American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e953894
DOI: 10.12659/AJCR.953894

Received: 2026.04.25
Accepted: 2026.08.04
Available online: 2026.08.13
Published: 2026.XX.XX

Total Left Main Occlusion ST-Elevation Myocardial Infarction Complicated by Cardiogenic Shock and Delayed Sinus Node Dysfunction

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Financial support: None declared
Conflict of interest: None declared

Patient: Male, 58-year-old
Final Diagnosis: Cardiogenic shock • sinus node dysfunction • STEMI
Symptoms: Chest pain • syncope
Clinical Procedure: Pacemaker implantation • percutaneous coronary intervention
Specialty: Cardiology

Objective: Rare disease

Background: Acute ST-segment elevation myocardial infarction (STEMI) due to total occlusion of an unprotected left main coronary artery (LMCA) is rare and often fatal, particularly when complicated by cardiogenic shock.

Case Report: We report the case of a 58-year-old man who presented with sudden, severe chest pain due to an extensive anterolateral STEMI. His condition rapidly deteriorated, with the development of severe hypoxemia, flash pulmonary edema, and cardiogenic shock. Endotracheal intubation was performed, followed by urgent primary percutaneous coronary intervention and initiation of intra-aortic balloon pump support. Coronary angiography revealed a 100% distal LMCA occlusion, which was successfully treated with balloon pre-dilation, aspiration thrombectomy, and drug-eluting stent implantation, restoring TIMI III coronary flow. Despite successful reperfusion, he required escalation to triple vasoactive therapy with norepinephrine, epinephrine, and dobutamine before gradual hemodynamic and respiratory recovery. A notable finding was an anomalous origin of a small circumflex artery arising from the right coronary artery. His course was complicated by delayed sinus node dysfunction, manifested by a brief daytime syncopal episode with a telemetry-documented 7-second sinus pause, necessitating implantation of a dual-chamber pacemaker. He was initiated on guideline-directed medical therapy for heart failure, with improvement in left ventricular ejection fraction from less than 30% at presentation to 40% to 45% prior to discharge.

Conclusions: This case provides incremental insight into survival after catastrophic total unprotected LMCA occlusion with profound cardiogenic shock, highlighting the role of rapid revascularization and aggressive hemodynamic support, and emphasizing vigilance for delayed post-infarction conduction complications.

Keywords: Coronary Occlusion • Pacemaker, Artificial • Shock, Cardiogenic

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953894

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Introduction

Acute left main coronary artery (LMCA) culprit ST-segment elevation myocardial infarction (STEMI) is uncommon, but is often catastrophic because a very large myocardial territory is abruptly jeopardized. Contemporary data suggest that LMCA culprit STEMI accounts for approximately 0.8% to 5% of STEMI presentations undergoing angiography, with variability related to cohort selection and definitions (subtotal versus total occlusion) [1]. True total or near-total unprotected LMCA occlusion is an even rarer and higher-risk subset, comprising well under 1% of primary percutaneous coronary intervention (PCI) cases in a large national database [2]. Despite its rarity, this presentation carries disproportionate clinical severity due to the extensive myocardial territory at risk.

Among patients who reach a hospital, cardiogenic shock is very common in LMCA culprit STEMI and is a major determinant of early mortality. Registry data consistently demonstrate that roughly 40% to 50% of patients with LMCA STEMI present with or develop cardiogenic shock [3]. In-hospital mortality remains high, often exceeding 25% overall and surpassing 40% in those complicated by cardiogenic shock [3]. Similarly, in the British Cardiovascular Intervention Society experience, focused on unprotected left main stem occlusion, periprocedural shock occurred in about 58%, and outcomes were poor, with in-hospital mortality about 43% [2]. Registry analyses of unprotected left main coronary artery percutaneous coronary intervention (LMCA PCI) show particularly poor survival in STEMI complicated by cardiogenic shock, with preprocedural TIMI 0 flow strongly associated with excess mortality [4].

Despite the severe early hazard, survival is possible, especially with rapid reperfusion, contemporary primary PCI strategies, aggressive hemodynamic support, and advanced critical care management. Longer-term outcomes reveal a sharp contrast between patients with and without cardiogenic shock, underscoring shock as the key prognostic inflection point [2]. Although early mortality remains substantial [2,5], selected patients who survive the acute phase can achieve meaningful recovery [6-8], highlighting the clinical relevance of reporting contemporary cases of total LMCA occlusion complicated by cardiogenic shock with favorable outcomes.

