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Case Reports

Myocarditis Mimicking STEMI Complicated by Complete Atrioventricular Block: Diagnostic and Therapeutic Insights

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Abstract

Background: Myocarditis, or myocardial inflammation, may share similar characteristics to Acute Coronary Syndrome (ACS), particularly ST-Elevation Myocardial Infarction (STEMI). This condition is further augmented when a Complete Atrioventricular Block (CAVB) is present. Despite being rare, this condition may pose additional diagnostic and therapeutic challenges.

Case Illustration: We report a 54-year-old woman with fatigue, dyspnea, fever, nausea, and watery diarrhea for three days. Upon admission, she experienced hypotension, pulmonary congestion, and a complete Atrioventricular (AV) block, with ST-segment elevation seen on the lateral leads. Initial laboratory results revealed markedly elevated high-sensitivity troponin T and C-reactive Protein (CRP). Bedside echocardiography showed a prominently reduced Ejection Fraction (EF) (40%) alongside the presence of regional wall motion abnormalities. Urgent coronary angiography revealed only non-obstructive coronary disease and no obstructive coronary disease. A temporary pacemaker and inotropic support were initiated. Given the presence of systemic prodromal symptoms and the absence of coronary obstruction, myocarditis was strongly suspected. High-dose intravenous methylprednisolone was given as an anti-inflammatory treatment in suspected fulminant myocarditis with cardiogenic shock and complete AV block. Recognizing that immunosuppressive therapy is not routinely recommended for all myocarditis cases, especially without biopsy confirmation. Cardiac magnetic resonance imaging subsequently confirmed myocarditis, demonstrating myocardial edema and subepicardial late gadolinium enhancement. The patient was discharged after receiving guideline-directed medical therapy and tapering corticosteroids, with preserved ventricular function on follow-up 1 month after discharge.

Conclusions: This report illustrates the importance of a stepwise diagnostic approach to differentiate myocarditis from STEMI, particularly when complicated by conduction disturbances such as CAVB. Early recognition and timely initiation of immunosuppressive therapy can lead to favorable outcomes.

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Keywords: Myocarditis, STEMI mimic, atrioventricular block, cardiac magnetic resonance, case report.

Introduction

Myocarditis is a myocardial inflammatory disease that can closely mimic Acute Coronary Syndromes (ACS), particularly ST-Elevation Myocardial Infarction (STEMI). Both may present with chest discomfort, ST-segment elevation, and markedly elevated troponin.[1-2] Diagnostic uncertainty is further increased when myocarditis is complicated by Complete Atrioventricular Block (CAVB), a rare but high-risk manifestation that can resemble infarction with conduction system involvement and may precipitate cardiogenic shock. In such scenarios, rapidly distinguishing myocarditis from STEMI is critical, as management strategies diverge substantially, especially regarding the role of urgent revascularization versus advanced imaging and, in selected cases, immunosuppressive therapy.[1,3]

Distinguishing myocarditis from ACS in such circumstances is crucial, as therapeutic strategies differ markedly. We report a case of acute myocarditis presenting as a STEMI mimic complicated by CAVB, initially managed as ACS, before subsequent multimodal evaluation established the correct diagnosis. This case demonstrates that a systematic diagnostic approach is key to identifying myocarditis, particularly in ACS-like presentations with conduction abnormalities.

