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Society of Cardiovascular Angiography and intervention Stage-B cardiogenic shock: An interventional-heart failure-critical care conundrum

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Abstract

The Society of Cardiovascular Angiography and Intervention (SCAI) classified cardiogenic shock (CS) into five stages ranging from A-E. There remains significant ambiguity regarding the assessment and management of SCAI Stage B. Given its nebulous nature that can rapidly escalate, prompt interventions are needed. Here, we describe the trajectory of cases that presented with SCAI Stage B CS.

Keywords

Cardiogenic shock; SCAI stage B; Mixed shock; Mechanical circulatory support

1. Introduction

Cardiogenic shock (CS) presents with varying hemodynamic profiles encompassing hypoperfusion without hypotension to extreme hemodynamic collapse. The recent Society

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Study design, literature review, and statistical analysis: AM, MB, PMB, ONG, DXZ, SV.
Data management, data analysis, drafting manuscript: AM, MB, PMB, ONG, DXZ, SV.
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Manuscript revision, intellectual revisions, mentorship: PMB, ONG, DXZ, SV.
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Declaration of competing interest

None.

Appendix A. Supplementary data

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of Cardiovascular Angiography and Intervention (SCAI) classification classifies CS from stages A-E. Early stages of CS typically involve stable macrovascular hemodynamics and there is a clear hemodynamic perturbation in SCAI Stages C-E, however prior data have demonstrated that there is delayed recognition in earlier SCAI stages.[1,2] SCAI stage B CS constitutes a tenuous clinical entity that can rapidly evolve into higher stages.[3] Oftentimes, CS may be superimposed with septic and rarely hypovolemic shock which compounds the clinical assessment making therapeutic interventions challenging.[4] The combination of cardiac arrest and CS is also associated with worse outcomes.[5] However in cases with SCAI stage B, there is ambiguity regarding optimal management which can affect the clinical course of the patient.[6] In this case series of SCAI stage B CS, we seek to describe the varying trajectories highlighting the clinical challenges in decision-making (Supplementary Table 1).

2. Case 1

A 79-year-old female with hyperlipidemia, hypertension, and atrial fibrillation presented with chest discomfort. Physical examination revealed stable hemodynamics with tachycardia (141 bpm) and electrocardiogram (ECG) showed anterior ST-segment-elevation myocardial infarction (STEMI) (Fig. 1). Emergent coronary angiography demonstrated a mid-left anterior descending artery (LAD) occlusion for which percutaneous coronary intervention (PCI) was performed. Her left ventriculogram demonstrated anteroapical regional wall motion abnormality with an estimated left ventricular ejection fraction (LVEF) of 35–40%. Post-procedure, the patient was persistently hypotensive with lactic acidosis (7.1 mmol/L). and severe functional mitral regurgitation without evidence of ischemic leaflet tethering. A right heart catheterization demonstrated a depressed cardiac output (1.5 L/min) secondary to mitral regurgitation. An intra-aortic balloon pump (IABP) was placed for afterload reduction and she was treated with intravenous diuretics, inotropes, and rate control for atrial fibrillation. However, she continued to have low urine output and therefore was escalated to a percutaneous left ventricular assist device (pLVAD) support. Given her improving hemodynamics, the Impella was gradually weaned and decannulated. She continued to do well for a few days but due to repeated episodes of atrial fibrillation with rapid ventricular response, she developed renal and mental status instability. Due to her advanced age and multiorgan involvement, the patient's family opted against additional aggressive cares, and she was subsequently transitioned to hospice care.

3. Case 2

A 55-year-old female presented with chest pain and stable hemodynamics with a normal ECG. While awaiting care in the emergency room, she developed a cardiac arrest due to ventricular fibrillation require chest compressions, single dose of epinephrine, and cardioversion. Repeat ECG after return of spontaneous circulation showed an anterior STEMI with stable vital signs (Fig. 2). Emergent coronary angiography demonstrated 100% proximal LAD occlusion, left ventricular end-diastolic pressure (LVEDP) of 42 mmHg and estimated LVEF of 35–40%. During her angiogram, she developed two additional episodes of ventricular tachycardia/fibrillation necessitating defibrillation. At this time, due to her hemodynamic instability, she received vasopressors (norepinephrine and

epinephrine) and mechanical circulatory support (MCS) with an Impella CP. After PCI, her right heart catheterization demonstrated significant improvement in her filling pressures. Post-procedure, her hemodynamics showed a decrease in biventricular filling pressures concerning for superimposed distributive shock. She developed leukocytosis, suspected to be secondary to a respiratory infection, and was treated appropriately with broad-spectrum antibiotics. Later, MCS was weaned and removed. Her echocardiogram post-discharge demonstrated normalization of LVEF (60%) and she was discharged home safely.