In this case report, we describe a 58-year-old man who survived a STEMI caused by total occlusion of the LMCA, complicated by profound cardiogenic shock requiring escalation to triple vasoactive medications and intra-aortic balloon pump (IABP).

Case Report

A 58-year-old male, heavy smoker, with a medical history significant for peripheral artery disease (occluded left common iliac artery), hypertension, and dyslipidemia, presented to the emergency department at the American University of Beirut Medical Center (AUBMC) with sudden-onset severe chest pain radiating to the back, which began approximately 30 minutes prior to hospital arrival. The patient had an episode of syncope lasting a few seconds, as reported by his wife, prior to transport to the hospital. Electrocardiography (ECG) showed ST-segment elevation in the anterior, anterolateral, and lateral leads (V2–V6, I, and aVL) (**Figure 1**). Upon presentation, his blood pressure was 110/62 mm Hg. Aspirin (300 mg) and ticagrelor (180 mg) were administered as loading doses. Intravenous unfractionated heparin (4000 units) was administered.

Upon arrival to the cardiac catheterization laboratory, the patient was agitated and in severe respiratory distress, reporting dyspnea and orthopnea. His oxygen saturation (SpO_2) was 78%; he was hypotensive (blood pressure 81/58 mm Hg) with weak peripheral pulses consistent with cardiogenic shock. During rapid sequence induction of general anesthesia, frothy pulmonary secretions were noted. Prior to intubation, he had an episode of vomiting. Oxygen saturation remained critically low despite mechanical ventilation and endotracheal suction. Salbutamol, hydrocortisone, and intravenous furosemide were administered, with minimal improvement in oxygenation. Norepinephrine infusion was initiated.

Emergency coronary angiography was performed via a right femoral access. Radial access was not attempted because the radial pulses were non-palpable; therefore, right femoral access was selected given the occluded left common iliac artery and need for rapid feasible access. Diagnostic angiography demonstrated a totally occluded distal LMCA (**Figure 2A**). The left system was engaged with a 6F guiding catheter. A guidewire was advanced into the distal left anterior descending artery (LAD), and another guidewire was positioned in the large first diagonal branch for protection. The lesion was predilated using a 2.5 × 20 mm balloon inflated up to 16 atm. This resulted in partial restoration of blood flow with visible filling defects (**Figure 2B**). The door-to-balloon time was approximately 60 minutes. Manual thrombectomy was subsequently performed using an aspiration catheter. A 4.0 × 15 mm drug-eluting stent was then deployed across the distal left main lesion with inflation up to 14 atm. Post-dilation was performed using a 4.5 × 12 mm balloon with inflation up to 18 atm, achieving good stent expansion and restoration of TIMI III coronary flow. The right coronary artery (RCA) was subsequently evaluated and was found to be large and dominant, with no significant obstructive disease. A small anomalous circumflex artery was noted arising from the RCA (**Figure 3**). Despite reperfusion and norepinephrine infusion, the patient remained hypotensive. Therefore, an IABP was inserted for hemodynamic support. Intravascular ultrasound was not performed after stenting because the patient remained unstable and further

