

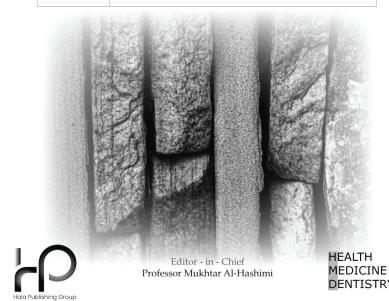
International Journal of Comprehensive Health, Medicine, and Dentistry

IJCHMD 2026;2(1) 11-18

International Journal of Comprehensive Health, Medicine, and Dentistry
Year 2026 Year 2026, Volume 2.

Original Article

Acute Anterior STEMI in a 21-Year-Old Male with Modifiable Lifestyle Risk Factors: A Case Report



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Abstract

Background:

Acute myocardial infarction (MI) in very young adults is rare but associated with significant morbidity and long-term risk.

Methods:

A 21-year-old obese male (BMI 35.2 kg/m²) with smoking history and family history of coronary artery disease presented with acute anterior ST-elevation myocardial infarction (STEMI). Coronary angiography revealed single-vessel disease with complete occlusion of the proximal left anterior descending (LAD) artery. Primary percutaneous coronary intervention (PCI) with drug-eluting stent (DES) implantation restored TIMI 3 flow. Post-procedure, the patient developed right bundle branch block but remained hemodynamically stable. High-sensitivity troponin I peaked at 242,850.6 pg/mL. He was discharged on optimized guideline-directed medical therapy (GDMT). On follow-up, he remained stable, with adjustment of medications and emphasis on aggressive secondary prevention.

Discussion:

This case illustrates the importance of recognizing acute coronary syndromes in very young adults, where modifiable risk factors—particularly obesity and smoking—play a critical role. Adherence to ESC/ACC guideline-based reperfusion and preventive strategies is essential to optimize outcomes.

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Received: 7 November 2025 **Revised:** 14 November 2025 **Accepted:** 12 December 2025

Published: 31 January 2026



Take-Home Message:

- Acute MI can occur in very young adults with modifiable risk factors such as smoking and obesity.
- Early reperfusion, GDMT, and lifestyle modification are crucial for long-term outcomes.

Abbreviations:

1. MI – Myocardial Infarction
2. STEMI – ST Elevation Myocardial Infarction
3. LAD – Left Anterior Descending
4. PCI – Percutaneous Coronary Intervention
5. GDMT – Guideline Directed Medical Therapy
6. DES – Drug-Eluting Stent
7. ECG – Electrocardiogram
8. BMI – Body Mass Index
9. LVEF – Left Ventricular Ejection Fraction
10. RBBB - Right Bundle Branch Block

2. History of Presentation

A 21-year-old Asian male (height 160 cm, weight 90 kg, BMI 35.2 kg/m²) presented to the emergency department with one hour of severe central chest pain radiating to the left arm, associated with diaphoresis. He reported a similar episode the previous night, treated at a local hospital where electrocardiogram (ECG) demonstrated anterior ST-elevation. He was administered aspirin, heparin, and ticagrelor but left against medical advice (AMA) after pain relief. On arrival, his blood pressure was 160/112 mmHg, pulse 96 bpm, respiratory rate 17 breaths per minute, and oxygen saturation 98% on room air. Cardiovascular examination showed no signs of heart failure, and he was classified as Killip class I. ECG confirmed sinus rhythm with 2–5 mm ST-segment elevation in V1–V6 with Q-wave formation.

3. Past Medical History

The patient had no history of hypertension, diabetes, or prior dyslipidemia. Notable cardiovascular risk factors included class II obesity (BMI 35.2 kg/m²), active cigarette smoking for three years, and a positive family history of premature coronary artery disease. He denied alcohol consumption or illicit drug use.

4. Differential Diagnosis

Differentials considered included ACS, myopericarditis, pulmonary embolism, and musculoskeletal chest pain. Given the classical symptoms, dynamic ECG changes, and elevated troponins, acute anterior STEMI was the working diagnosis.

5. Investigations

Initial laboratory testing revealed a markedly elevated high-sensitivity troponin I of 6,272 pg/mL, peaking at 242,850.6 pg/mL. Renal function was within normal limits at presentation (creatinine 0.92 mg/dL) with a mild rise to 1.16 mg/dL during admission. Complete blood count showed hemoglobin 16 g/dL, leukocytosis with WBC 15.9 × 10⁹/L, and platelets 338 × 10⁹/L. Electrolytes were sodium 135 mmol/L, potassium 4.8 mmol/L, chloride 98 mmol/L, and bicarbonate 22 mmol/L.

Liver enzymes were mildly elevated (ALT 61 U/L). The lipid profile demonstrated dyslipidemia with total cholesterol 234 mg/dL, triglycerides 252 mg/dL, LDL 145 mg/dL, and HDL 39 mg/dL.