4. Case 3

A 76-year-old male with hypertension, hyperlipidemia, and tobacco abuse, presented with substernal chest discomfort and stable hemodynamics. ECG demonstrated anterolateral STEMI (Fig. 3). His initial laboratory parameters showed leukocytosis, elevated troponin and mildly elevated lactate (1.44 mmol/L). Emergent coronary angiography demonstrated 100% proximal LAD artery occlusion with heavy thrombotic burden. The left ventriculogram demonstrated an LVEF of 30–35% with severe apical and anterolateral hypokinesis with an elevated LVEDP of 41 mmHg. pLVAD was placed and the distal left main and proximal LAD were treated using aspiration thrombectomy, high-pressure noncompliant balloon inflation, and dedicated two-stent bifurcation technique. The Impella was left in situ to assist with the post-procedure hemodynamic management.

In the intensive care unit, he developed persistent hypotension with increasing doses of norepinephrine and epinephrine support. He developed worsening multiorgan failure, severe lactic acidosis, acute kidney injury and acute liver failure. He was diagnosed with concomitant sepsis secondary to community-acquired pneumonia. Due to likely disseminated intravascular coagulopathy, he developed oozing from multiple intra-arterial intravenous sites including his pLVAD access site. Despite treatment with inotropes, vasopressors, fluid boluses, invasive mechanical ventilation and renal replacement therapy, he developed ventricular tachycardia and ventricular fibrillation arrest. Due to his poor prognosis and multiorgan failure, he was made comfort care by his family and he passed subsequently.

5. Case 4

A 39-year-old patient presented with sub-sternal chest discomfort of 3 hour duration and stable hemodynamics, and ECG showed an anterior STEMI with Q-waves (Fig. 4). Emergent coronary angiography was considered due to ongoing symptoms of <12 hours, and this demonstrated mid LAD artery occlusion, chronic total occlusion of left circumflex artery and severe right coronary artery disease. A left ventriculogram demonstrated a LVEF of 15% with severe global hypokinesis and LVEDP of 32 mmHg. He underwent culprit lesion PCI, but due to slow flow at the end of the case, an IABP was placed. Echocardiography revealed an LVEF of 15%, presence of an apical mural thrombus, and mild-moderate mitral regurgitation. He had gradual improvement in symptoms during day 2–3, however, he developed new onset chest pain on day 3 with new antero-lateral STEMI on ECG. Repeat coronary angiography demonstrated subacute stent thrombosis of the proximal LAD stent requiring aspiration thrombectomy and balloon angioplasty. Due to LV

mural thrombus, he was transitioned to a TandemHeart device for greater circulatory support as a bridge to decision. During his intensive care unit stay, he developed multiple episodes of ventricular tachycardia that were cardioverted. Due to inability to wean off the pLVAD, he underwent durable LVAD placement as an inpatient. However, he continued to have severe right ventricular failure (unmasked after LVAD implantation), multiple ventricular arrhythmias, and subsequently developed worsening multiorgan failure, coagulopathy and ischemic strokes. He subsequently passed away 28 days after admission.