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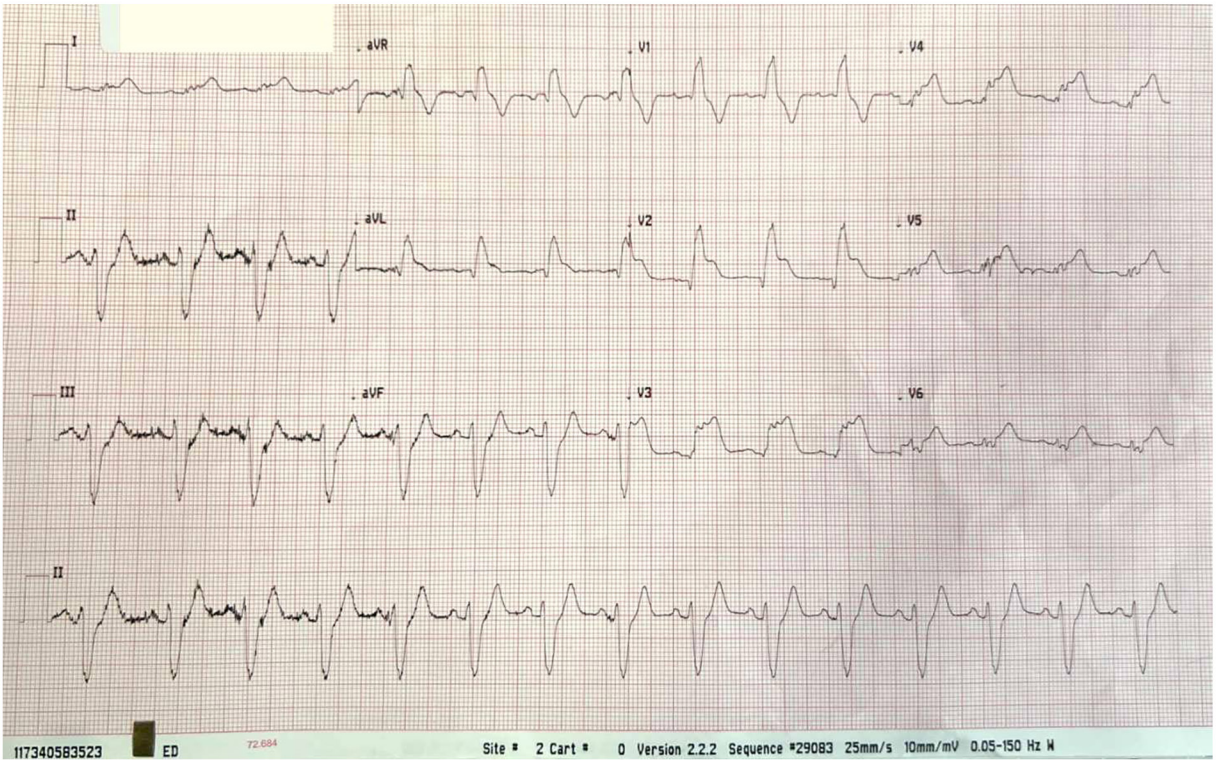

Figure 1. Electrocardiogram showing ST-segment elevation in leads V2–V6, I, and aVL.

imaging would have delayed urgent IABP insertion through the same femoral access.

In the cardiac care unit, despite escalating vasopressor support with norepinephrine up to 20 mcg/min, he remained hypotensive; therefore, dobutamine infusion was initiated, followed by the addition of an epinephrine infusion. Within a few hours, the doses of vasoactive agents were escalated to norepinephrine 20 mcg/min, epinephrine 0.12 mcg/kg/min, and dobutamine 7.5 mcg/kg/min. During vasopressor escalation, blood cultures were obtained, and empiric broad-spectrum antibiotics and hydrocortisone 100 mg every 8 hours were initiated to cover for a possible septic component related to aspiration pneumonia, likely resulting from the vomiting episode prior to intubation. Notably, the highest recorded temperature was 38.2 °C. Blood cultures subsequently returned negative. The antibiotic course was completed during the hospital stay.

Over the next few hours after catheterization, oxygen saturation gradually improved. Epinephrine was tapered and discontinued over 2 days, followed by tapering and discontinuation of norepinephrine. Hydrocortisone was also tapered and discontinued, and the IABP was removed. Subsequently, dobutamine was gradually tapered and discontinued. During the first week of admission, the patient received intravenous furosemide, then was transitioned to oral furosemide. Notably, 2 days after the intubation, he developed an episode of bloody endotracheal secretions while on mechanical ventilation. Inhaled tranexamic acid was administered, resulting in improvement of the bloody secretions. Six days after the endotracheal intubation, he was successfully extubated.

Subsequently, he was started on guideline-directed heart failure medical therapy (bisoprolol 1.25 mg twice daily, eplerenone 50 mg daily, dapagliflozin 10 mg daily, and valsartan 40 mg daily) and continued on oral furosemide.

Ten days after presentation and 3 days after the initiation of low-dose bisoprolol, he had a brief syncopal episode while awake during the daytime. Continuous telemetry documented a 7-second sinus pause (**Video 1** showing asystole on the telemetry). Earlier ECGs demonstrated sinus rhythm with right bundle branch block and left posterior fascicular block. Although a contribution from bisoprolol could not be excluded, the dose was low and beta-blockade was considered an essential component of his post-infarction heart failure therapy. No other concomitant medication known to contribute or other apparent reversible precipitant was identified, and the recent extensive myocardial infarction was considered the most plausible underlying cause of the delayed sinus node dysfunction.