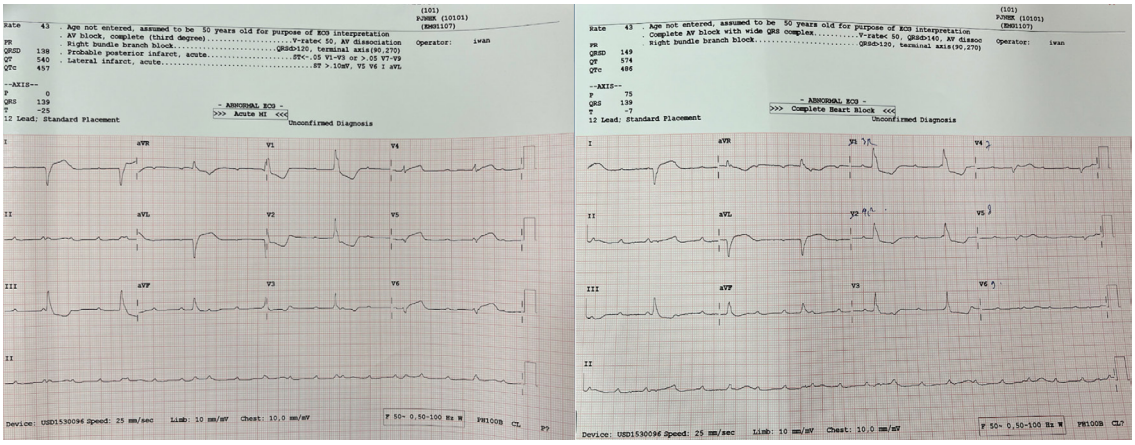

Figure 1. Initial 12-lead electrocardiograms (ECG) on presentation. The tracings demonstrate complete (third-degree) atrioventricular block with a ventricular escape rhythm at 40 bpm, wide QRS complexes with right bundle branch block morphology, and ST-segment elevation in leads I, aVL, V5–V6, with reciprocal ST depression in the inferior leads and right precordial leads. These findings were initially suggestive of acute myocardial infarction with high-grade conduction disturbance.

Case Illustration

A 54-year-old woman was brought into the emergency department with progressive fatigue, dyspnea, fever, nausea, and watery diarrhea for three days. She denied chest pain or palpitations. No history of hypertension, diabetes mellitus, dyslipidemia, or significant family history of cardiovascular disease was recognized. Upon admission, her blood pressure was 90/60 mmHg, heart rate 44 bpm, respiratory rate 28/min, and oxygen saturation 95% on room air. Physical examination revealed bilateral pulmonary rales and signs of dehydration, but no peripheral edema.

Electrocardiography (ECG) demonstrated a CAVB with a ventricular rate of 40 bpm, ST-segment elevation in leads I, aVL, V5–V6, and reciprocal depression in inferior and right precordial leads (Fig 1). Initial laboratory results showed markedly elevated high-sensitivity troponin T (6212 ng/L) and C-reactive Protein (CRP) levels (62 mg/L). Bedside echocardiography showed a prominently reduced Ejection Fraction (EF) (40%) alongside the presence of regional wall motion abnormalities, particularly in the anterolateral and inferolateral segments.

The initial working diagnosis was late-onset STEMI high lateral, complicated by CAVB with ventricular escape rhythm. The patient was initially planned to undergo primary percutaneous coronary intervention; however, the patient's coronary angiography revealed a non-significant coronary artery disease (30% proximal Left Anterior Descending [LAD] stenosis) (Fig 2). To tackle the CAVB, a temporary pacemaker was placed. Myocarditis was strongly suspected afterwards, given the lack of plausible evidence in the patient's

coronary angiography and the presence of systemic prodromal symptoms.

The patient developed cardiogenic shock with Society for Cardiovascular Angiography and Interventions (SCAI) stage C and was started on inotropic therapy. A high-dose intravenous methylprednisolone (500 mg/day for three days, followed by tapering) was initiated alongside antibiotics for concomitant pneumonia. Dobutamine infusion and supportive care were continued. The patient demonstrated gradual clinical improvement, with a reduction of CRP and hs troponin T level (Table 1), EF recovery to 56%, and restoration of sinus rhythm. Blood culture was performed to search for the aetiology of myocarditis, but it showed no bacterial growth.