6. Discussion

Despite significant advances in the pharmacologic and device-based therapies for Cardiogenic Shock (CS), the mortality rate remains significantly high at ≥ 50%.[7] The Society for Cardiovascular Angiography and Interventions (SCAI) developed and released the shock stages classification in 2019 which intended to standardize communication about the diagnosis, risk stratification, presentation, and severity of CS across various research protocols and individual centers.[1] It is a five-stage system (stages A-E) that reflects gradations of shock severity and can be utilized for guiding treatment and classifying outcomes.[1]

CS follows a spectrum of SCAI stages and has significant variability in presentation. There remains significant heterogeneity in patients presenting with SCAI Stage B CS which may be independent of the shock severity.[8,9] CS is frequently seen in the cardiac intensive care unit. It often presents in the setting of either primary or secondary septic shock and can display mixed behavior.[7] Detailed understanding of hemodynamics at frequent time intervals and use of pulmonary artery catheterization to assist with decision making for pharmacological or MCS is of paramount importance in such patients with mixed shock.[10] Also, though left ventricular failure from acute myocardial infarction still constitutes the chief etiology in most cases, alternate etiologies such as functional or ischemic mitral regurgitation, ventricular septal defect and papillary muscle rupture should be entertained in patients who develop CS in a delayed manner.[11] Though the IABP is very effective in patients with valvular complications, these patients might often require escalation to higher MCS devices to achieve optimal diuresis and pulmonary decongestion goals.[12] Also, despite accumulating evidence on the benefit of MCS devices in these patients, we still need to determine the most suitable device features, the timing of support, and the most relevant patient population to maximize the clinical benefits. In addition, randomized controlled trials to establish the use of MCS devices to improve outcomes are still pending.

Although AMI remains the commonest inciting pathology leading to CS, there has been a steadily rising trend of other etiologies causing CS.[13,14] In recent times, acute decompensated heart failure is frequently associated with cardiogenic shock, whereas arrhythmias and valvular heart disease are noted less frequently.[14–16] Careful evaluation of hemodynamics, prompt optimization of hemodynamic insult, prevention of hemo-metabolic cascade of multiorgan failure and rapid reversal of inciting etiology (such as primary PCI for acute myocardial infarction) are important tenets in the management of such patients.[17,18] Patients at each SCAI shock stage behave differently and may have a different trajectory of their disease process. Acknowledging the dynamic progression and varying trajectories of

different SCAI Shock stages is crucial for recognizing patients who are deteriorating despite therapy and need escalation of therapy or transfer to a higher level of care.[19]

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

CS	cardiogenic shock
ECG	electrocardiogram
IABP	intra-aortic balloon pump
LAD	left anterior descending artery
LVEDP	left ventricular end-diastolic pressure
LVEF	left ventricular ejection fraction
MCS	mechanical circulatory support
PCI	percutaneous coronary intervention
pLVAD	percutaneous left ventricular assist device
SCAI	society of cardiovascular angiography and intervention
STEMI	ST-segment-elevation myocardial infarction