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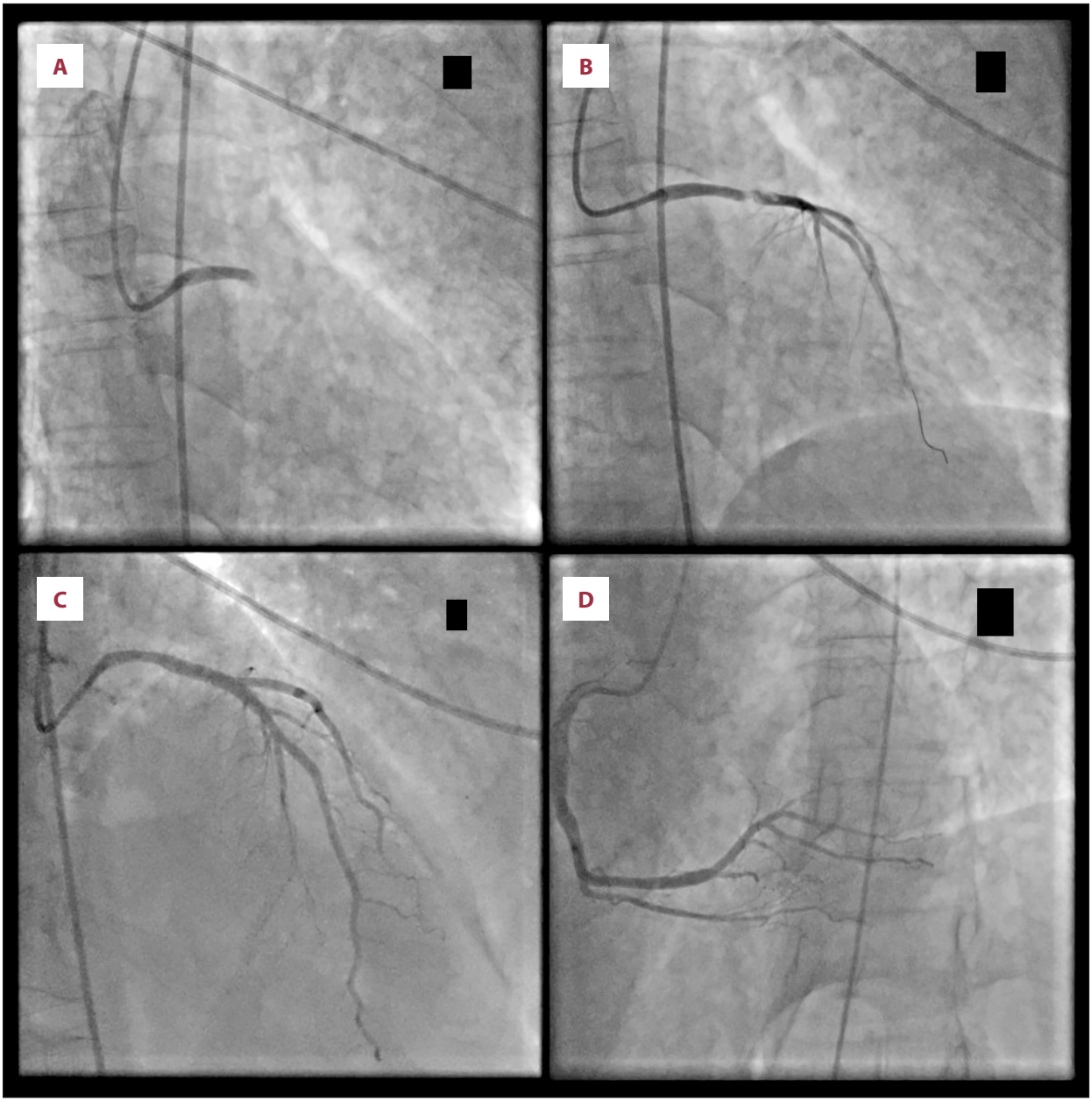

Figure 2. Coronary angiogram showing the percutaneous coronary intervention. (**A**) Initial coronary angiogram demonstrating complete thrombotic occlusion of the distal left main coronary artery. (**B**) Restoration of partial coronary flow following balloon dilatation with visible filling defects. (**C**) Deployment of a drug-eluting stent across the distal left main lesion with restoration of TIMI III coronary flow. (**D**) Post-procedural right coronary angiography demonstrating a large-caliber right coronary artery without significant luminal stenosis.

