

ST elevation myocardial infarction in a teenager: A case report

Alisha Inayat

Abstract

The case report discusses a young male who presented on 14th June 2024, to the emergency department of Shifa International Hospital, Islamabad with epigastric pain. He was subsequently diagnosed as ST-elevation myocardial infarction (STEMI) and moved to the catheterisation lab for urgent coronary angiography and coronary reperfusion. This case highlights the fact that underestimation of possible coronary artery disease (CAD) in young patients can lead to significant morbidity and mortality if not diagnosed and treated timely.

Keywords: STEMI, myocardial infarction, coronary artery disease, teenager, coronary vasospasm.

DOI: <https://doi.org/10.47391/JPMA.22693>

Introduction

Myocardial infarctions, including STEMI, account for less than 1% of all cases in individuals under 30.¹ Considering these statistics, STEMI in patients under 20 years age is extremely rare. Generally, myocardial infarctions are much more common in older populations, particularly those with traditional risk factors such as hypertension, diabetes, hypercholesterolaemia and family history of CAD, thus making STEMI uncommon in teenagers and young adults.² Nevertheless, the incidence is rising among patients <35 years of age secondary to risk factors like smoking, obesity, hyperuricaemia, non-obstructive coronary artery disease and genetic polymorphisms related to blood coagulation, fibrinogen and homocysteine.¹ Binge drinking alcohol and drug abuse are also recognized contributors in this age group.³ However, in recent years, there has been a growing recognition of myocardial infarction occurring in the absence of obstructive coronary artery disease, particularly in younger patients. This entity is referred to as Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) and encompasses a heterogeneous group of underlying mechanisms, including coronary vasospasm, coronary thrombosis, microvascular dysfunction, and myocarditis.⁴ MINOCA has been reported to account for

approximately 5–10% of all myocardial infarctions, with a higher prevalence in younger individuals and those without conventional cardiovascular risk factors. However, the case being discussed here had no predisposing condition or risk factors identified, thus making it an extremely rare and interesting case that has not been previously reported anywhere.

Case Report

A 19-year-old male with no previously known comorbidities presented to the emergency department of Shifa International Hospital, Islamabad on 14th June 2024, with a history of diarrhoea one day prior, which had settled on his own. He complained of severe epigastric pain radiating to the retrosternal region since the previous night, along with vomiting. He had no history of smoking, drinking alcohol or substance abuse. He was a religious student by profession. He had no significant family history of coronary artery disease or metabolic syndrome. He had no drug or food allergies.

He was vitally stable on arrival, heart rate: 77 beats/minute, blood pressure: 126/80 mmHg, respiratory rate: 18 breaths/minute, SpO₂: 99% on room air, afebrile, Glasgow Coma Scale (GCS): 15/15. Pain score was 10/10 (zero being no pain; 10 being the worst pain imaginable). His examination was completely unremarkable.

He was prescribed symptomatic relief based on a provisional diagnosis of acute gastritis. During the emergency department (ED) stay, he remained vitally stable but with non-remitting pain even after pain relief was topped up with opioids. It was eventually decided to perform an electrocardiogram (ECG) as part of the protocol for patients presenting with epigastric pain. The ECG showed ST elevations in the anterolateral leads (I, aVL, V2, V3, V4, V5 and V6) as shown in Figure 1.

A STEMI alert was announced as part of the hospital's local policy and the patient was immediately moved to the resuscitation room. All medications included in the hospital's acute coronary syndrome protocol were administered to the patient (aspirin 300 mg, ticagrelor 180 mg, rosuvastatin 40 mg orally and Inj. Heparin 5000 IU intravenously). Catheterisation lab was activated and the patient was quickly moved for emergent coronary angiography. Angiography was performed via the left

Emergency Department, Shifa International Hospital, Islamabad, Pakistan.