Transthoracic echocardiography revealed a left ventricular ejection fraction (LVEF) of 35–40% with akinesia of the anterior wall, apex, and mid-septum, consistent with acute anterior infarction. Diastolic function was grade I impaired relaxation, while right ventricular systolic function was preserved (TAPSE 20 mm). Cardiac chambers and valves were normal, and the inferior vena cava was normal and collapsing. Urgent coronary angiography, performed via right radial access, demonstrated single-vessel coronary artery disease with complete occlusion of the proximal LAD. Left ventricular end-diastolic pressure was elevated at 30 mmHg. A DES (ONYX 3.5 × 38 mm, Medtronic) was successfully deployed, restoring patency (100% → 0% stenosis) with a post-intervention LVEF of 35–40%. The procedure was uncomplicated, with stable periprocedural hemodynamics (BP 114/66 mmHg, HR 96/min, SpO₂ 98%).

6. Management

The patient was diagnosed with acute anterior wall myocardial infarction, Killip class I. He underwent emergent primary percutaneous coronary intervention with implantation of a drug-eluting stent to the proximal LAD, restoring TIMI 3 flow. Post-procedure, he developed a new right bundle branch block (RBBB) but remained hemodynamically stable. GDMT was initiated, including dual antiplatelet therapy, high-intensity statin, beta-blocker, angiotensin receptor blocker (later transitioned to angiotensin receptor-neprilysin inhibitor), and spironolactone.

7. Outcome and Follow-Up

The patient had an uncomplicated hospital course and was discharged on day 4 in stable condition with counseling on smoking cessation, dietary modification, and adherence to GDMT. At 10-day follow-up, he reported mild muscle aches but no symptoms of heart failure. ECG demonstrated sinus rhythm with normal intervals. Medications were optimized, including uptitration of spironolactone to 50 mg daily, transition from valsartan to sacubitril/valsartan 50 mg BID, and addition of ezetimibe for lipid management. Laboratory monitoring (creatinine, potassium, ALT, urine DR) was arranged, and repeat echocardiography was scheduled for 6–12 weeks. He remains clinically stable with reassessment planned at one month.

8. Discussion

Acute MI in very young adults is rare but increasingly recognized, particularly in South Asia.

Epidemiological studies demonstrate that smoking, obesity, dyslipidemia, and positive family history are the most prevalent contributors¹⁻³. Our patient, at 21 years of age, presented with several of these risk factors, emphasizing the multifactorial nature of premature coronary artery disease.

From a pathophysiological perspective, cigarette smoking accelerates atherosclerosis through endothelial dysfunction, inflammation, and platelet activation, while obesity adds systemic inflammation, insulin resistance, and atherogenic lipid abnormalities^{4,5}.

The clustering of these factors in genetically predisposed individuals can lead to acute coronary occlusion at a very young age.

From a clinical perspective, this case reinforces the importance of maintaining suspicion for acute coronary syndromes even in patients in their early 20s. Prior studies have shown that young patients are often misdiagnosed, delaying reperfusion therapy and worsening outcomes^{6,7}. In our patient, initial presentation at a local hospital led to ACS management, but leaving AMA contributed to re-presentation with STEMI. Guidelines emphasize urgent reperfusion in STEMI, ideally within 120 minutes of diagnosis.

Both the 2023 ESC Guidelines for ACS¹¹ and the 2021 ACC/AHA/SCAI¹² Guideline for Coronary Revascularization recommend primary PCI as the preferred strategy, with dual antiplatelet therapy, beta-blockers, renin-angiotensin system inhibitors, and high-intensity statins as part of guideline-directed medical therapy (GDMT)^{8,9}. Our management aligned with these recommendations, with successful LAD stenting and initiation of GDMT.

An additional learning point is the post-PCI development of right bundle branch block, which underscores the potential for electrical instability after reperfusion. Although the patient remained hemodynamically stable, vigilance is required as conduction abnormalities are associated with larger infarct size and worse prognosis.

This case has broader public health implications. South Asian registries demonstrate that myocardial infarction occurs at younger ages compared with Western populations, with smoking and obesity as dominant drivers¹⁰. Addressing these modifiable risk factors requires not only individual counseling but also population-level interventions, including tobacco cessation programs, early cardiovascular screening, and promotion of healthy lifestyles among adolescents and young adults.

9. Conclusions

Premature myocardial infarction can occur even in very young adults, with modifiable lifestyle factors playing a central role. Recognition of ACS in this population is critical, and management should follow established guideline-directed pathways. Long-term outcomes depend on adherence to GDMT and aggressive secondary prevention strategies.

10. Take-Home Messages

- Premature myocardial infarction can occur in very young adults, particularly with risk factors such as smoking and obesity.
- Early reperfusion, GDMT optimization, and aggressive lifestyle modification are essential to long-term survival.

