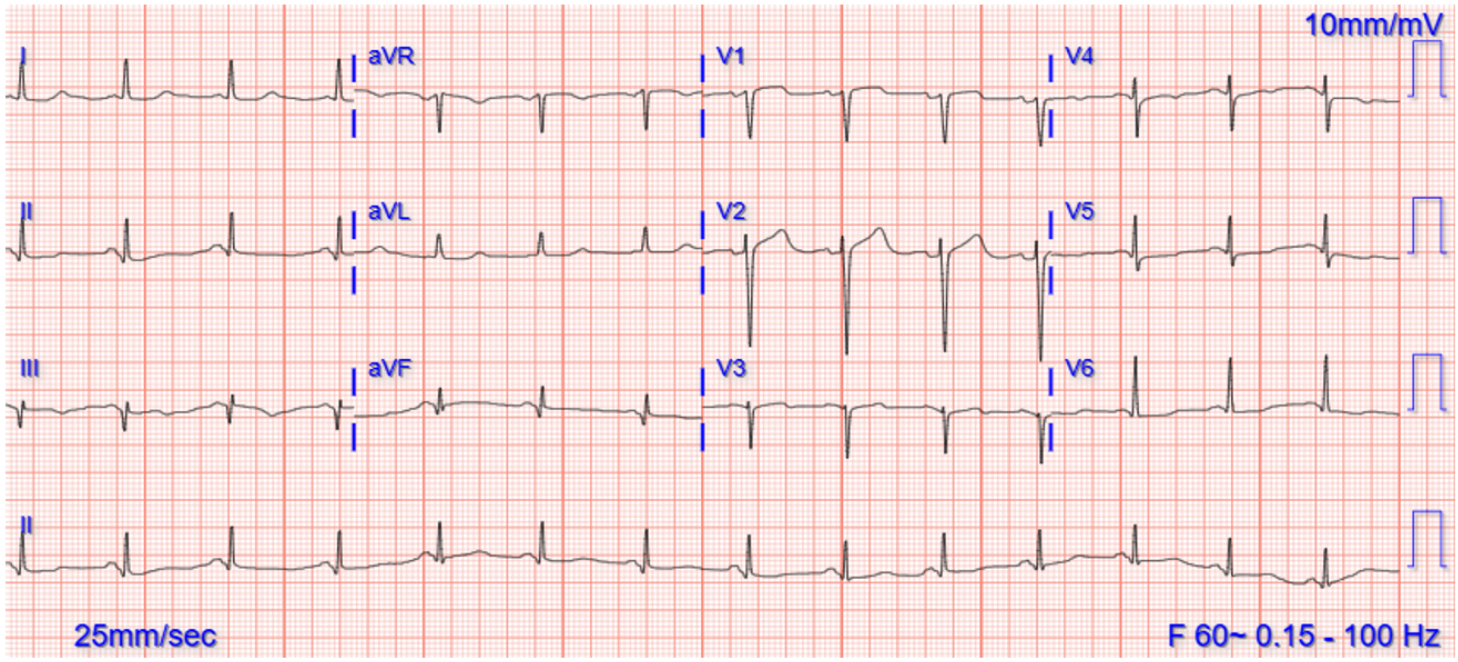


Figure 3

EKG after the cardiac Cath on the 5th day with down trending troponin trend

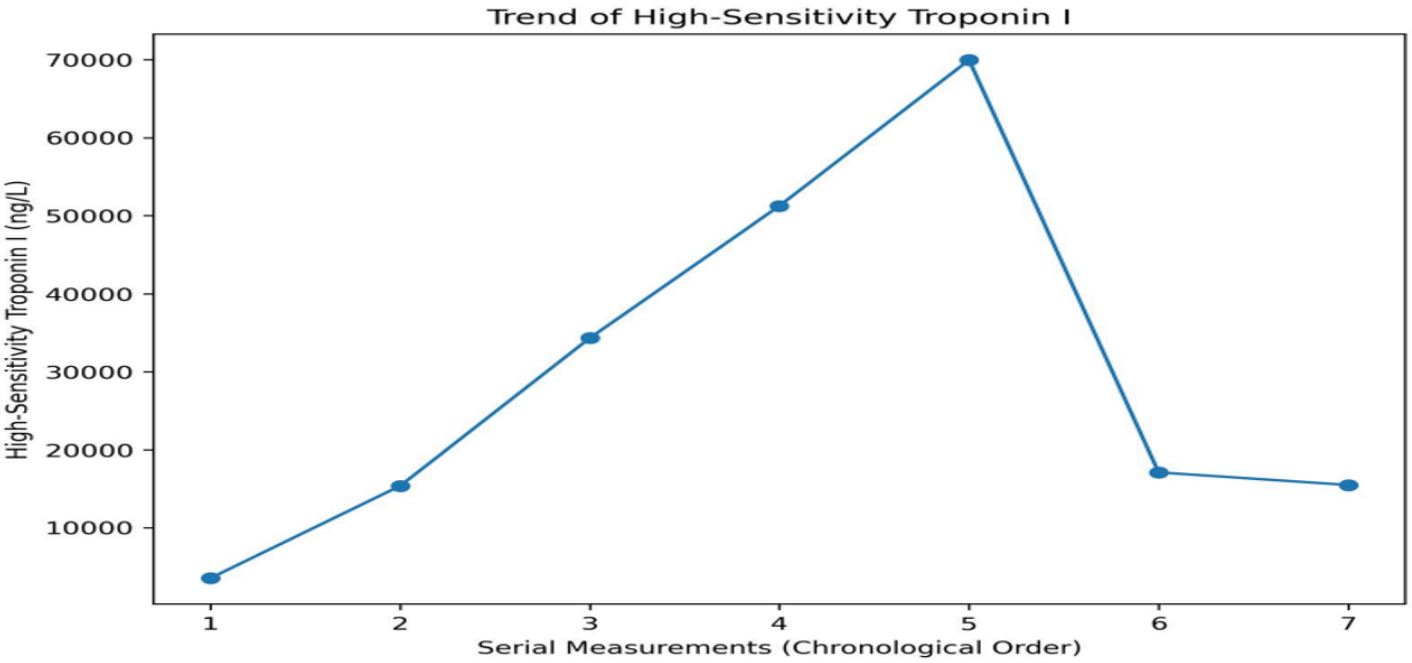


Figure 4

Serial troponin trend from day of admission till discharge