Correspondence: Alisha Inayat. e-mail: alishainayat309@gmail.com

ORCID ID: 0009-0008-2539-6263

Submission completed: 21-12-2024 **1st Revision received:** 07-03-2025

Acceptance: 13-06-2026

Last Revision received: 12-06-2026

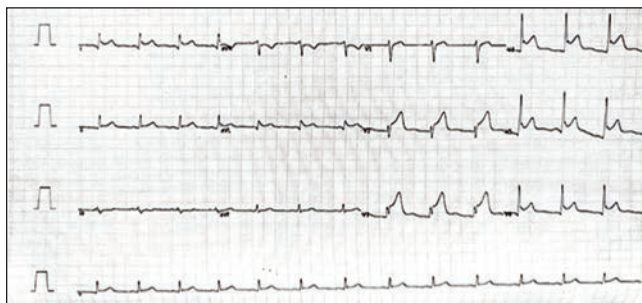


Figure-1: ST elevations in anterolateral leads I, avL, v2, v3, v4, v5, v6.

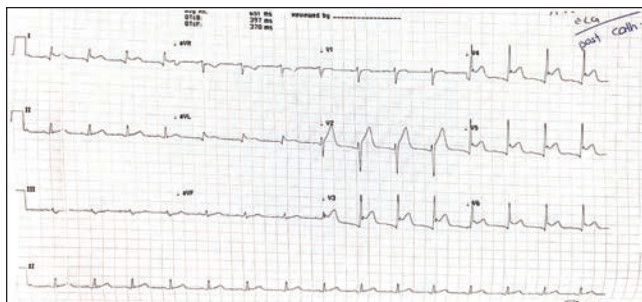


Figure-2: Persistent ST elevations in anterolateral leads.

radial approach using 6 French JR 4 and JL 3.5 catheters. Coronary angiography revealed normal epicardial coronary arteries with the exception of mid systolic bridging of the left anterior descending artery. Myocardial bridging refers to an intramyocardial course of a coronary artery segment, resulting in systolic compression. In most cases, particularly when mild, it is considered a benign anatomical variant and is often clinically insignificant; however, in certain circumstances, it has been associated with myocardial ischaemia, coronary vasospasm, and acute coronary syndromes. A left ventriculogram was not performed (reason not disclosed in patient's confidential medical record). The conclusion was normal coronary arteries; likely coronary vasospasm.

The recommendation after coronary angiography was to prescribe calcium channel blockers, single antiplatelet therapy, and a statin.

The patient was shifted to the cardiac care unit (CCU) after angiography. ECG changes persisted post angiography as well (ST elevations in the anterolateral leads) as shown in Figure 2.

Echocardiogram showed an ejection fraction of 65% and mild tricuspid regurgitation.

His blood workup came back as shown in the table. Although serial cardiac biomarkers could not be obtained due to the patient's early discharge against medical advice, the markedly elevated initial troponin I (26,354 pg/mL) and CK-MB (72.6 ng/mL) levels were consistent with an acute

Table: Lab parameters with their values and reference ranges

Laboratory Parameter	Value with Unit	Reference Range (as per our hospital's laboratory)
TLC	16,970 /UL	4000 /UL - 11000 /UL
Hb	15 g/dL	Male (13 - 18) g/dL; Female (11.6 - 16.5) g/dL
Platelets	256,000 /UL	150,000 /UL - 400,000 /UL
Trop-I	26,354 pg/mL	Female (up to 15.6 pg/mL); Male (up to 34.2 pg/mL)
CKMB	72.6 ng/mL	Female (up to 3.4 ng/mL); Male (up to 7.2 ng/mL)
Sodium	136 mEq/L	(136 - 145) mEq/L
Potassium	3.5 mEq/L	(3.5 - 5.1) mEq/L
Chloride	99 mEq/L	(98 - 107) mEq/L
Bicarbonate	25 mEq/L	(22-29) mEq/L
Ionized calcium	4.5 mg/dL	(4.6 - 5.3) mg/dL
BUN	6 mg/dL	Adult 18 - 60 years: 6-20 mg/dL; 60-90 years: 8-23 mg/dL
Creatinine	0.8 mg/dL	Male (0.72 - 1.25) mg/dL; Female (0.57 - 1.11) mg/dL