Given the prolonged pause with syncope, its occurrence while awake, and the anticipated need to continue beta-blocker therapy, observation alone was considered unsafe. Temporary pacing was not favored because it would provide only short-term protection while concern for recurrence and the need for beta-blocker therapy remained. Therefore, a permanent dual-chamber pacemaker was implanted (**Figure 4**). **Figure 5** shows the ECG after pacemaker insertion.

Prior to discharge, the patient was ambulatory and able to move independently. A repeat echocardiogram showed mildly impaired left ventricular systolic function with an estimated ejection fraction of 40% to 45% (it was < 30% on day of presentation). Regional wall motion abnormalities included hypokinesis of the basal anterior, mid-anterior, and apical lateral segments, and akinesis of the mid-anteroseptal, apical anterior, and apical segments. All remaining segments demonstrated normal wall motion.

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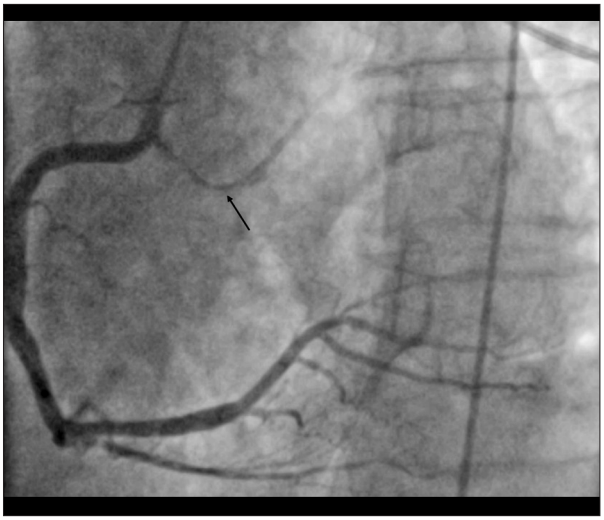
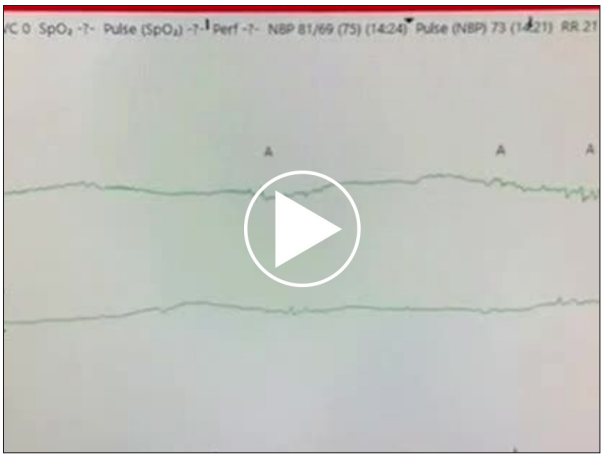


Figure 3. Right coronary angiography showing a small, anomalous circumflex artery arising from the right coronary artery.



Video 1. Continuous telemetry recording demonstrating a 7-second asystolic pause associated with the brief syncopal episode.

The patient tolerated heart failure medical therapy well, although his systolic blood pressure remained in the 90s. After a total hospitalization duration of 14 days, he was discharged with a plan to initiate sacubitril/valsartan during follow-up, contingent on blood pressure tolerance.

At follow-up, the patient was clinically well and able to perform his daily activities without limitation. No further syncopal episodes were reported. He regularly monitored his blood pressure at home, with systolic readings generally in the 90s and occasionally in the 80s. Pacemaker interrogation performed 10 days after discharge demonstrated an atrial-sensed, ventricular-sensed (AS–VS) rhythm 99% of the time and an atrial-paced, ventricular-sensed (AP–VS) rhythm 1% of the time. Echocardiography performed 2 months after discharge showed