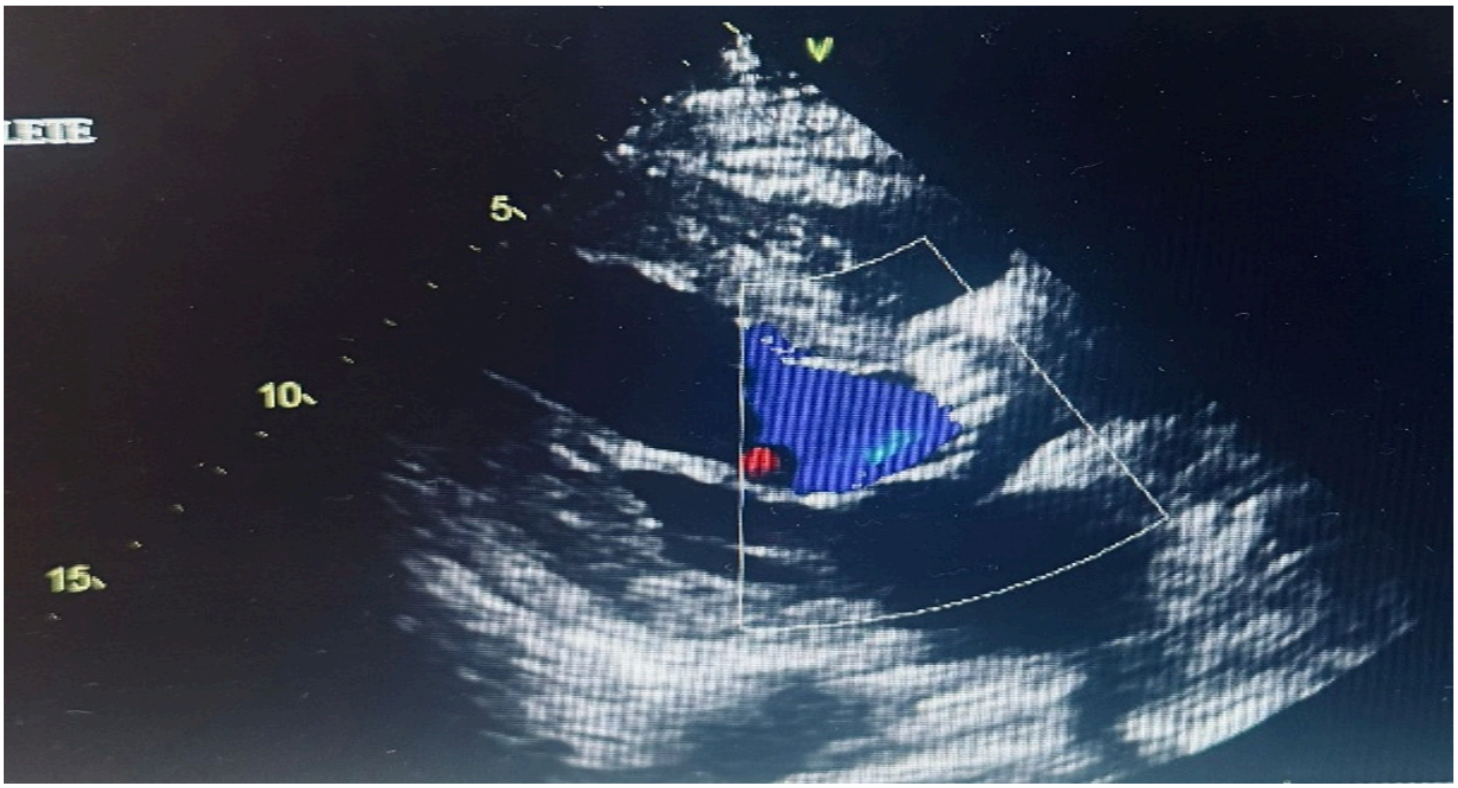


Figure 5

Transthoracic echocardiography with LVEF of 40-45% with severe aortic valve stenosis

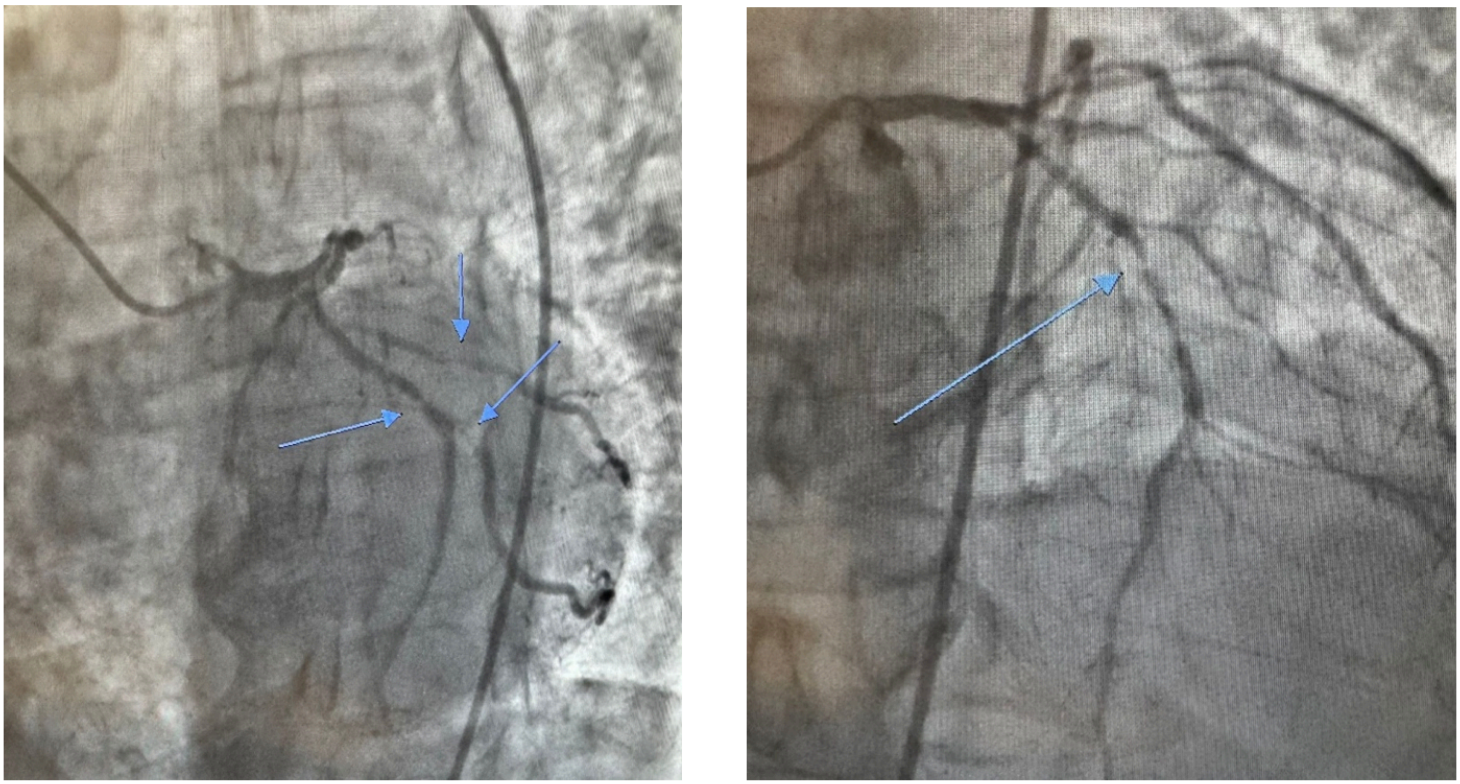


Figure 6

Coronary angiography; severe multivessel coronary artery disease with critical mid–left anterior descending (LAD) artery stenosis (90–95%), significant left circumflex (LCx) bifurcation disease involving the obtuse marginal branches, and diffuse right coronary artery (RCA) disease with notable involvement of the posterior descending artery (PDA)