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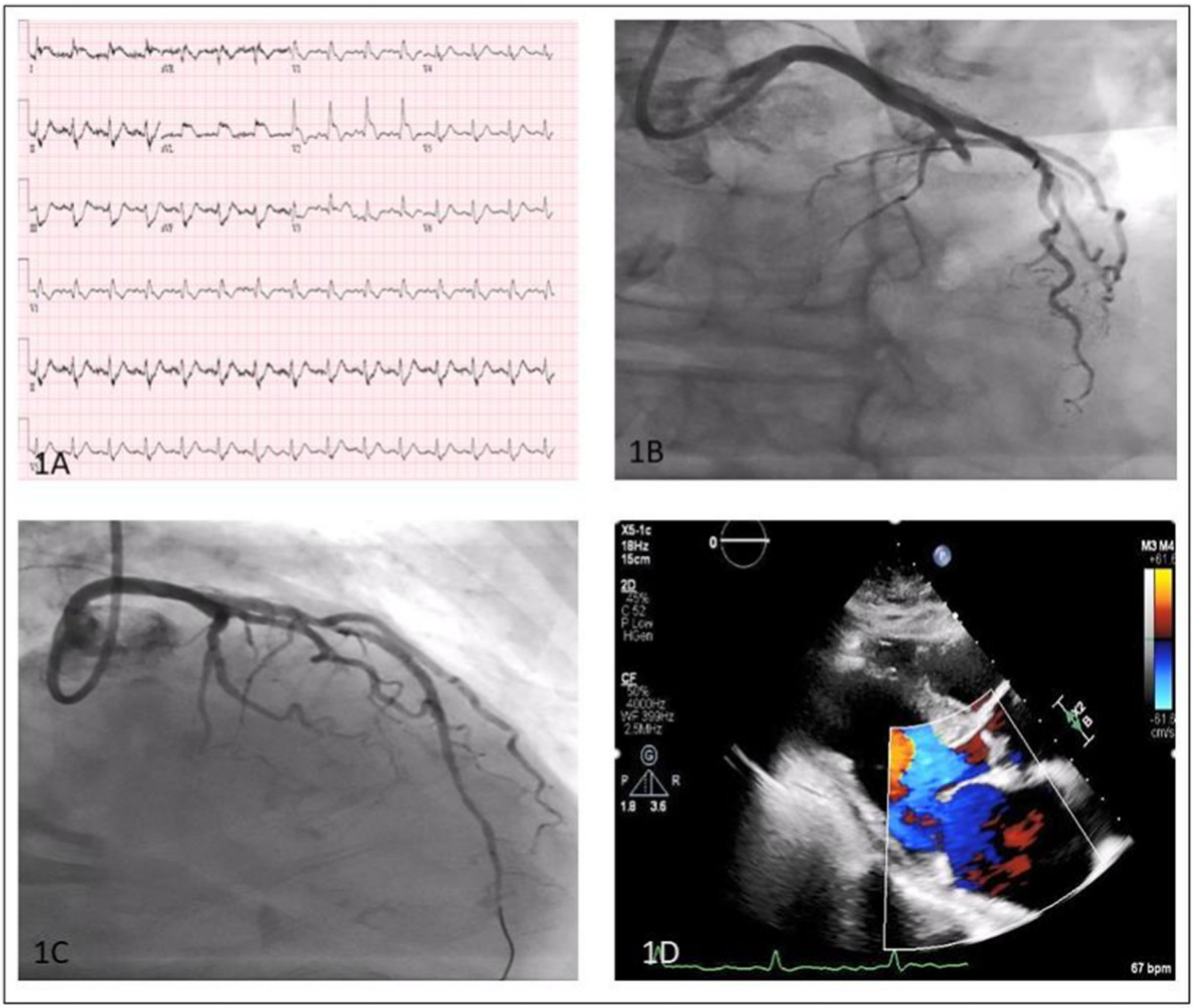

Fig. 1.

Clinical Case 1

Electrocardiogram (A), coronary angiogram (pre [B] and post[C] percutaneous coronary intervention), and echocardiogram (D) demonstrating anterior ST-segment elevation myocardial infarction with evidence of mid-left anterior descending artery thrombotic occlusion and severe mitral regurgitation without ischemic leaflet tethering.

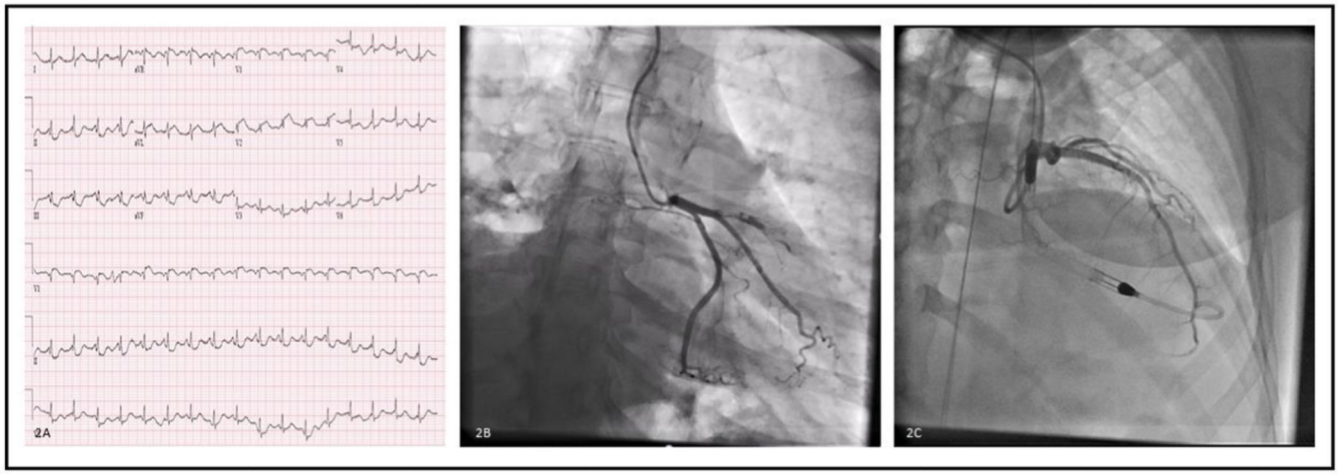

Fig. 2.