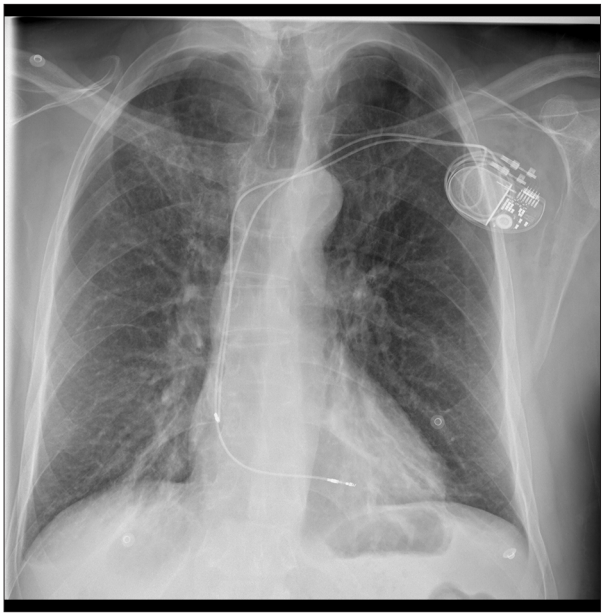


Figure 4. Chest X-ray following dual-chamber pacemaker insertion.

stable left ventricular systolic function, with an ejection fraction of 40% to 45%.

Discussion

STEMI caused by total LMCA occlusion is an uncommon but frequently catastrophic presentation [1]. Because the LMCA supplies a large proportion of the left ventricular myocardium, acute occlusion often results in extensive ischemia, with patients frequently presenting with acute pulmonary edema or severe cardiogenic shock [1]. Consequently, the prognosis remains poor, with high reported in-hospital mortality rates. In line with these findings, the ASTER registry demonstrated that unprotected LMCA STEMI carries a particularly poor prognosis, with high in-hospital mortality despite emergency PCI, especially in elderly patients, diabetics, and cases where optimal TIMI III reperfusion is not achieved [5].

This case is best considered incrementally informative, highlighting a rare integrated trajectory of survival after total distal LMCA occlusion with profound cardiogenic shock managed using triple vasoactive therapy and IABP, followed by delayed sinus node dysfunction requiring permanent pacing. It emphasizes that late conduction complications can emerge even after successful reperfusion and hemodynamic recovery.

Electrocardiographic Findings of LMCA Disease

Early ECG recognition is essential for prompt triage and reperfusion. Severe but subtotal occlusion of LMCA is classically

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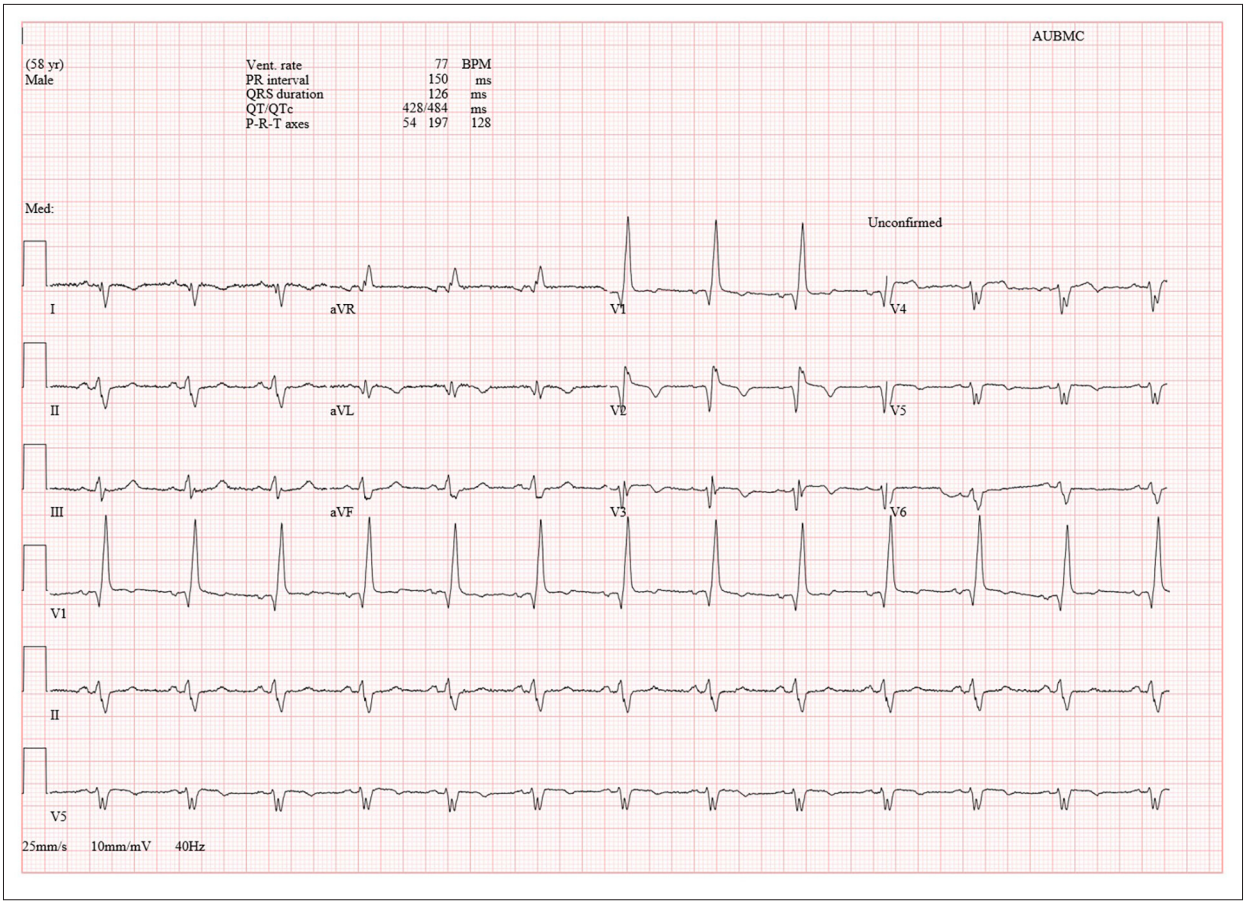