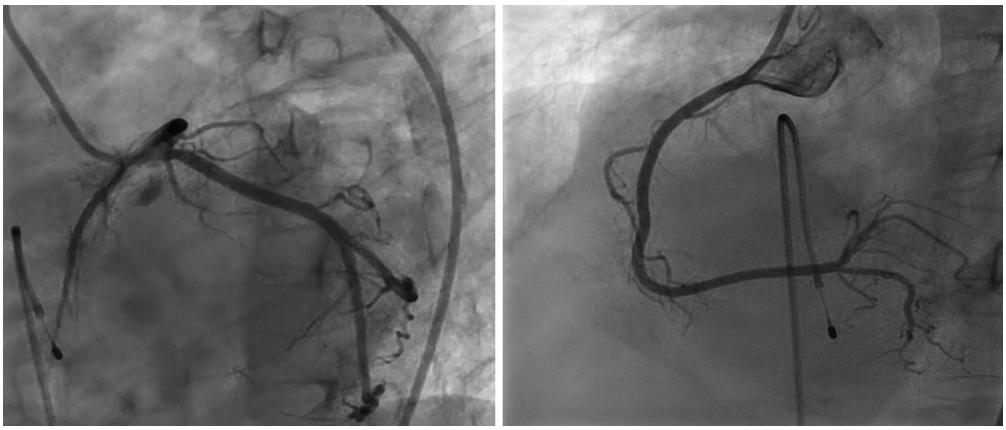

Figure 2. Coronary angiography images. (Left) Left coronary system demonstrating normal left main (LM), left anterior descending (LAD) artery with only non-obstructive coronary disease, and normal left circumflex (LCX). (Right) Right coronary artery (RCA) without significant stenosis. Overall, no evident obstructed coronary artery was seen, supporting the diagnosis of myocarditis.

Table 1. Laboratory investigations from day of admission to discharge.

Blood investigations	Day 0	Day 1	Day 2	Day 3	Day 4	Day 6	Day 8	Day 9	Day 10
Haemoglobin (g/dL)	12.5	-	-	-	10.6 ↓	13.3	14.5	-	-
Haematocrit (%)	37.7	-	-	-	31.5 ↓	39.2	43.3	-	-
Leukocyte (/µL)	7720	-	-	-	14640 ↑	15810 ↑	21700 ↑	-	-
Segment (%)	70	-	-	-	94.6 ↑	94.3 ↑	-	-	-
Lymphocytes (%)	19.9	-	-	-	2.3	1.5	-	-	-
Thrombocyte (/µL)	227000	-	-	-	213000	257000	384000	-	-
Albumin (g/dL)	-	3.6	-	-	-	-	-	-	-
Total bilirubin (mg/dL)	0.5	-	-	-	-	-	-	-	-
SGPT (U/L)	-	142 ↑	-	-	191 ↑	-	-	-	-
SGOT (U/L)	-	263 ↑	-	-	96 ↑	-	-	-	-
AFP (U/L)	-	-	73	-	-	-	-	-	-
hs Troponin T (ng/L)	6212 ↑	-	-	-	-	-	-	-	191 ↑
NT-pro BNP (pg/mL)	-	-	61273 ↑	-	-	-	-	-	5373 ↑
Ureum (mg/dL)	60	101	-	-	78	-	-	-	-
Creatinine (mg/dL)	1.63 ↑	1.34 ↑	-	-	0.95	-	-	-	-

eGFR (mL/ min/1,73 m²)	37 ↓	47 ↓	-	-	71	-	-	-	-
Natrium (mmol/L)	132	-	136	136	135	-	-	-	-
Potassium (mmol/L)	4.0	-	3.7	3.9	4.0	-	-	-	-
Chloride (mmol/L)	95	-	98	96	91	-	-	-	-
Calcium (mmol/L)	2.16	-	2.0	2.03	2.16	-	-	-	-
Magnesium (mg/dL)	2.5	-	2.7	2.7	2.6	-	-	-	-
CRP (mg/L)	62 ↑	-	-	-	23 ↑	27 ↑	-	-	-
Urinalysis	-	Leuko-cytes +3, leukocyte esterase +2	-	-	-	-	-	Negative leukocyte, negative leukocyte esterase	-

AFP = alpha-fetoprotein; SGPT = alanine aminotransferase; SGOT = aspartate aminotransferase; hs-Troponin T = high-sensitivity troponin T; NT-proBNP = N-terminal pro–B-type natriuretic peptide; CRP = C-reactive protein; eGFR = estimated glomerular filtration rate.

At a 1-month outpatient follow-up, the 12-lead ECG demonstrates sinus rhythm at 84 bpm, right axis deviation, a PR interval of 150 ms, a QRS duration of 80 ms, poor R-wave progression, and low-voltage QRS complexes. No pathological Q waves, no ST-segment elevation or depression, and no T-wave inversion were observed. (Figure 2). These findings were consistent with the initial emergency room presentation, supporting recovery from acute myocarditis.

Cardiac Magnetic Resonance imaging (CMR) was performed during follow-up. This examination revealed myocardial edema and subepicardial Late Gadolinium Enhancement (LGE) in the lateral wall (Fig 4), two features consistent with acute myocarditis. The patient was discharged and was given heart failure Guideline-Directed Medical Therapy (GDMT) and tapering corticosteroids. At one-month follow-up, she remained asymptomatic with preserved ventricular function.