Clinical Case 2

Electrocardiogram (A), echocardiogram (B) coronary angiogram (pre [C] and post[D] percutaneous coronary intervention along with the presence of left ventricular assist device [Impella]), and echocardiogram (E) demonstrating anterior ST-segment elevation myocardial infarction with evidence of proximal left anterior descending artery thrombotic occlusion and normal left ventricular systolic function.

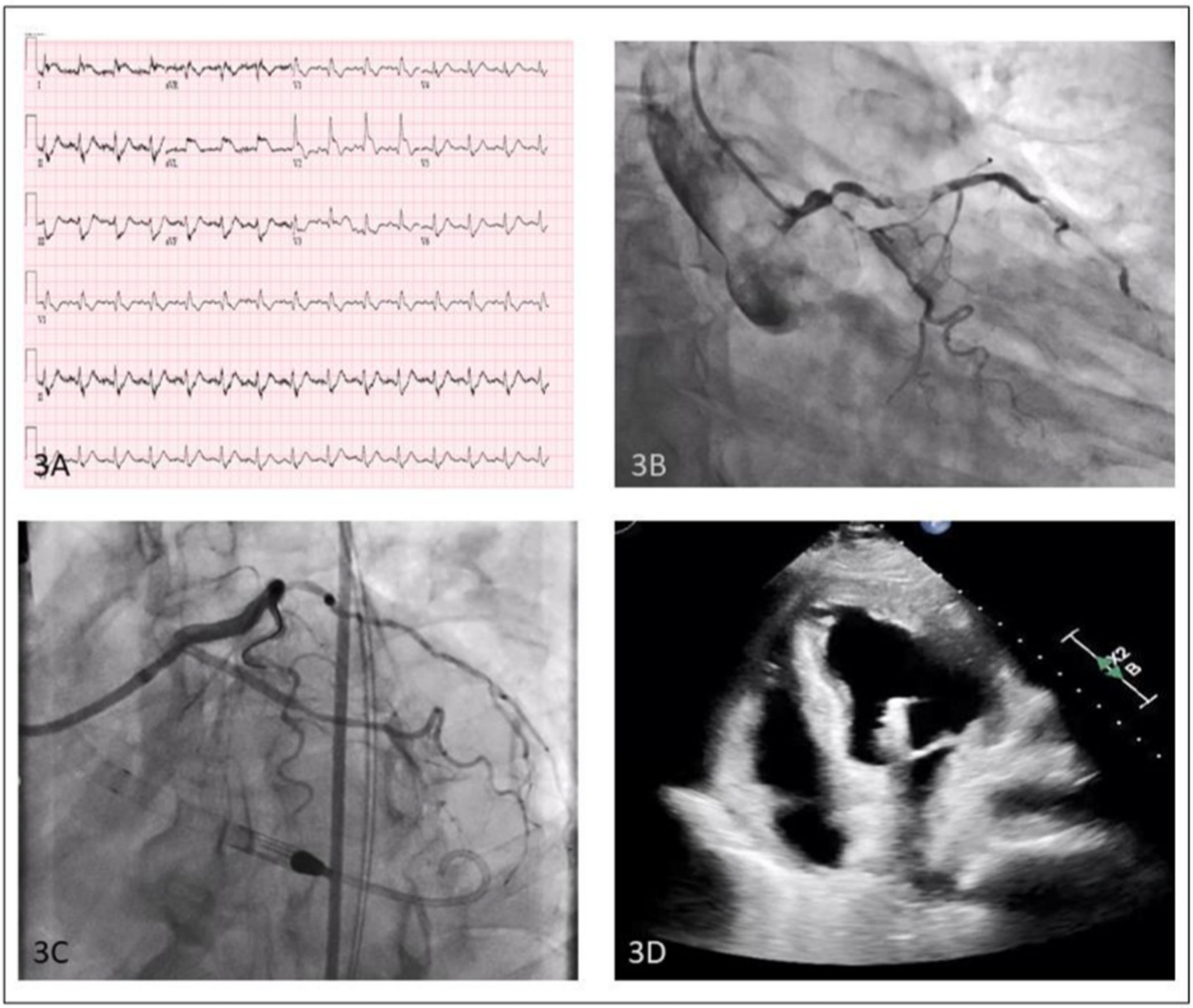

Fig. 3.