Figure 5. Electrocardiogram after pacemaker insertion.

associated with diffuse ST-segment depression in multiple leads accompanied by ST elevation in lead aVR, reflecting subendocardial ischemia [9-11]. In contrast, acute total LMCA occlusion can present with extensive ST-segment elevation in the anterior leads (V2–V6), with or without involvement of V1, as well as the high lateral leads (I, aVL), often accompanied by reciprocal inferior ST depression and bi-fascicular conduction abnormalities, typically right bundle branch block with left anterior fascicular block [11,12], while left posterior fascicular block is a rare manifestation in STEMI [13].

Management of Myocardial Infarction–Related Cardiogenic Shock

In patients presenting with STEMI complicated by cardiogenic shock, current guideline-directed management emphasizes immediate culprit vessel reperfusion therapy, typically with primary PCI [14]. Management of cardiogenic shock often requires aggressive pharmacologic support with vasoactive agents as well as mechanical circulatory support [15]. Vasoactive therapy is required in most patients to maintain systemic perfusion [16]. Norepinephrine is generally recommended as the first-line vasopressor because it provides effective blood pressure support with a lower risk of arrhythmias [16,17]. Temporary mechanical circulatory support may be required when pharmacological therapy fails to maintain adequate perfusion [16,17]. The IABP, once widely used, is no longer recommended routinely after the IABP-SHOCK II trial demonstrated no mortality benefit [16-18]. Advanced devices such as venoarterial extracorporeal membrane oxygenation (VA-ECMO), Impella, or TandemHeart may provide more substantial circulatory support [16,17]. Device selection depends on the dominant pathophysiological mechanism: VA-ECMO offers complete cardiopulmonary support but increases left ventricular afterload, whereas microaxial pumps such as Impella provide direct LV unloading and can reduce myocardial wall stress in patients with isolated left ventricular failure [16,17].

It is worth noting that an IABP was used in our case because alternative mechanical circulatory support devices, such as Impella or VA-ECMO, were not available at AUBMC. Although contemporary practice increasingly favors advanced mechanical circulatory support for the management of severe cardiogenic shock, IABP remains a practical and widely available option, particularly in resource-limited settings or as a bridge to more advanced support.

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In our case, survival despite total left main occlusion likely reflects a synergistic interplay of rapid revascularization and hemodynamic support. Additionally, a small left circumflex artery (LCX) arising anomalously from the RCA may have provided partial lateral wall perfusion, while timely primary PCI and circulatory support with vasoactive agents and IABP helped maintain coronary and systemic perfusion during cardiogenic shock. However, this interpretation remains speculative. The present report demonstrates temporal coexistence of anomalous LCX anatomy and survival after catastrophic LMCA occlusion, but it cannot prove that the anomalous vessel improved survival or materially altered the clinical course.