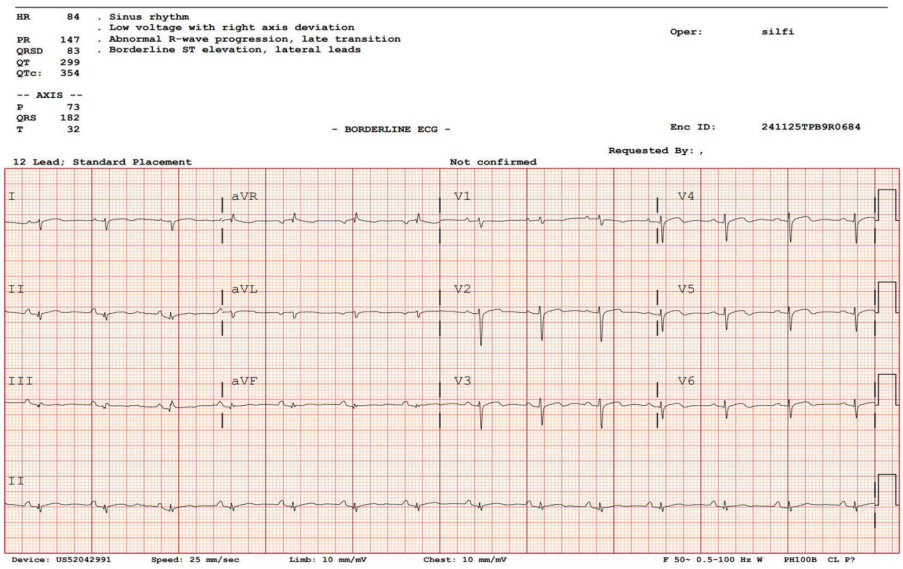

Figure 3. The follow-up ECG demonstrates sinus rhythm at 84 bpm with low QRS voltage, right axis deviation, delayed R-wave progression, and borderline ST-segment elevation in the lateral leads. Complete atrioventricular block has resolved.

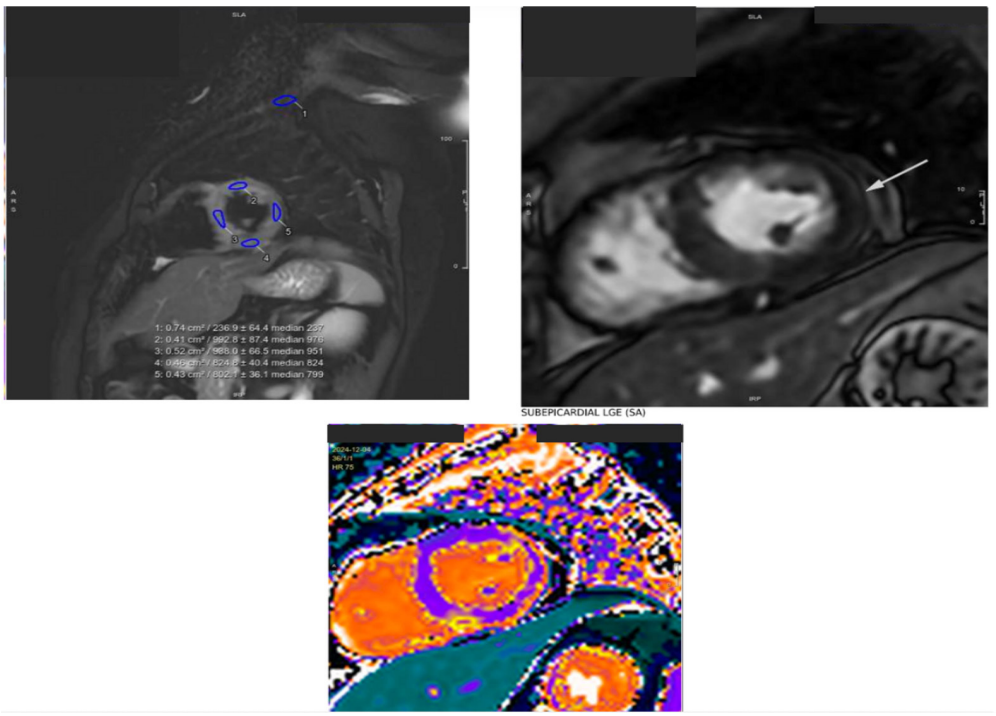

Figure 4. Cardiac magnetic resonance (CMR) images demonstrating myocardial inflammation. (Top left) T2-weighted imaging in all mid-segments of the left ventricle (LV) shows increased signal intensity consistent with myocardial edema (blue regions of interest). (Top right) Late gadolinium enhancement (LGE) was identified in the subepicardial region of the lateral wall (arrow), indicating myocardial injury and fibrosis. (Bottom) Native T1 mapping with elevated value in mid lateral segment, further supporting presence of myocardial edema. These findings fulfill the updated Lake Louise criteria and confirm the presence of acute myocarditis.