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1. Visual Summary

Table 1: Timeline of the Case

Timeline	Events
Day 1	In the Emergency Department, a young male with history of smoking and positive family history for CAD presented with 1-hour severe central chest pain. He had a prior episode the previous night, partially relieved after ACS at another hospital. On arrival: BP 160/112, HR 96. ECG showed ST elevation in V1–V6 with Q waves. Diagnosed with anterior wall STEMI (Killip I). Initiated on ACS protocol and referred for urgent PPCI.
Day 1	In the cardiac catheterization laboratory, angiography revealed an occluded proximal LAD with LVEDP 30 mmHg and EF 35%. Successful PCI with drug-eluting stent placement to LAD was performed. Post-procedure: sinus tachycardia, persistent ST elevation, EF ~35%. No immediate complications noted.
Day 2	In the Coronary Care Unit (CCU), the patient was stable but developed new RBBB and right axis deviation. Reported atypical chest pain and tachycardia but no fluid overload. Echocardiography: EF 35–40%, anterior/apical akinesia, grade I diastolic dysfunction. GDMT optimized with uptitration of ARB, initiation of beta-blocker, aldosterone antagonist, and IV furosemide as needed.
Day 3	In the Step-Down Unit, the patient remained hemodynamically stable. Vitals: BP ~90/56, SpO ₂ 98%. Continued mild chest discomfort but no signs of heart failure. Medications adjusted (switch to bisoprolol, discontinuation of enoxaparin). Routine labs and ECG monitoring performed.

Table 2: Equipment List**Percutaneous Coronary Intervention of the Proximal LAD Access**

- Ultrasound machine for vascular access
- 6-French radial sheath (Terumo, Japan)
- 0.035" J wire
- TR Band radial artery compression device (Terumo, Japan)

Guiding System

- 6-French Judkins Left (JL 3.5) guiding catheter (Medtronic, USA)
- 0.014" Balance Middle Weight (BMW) coronary guidewire (Abbott Vascular, USA)

Lesion Preparation

- 3.75 × 15 mm NC balloon (Boston Scientific, USA)

Stent Deployment

- 3.5 × 38 mm Onyx™ DES (Medtronic, USA)
- 3.0 × 15 mm non-compliant post-dilatation balloon (BrosMed, China)

Adjuncts

- Intravenous unfractionated heparin
- Intracoronary nitroglycerin
- Standard cardiac monitoring and pressure transduction system

**Figure 1: Baseline ECG.**



Figure 2: Post PCI ECG.



Figure 3: Coronary angiography before PCI

Complete occlusion of the proximal left anterior descending (LAD) artery with absent distal flow (TIMI 0).

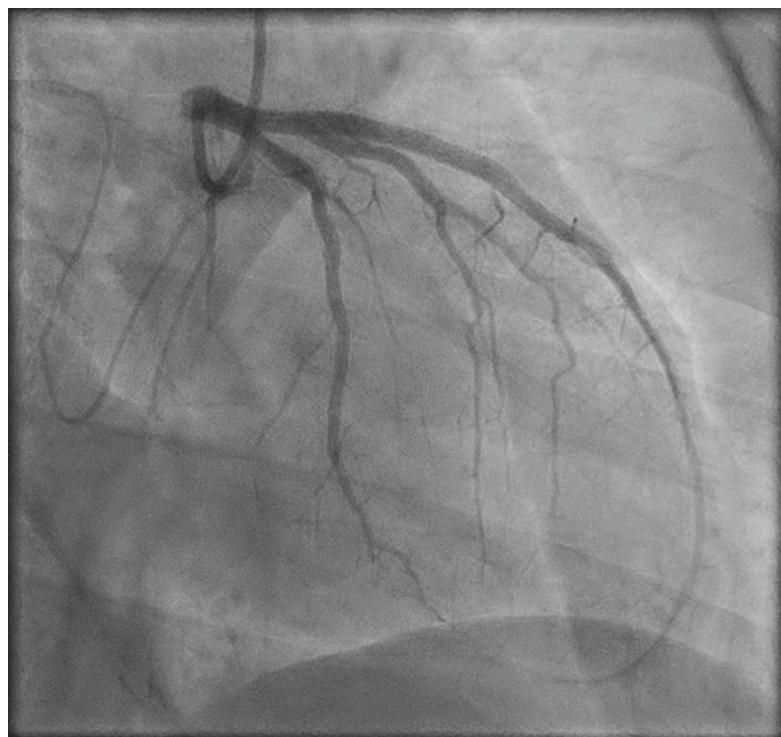


Figure 4: Coronary angiography after PCI

Successful revascularization of the proximal LAD with deployment of a drug-eluting stent and restoration of TIMI 3 flow.

Video 1. Coronary angiography before PCI

Complete occlusion of the proximal left anterior descending (LAD) artery with absent distal flow (TIMI 0).

Video 2. Coronary angiography after PCI

Successful revascularization of the proximal LAD with deployment of a drug-eluting stent and restoration of TIMI 3 flow.