TLC: total leukocyte count; Hb: haemoglobin; Trop-I: troponin I; CKMB: creatine kinase B; BUN: blood urea nitrogen; pg: picogram; mEq: milliequivalent; mg: milligram; L: litre; dL: decilitre; ng: nanogram; mL: millilitre

myocardial injury. In the appropriate clinical context and with persistent ST-segment elevation on electrocardiography, these findings supported the diagnosis of acute ST-elevation myocardial infarction. The absence of serial measurements limited assessment of biomarker trends and infarct evolution. He was inquired in detail once again (this time by his cardiologist) regarding any history of substance abuse such as cocaine or others but he strongly denied. His family also confirmed his statement to be accurate. He had financial constraints and requested to leave against medical advice after 8 hours of stay in the hospital. He was discharged against medical advice and was advised to go to a government tertiary care setup for further management and observation. Prior to discharge, he and his family were counselled in detail regarding the diagnosis, potential complications, and warning symptoms warranting immediate medical attention, including recurrent chest pain, syncope, dyspnoea, or palpitations. He was advised strict outpatient cardiology follow-up, repeat cardiac biomarker assessment, echocardiographic reassessment, and further evaluation including cardiovascular magnetic resonance imaging to exclude myocarditis or other underlying pathology. He was also counselled regarding medication adherence and avoidance of potential vasospastic triggers. Unfortunately, the patient was subsequently lost to follow-up, and long-term outcomes could not be assessed.

Consent for publication of this case study was obtained from the patient prior to discharge.

Discussion

This report highlights a unique case of STEMI in a teenager. He had no risk factors for coronary artery disease and no history of substance abuse that could have precipitated coronary vasospasm leading to STEMI. The underlying cause of his STEMI remains uncertain and warrants further research into factors that can cause coronary vasospasm.

The absence of obstructive coronary artery disease on angiography places this presentation within the spectrum of Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA), a diagnostic entity increasingly recognized in younger patients.

Coronary vasospasm is a well-recognized cause of MINOCA and can result in transient coronary occlusion, leading to myocardial ischaemia and infarction. Established precipitants include cocaine use, ethanol intoxication, withdrawal of calcium channel blockers, tobacco use, and connective tissue disorders.⁴ Hypersensitivity-mediated coronary vasospasm, such as Kounis syndrome, has also been reported, where allergic reactions provoke coronary spasm via inflammatory mediators.⁵ However, our patient had no history of substance use, medication exposure, or clinical features suggestive of hypersensitivity reactions, making these causes unlikely.

During coronary angiography, diffuse coronary vasospasm was suspected based on angiographic interpretation by the attending cardiologist. Although transient vasospasm involving access vessels such as the radial artery has been described in the literature, objective documentation or provocative testing was not performed in this case, representing a limitation and warranting cautious interpretation of this finding.

Myocarditis remains an important differential diagnosis in young patients presenting with chest pain, ST-segment elevation, and elevated cardiac biomarkers. Viral myocarditis—particularly due to Parvovirus B19—has been associated with endothelial dysfunction and coronary vasospasm, occasionally mimicking acute myocardial infarction.⁶ While myocarditis could not be definitively excluded in this case, the angiographic appearance of normal coronary arteries and the clinical impression of vasospasm reduced its likelihood. Nonetheless, the absence of advanced imaging limited definitive differentiation.

Systemic vasculitides involving the coronary arteries, including Kawasaki disease, Takayasu arteritis, polyarteritis nodosa, ANCA-associated vasculitis, Kawasaki-like syndrome associated with SARS-COV-2 infection and giant cell arteritis, have been implicated in coronary vasospasm and myocardial infarction.⁷ Additionally, vasospastic angina leading to myocardial infarction has been reported in eosinophilic granulomatosis with polyangiitis, even in the absence of overt systemic manifestations.⁸ Although there were no clinical features suggestive of systemic autoimmune disease in our patient, these entities remain important considerations, particularly in young patients with unexplained coronary events.