Discussion

The case provided addresses the diagnostic complexity of acute myocarditis presenting with simultaneous ST-segment elevation and CAVB. Such coexistence is particularly rare.[1-3] The complete AV block in this case is most likely due to direct inflammatory involvement of the AV node and the His (Purkinje) system. In myocarditis, immune-mediated cell injury and edema within the specialized conduction tissue can acutely interrupt impulse propagation from atria to ventricles, producing a high-grade or complete block that may improve as inflammation resolves, as observed in our patient.[4] While ST-segment elevation is frequently reported in myocarditis, which has been found to occur in up to 85% of cases, high-grade AV block remains distinctly uncommon and is generally associated with more fulminant phenotypes or specific etiologies such as giant cell myocarditis, sarcoidosis, or Lyme carditis.[1-3] The presence of both findings initially justified an emergent diagnosis of ACS, emphasizing the real-world challenge of differentiating myocarditis from STEMI.

In our patient, several features ultimately favored a diagnosis of acute myocarditis over true STEMI.[2] Clinically, the presentation was dominated by systemic prodromal symptoms (fever, diarrhea, malaise) and absence of typical ischemic chest pain, which is more consistent with viral or immune-mediated myocardial inflammation than with plaque rupture. On ECG, the initial tracing showed complete AV block with lateral ST-segment elevation and reciprocal changes, but follow-up ECG demonstrated restoration of sinus rhythm with low-voltage QRS complexes, poor R-wave progression, and only borderline lateral ST elevation without the development of pathological Q waves, suggesting transient inflammatory injury rather than fixed transmural necrosis. Coronary angiography revealed only discrete proximal LAD stenosis and no culprit obstructive lesion, while subsequent CMR demonstrated diffuse myocardial edema and subepicardial LGE in the lateral wall. This constellation of clinical, electrocardiographic, angiographic, and tissue-characterization findings supported myocarditis as the unifying diagnosis.[2-3]

In this patient, by performing an urgent coronary angiography, we managed to quickly exclude the presence of obstructive coronary artery disease, thereby redirecting the diagnostic approach toward non-ischemic causes. This stepwise process is consistent with guideline-based recommendations that mandate invasive evaluation when ACS cannot be confidently excluded.[2-3] The systemic prodromal features fever, diarrhea, and malaise added further clinical weight to the suspicion of viral or immune-mediated myocarditis.[5-7]

The subsequent use of CMR was decisive. CMR, incorporating T2-weighted imaging, native T1 mapping, and LGE, is now established as the primary non-invasive reference standard for diagnosing myocarditis.[1,5]

In this case, the combination of myocardial edema and subepicardial LGE fulfilled the updated Lake Louise criteria, hence, providing reliable confirmation. Cardiac magnetic resonance in this patient met the updated 2018 Lake Louise criteria for acute myocarditis, showing myocardial edema and non-ischemic subepicardial late gadolinium enhancement in the lateral wall. These findings confirmed the diagnosis and, given the limited extent of LGE together with the patient's functional recovery, suggest a relatively favorable prognosis. Beyond diagnostic accuracy, LGE and tissue mapping carry prognostic significance; extensive LGE (>20% of left ventricular mass) and increased extracellular volume have been linked to a higher risk of sudden cardiac death and adverse remodelling.[1] Our patient demonstrated localized subepicardial LGE with functional recovery, underscoring the heterogeneity of outcomes in myocarditis.