Clinical Case 3

Electrocardiogram (A), coronary angiogram (pre [B] and post[C] percutaneous coronary intervention along with the presence of left ventricular assist device [Impella]), and echocardiogram (D) demonstrating anterior ST-segment elevation myocardial infarction with evidence of proximal left anterior descending artery thrombotic occlusion and decreased left ventricular systolic function.

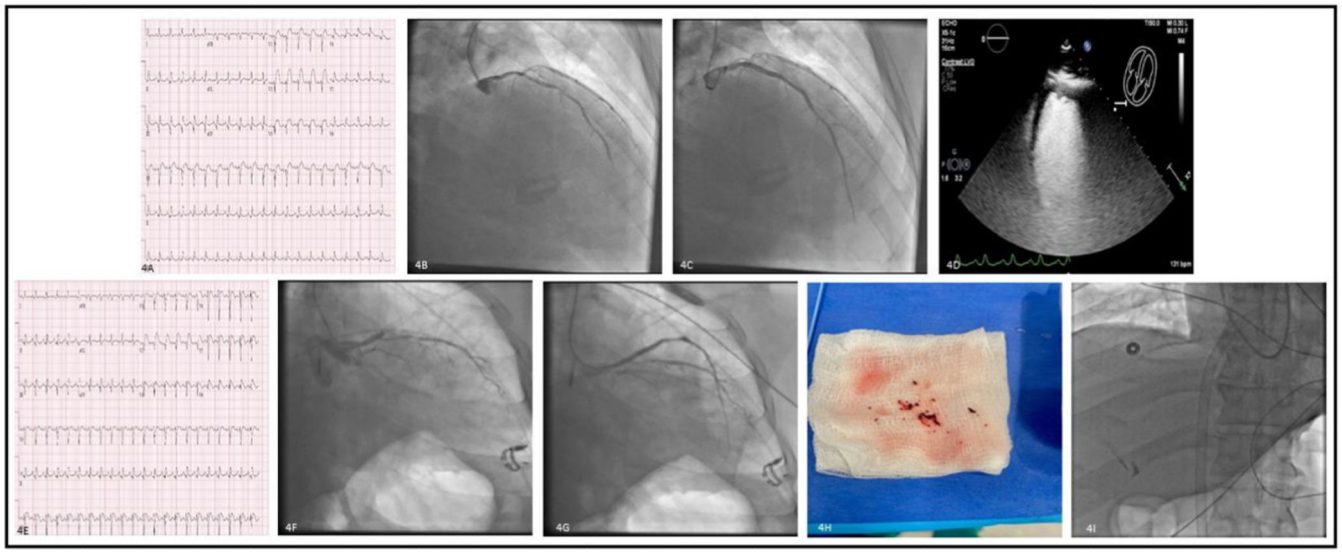

Fig. 4.

Clinical Case 4

Electrocardiogram (A), coronary angiogram (pre [B] and post[C] percutaneous coronary intervention), and echocardiogram (D) demonstrating anterior ST-segment elevation myocardial infarction with evidence of proximal left anterior descending artery thrombotic occlusion, decreased left ventricular systolic function, and apical mural thrombosis. Repeat electrocardiogram (E) on day 3 demonstrating antero-lateral ST-segment-elevation. Repeat coronary angiography demonstrating sub-acute stent thrombosis of proximal left anterior descending artery stent (F) needing repeat balloon angioplasty (G) and aspiration thrombectomy demonstrating coronary thrombus (H). Subsequent stabilization of cardiogenic shock using TandemHeart ® percutaneous left ventricular assist device (I).