Conduction System Injury

Extensive anterior or septal infarction may involve the His–Purkinje conduction system, resulting in significant conduction disturbances. In such cases, these abnormalities often reflect extensive myocardial injury rather than isolated electrical dysfunction [19]. Moreover, sinus node dysfunction with sinus bradycardia or sinus pauses can occur after acute myocardial infarction, most classically in inferior wall myocardial infarction, and is usually related to ischemia of the sinus node/its arterial supply [20]. The coronary artery most often implicated is the RCA because the sinus node artery arises from the RCA in most individuals. Less commonly, it arises from the LCX, and in rare instances, a dual arterial supply is present; therefore, proximal RCA occlusion is the typical culprit, although proximal LCX disease can also lead to post-myocardial infarction sinus pauses or sinus node dysfunction, depending on the individual coronary anatomy [20,21]. For patients with persistent high-grade atrioventricular block or symptomatic sinus node dysfunction following myocardial infarction, permanent dual-chamber pacemaker implantation may be indicated according to current guideline recommendations [19].

Moreover, acute myocardial ischemia can lead to sinus node dysfunction due to abnormalities in autonomic tone triggered by the acute myocardial ischemia [22]. In addition, mechanical stretch following myocardial infarction and heart failure may impair the highly sensitive sinus node automaticity, contributing to delayed sinus pause independent of direct arterial supply [22]. Furthermore, although ischemia can cause direct injury to the SA node tissue itself, which is often vascular injury, it can also be a result of the broader physiological stressors associated with a myocardial infarction [22]. In the present case, the delayed sinus pause occurring 10 days after extensive LM–LAD infarction is more plausibly explained by indirect damage or ischemic injury to the sinus node region and adjacent atrial tissue, compounded by subacute post-infarction remodeling rather than by direct occlusion of a canonical sinus node artery; however, this remains only a hypothesis. Moreover, given the role of LCX in atrial perfusion [23], the anomalous origin of the LCX from the RCA may have further altered atrial perfusion and contributed to this atypical presentation. However, this remains speculative, and a direct causal relationship cannot be established.

Indications for Aspiration Thrombectomy

The role of manual thrombus aspiration in acute STEMI has evolved substantially in light of contemporary evidence. Current guidelines no longer recommend its routine use during primary PCI (Class III, Level of Evidence A), based on large randomized trials such as TASTE and TOTAL, which demonstrated no reduction in major clinical outcomes, including mortality and reinfarction, and identified a potential increase in stroke risk [24]. However, thrombus aspiration may still be considered as a selective bailout strategy in cases with a high residual thrombus burden or impaired flow after initial vessel recanalization. Overall, current practice supports a highly selective rather than routine approach to thrombus aspiration [24]. In the present case, given the catastrophic presentation with left main coronary artery occlusion and the anticipated large thrombus burden (**Figure 2B**), manual thrombus aspiration was selectively performed as a bailout strategy to facilitate rapid flow restoration, reduce distal embolization, and optimize subsequent revascularization.

Comparison With Previous Case Reports

In contrast to previously reported survivors of LMCA STEMI complicated by cardiogenic shock, Ibdah et al described a younger 44-year-old patient rescued with urgent PCI and IABP after ventricular tachycardia (VT)/cardiac arrest, with immediate post-procedural blood pressure improvement, and subsequent vasopressor tapering and discontinuation [6]; Rao et al reported a 35-year-old patient requiring vasoactive agents, IABP, and VA-ECMO [7]; while our patient survived without VA-ECMO despite profound shock, severe pulmonary edema, and prolonged ventilation. Khanal et al described a 60-year-old patient with VT/asystole requiring cardiopulmonary resuscitation, temporary pacing, IABP, and vasopressors [8]. Compared with the above-cited cases, the additional contribution of the present case report lies in documenting the combined presentation of total distal LMCA occlusion, profound cardiogenic shock requiring escalation to triple vasoactive therapy, IABP-only mechanical support in a resource-limited setting, and delayed symptomatic sinus node dysfunction requiring permanent pacemaker implantation.

Conclusions

This case highlights the importance of rapid recognition and aggressive multidisciplinary management in patients presenting

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with total LMCA occlusion complicated by cardiogenic shock, and illustrates the potential for survival when prompt revascularization and aggressive hemodynamic support are successfully implemented. Additionally, it underscores the occurrence of significant conduction system injury following extensive myocardial infarction, occasionally necessitating permanent pacing therapy.

Department and Institution Where Work Was Done

American University of Beirut Medical Center, Beirut, Lebanon.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report, including the use of clinical data and imaging derived from his medical record.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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