Therapeutically, the patient's favourable response to high-dose corticosteroids raises important considerations. In this case, the decision to initiate high-dose intravenous methylprednisolone was guided by the fulminant presentation with cardiogenic shock, severe LV dysfunction, and high-grade AV block.[8] All of these are strongly suggestive of an intense inflammatory process involving both the myocardium and the conduction system. Contemporary consensus documents and guidelines acknowledge that, although routine immunosuppression is not recommended for uncomplicated lymphocytic myocarditis, empirical intravenous corticosteroids may be considered in fulminant or complicated acute myocarditis to stabilize hemodynamics while awaiting or in the absence of definitive etiologic clarification.[8] High-dose glucocorticoids are intended to attenuate immune-mediated myocardial injury, reduce myocardial edema, and mitigate inflammatory infiltration, which may lead to faster recovery of ventricular function and resolution of high-grade block (as observed in our patient). Although the role of immunosuppression in myocarditis remains debated, emerging evidence suggests that in selected infection-negative or immune-mediated myocarditis, corticosteroids may expedite recovery and reduce arrhythmic risk.[3] The clinical improvement in this case, resolution of CRP as an inflammatory parameter with restoration of sinus rhythm and normalization of EF, aligns with such findings. This case highlights the unique coexistence of STEMI-like ST-segment elevation, complete AV block, and non-obstructive coronary arteries, a combination that strongly points toward acute myocarditis rather than true infarction and underscores the diagnostic challenge for clinicians.

Finally, this case reiterates how the integration of clinical suspicion, invasive and non-invasive imaging, and individualized therapy plays such an important role in managing patients. For clinicians, the key learning point is that myocarditis should always be one of the main differential diagnoses of patients with STEMI-like presentations complicated by conduction abnormalities, particularly when coronary angiography reveals non-obstructive disease. Early recognition and tailored management may significantly alter the trajectory of these patients, converting a potentially fatal presentation into a favorable recovery.

Conclusion

The provided case demonstrates how diagnosing myocarditis mimicking STEMI that is further complicated by complete AV block may be a challenge among physicians. A structured approach integrating coronary angiography and advanced CMR is essential to establish a reliable diagnosis. Early recognition and timely initiation of corticosteroid therapy were temporally associated with the patient's clinical and functional improvement. However, the favorable course was likely multifactorial and may also reflect supportive heart failure management and the self-limited nature of the underlying inflammatory process. Clinicians should remain vigilant for myocarditis in ACS-like presentations with conduction abnormalities, as prompt differentiation has major therapeutic and prognostic implications.

Learning Points

- Acute myocarditis can closely mimic STEMI when presenting with ST-segment elevation and markedly elevated troponin, particularly when accompanied by complete atrioventricular block.
- The coexistence of STEMI-like ECG changes, complete AV block, and non-obstructive coronary arteries should prompt consideration of myocarditis and trigger advanced tissue-characterization imaging with CMR.
- CMR fulfilling the updated 2018 Lake Louise criteria (myocardial edema and non-ischaemic subepicardial LGE) is pivotal for confirming myocarditis and provides prognostic information based on the extent and distribution of LGE.
- In conclusion, biopsy-unproven myocarditis with cardiogenic shock and complete AV block, high-dose corticosteroid therapy may be considered on an individualized basis, recognizing that improvement is likely multifactorial and that routine immunosuppression is not recommended for all myocarditis phenotypes.

List of Abbreviations

ACS	Acute Coronary Syndrome
AFP	Alpha-fetoprotein
AV	Atrioventricular
bpm	Beats per minute
CAVB	Complete Atrioventricular Block
CMR	Cardiac Magnetic Resonance
CRP	C-reactive protein
ECG	Electrocardiogram
EF	Ejection Fraction
eGFR	Estimated glomerular filtration rate
GDMT	Guideline-Directed Medical Therapy
hs-Troponin T	High-sensitivity troponin T
LAD	Left Anterior Descending
LGE	Late Gadolinium Enhancement
LM	Left Main
LCX	Left Circumflex
LV	Left Ventricle
NT-proBNP	N-terminal pro–B-type natriuretic peptide
RCA	Right Coronary Artery
SCAI	Society for Cardiovascular Angiography and Interventions
SGOT	Aspartate aminotransferase
SGPT	Alanine aminotransferase
STEMI	ST-Elevation Myocardial Infarction

Ethical Clearance

Not applicable.

Publication Approval

All authors are consent to the publication of this manuscript. Written informed consent was obtained from the patient.

Authors Contributions

RM and HA collected clinical data, prepared the figures, and drafted the manuscript. RS, CA, DYH, BW supervised clinical management, provided critical revisions, and contributed to the final interpretation. All authors read and approved the final version of the manuscript.

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Conflict of Interest

None.

Availability of Data and Materials

Available upon request.

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Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

The authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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