Current diagnostic algorithms for MINOCA, including the European Society of Cardiology (ESC) 2020 Position Paper,⁹ emphasize a structured, stepwise evaluation to identify the underlying etiology. This includes exclusion of alternative diagnoses, intracoronary imaging where appropriate, and early use of cardiovascular magnetic resonance imaging (CMRI) to differentiate between myocardial infarction, myocarditis, and stress-induced cardiomyopathy. CMRI is now considered a key investigation in MINOCA presentations, as it provides tissue characterization that can guide diagnosis, management, and prognosis. Unfortunately, CMRI could not be performed in this patient due to early discharge, leaving diagnostic uncertainty.

This case underscores that serious cardiac events can occur at any age and highlights the importance of maintaining diagnostic vigilance, even in young patients without traditional cardiovascular risk factors. It also reinforces the need for early electrocardiographic evaluation in patients presenting with atypical symptoms such as epigastric pain, regardless of age. While the inability to complete a comprehensive diagnostic workup limits definitive conclusions, this case contributes to the growing body of literature on MINOCA in adolescents and emphasizes the importance of structured evaluation and follow-up in such presentations.

This case has certain limitations, as it reviews only a single case report in which a teenager with no previously known comorbid conditions or risk factors for CAD presented with STEMI. Additionally, the patient did not stay long enough for any further workup to be done to rule out known possible causes for coronary vasospasm such as toxicology screen, serial troponins, and CMRI. Hence, more literature is needed on this topic with other similar cases being reported or research being conducted to answer all these questions that this study leaves unanswered.

Conclusion

The presented case is an extremely rare and probably first reported case in South Asia of a nineteen-year-old boy with STEMI secondary to coronary vasospasm of unknown etiology. This underscores the importance of early electrocardiographic evaluation in patients presenting with atypical chest or epigastric pain, even in the absence of traditional cardiovascular risk factors. It highlights the diagnostic challenges associated with myocardial infarction with non-obstructive coronary arteries in young individuals and emphasizes the need for a systematic diagnostic approach, including cardiovascular magnetic resonance imaging when feasible. Furthermore, this case illustrates that serious cardiac events are not confined to any specific age group and reinforces the need for clinicians

to maintain a broad differential diagnosis and a high index of suspicion. It also reinforces the old school protocol of getting an ECG done on ALL patients presenting with epigastric pain.

Although limited by incomplete diagnostic evaluation and lost to follow-up, such rare presentations contribute to the existing literature and may prompt further research into unexplained coronary vasospasm and myocardial infarction in adolescents.

Disclaimer: None.

Conflict of Interest: None.

Funding Sources: None.

References

1. Usuda D, Tanaka R, Suzuki M, Takano H, Hotchi Y, Shimozaawa S, et al. ST-elevation acute myocardial infarction in a young man. *J Med Cases*. 2022;13:281-9. doi:10.14740/jmc3939.
2. Hajar R. Risk factors for coronary artery disease: historical perspectives. *Heart Views*. 2017;18:109-14. doi:10.4103/HEARTVIEWS.HEARTVIEWS_106_17.
3. Sood A, Singh A, Gadkari C. Myocardial infarction in young individuals: a review article. *Cureus*. 2023;15. doi:10.7759/cureus.37102.
4. Escalon JG, Bang TJ, Broncano J, Vargas D. Myocardial infarction with nonobstructive coronary arteries (MINOCA): potential etiologies, mimics and imaging findings. *Curr Probl Diagn Radiol*. 2021;50:85-94. doi:10.1067/j.cpradiol.2020.02.014.
5. Kounis NG. Coronary hypersensitivity disorder: the Kounis syndrome. *Clin Ther*. 2013;35:563-71. doi:10.1016/j.clinthera.2013.02.022.
6. Kumar A, Bagur R, Béliveau P, Potvin JM, Levesque P, Fillion N, et al. Acute myocarditis triggering coronary spasm and mimicking acute myocardial infarction. *World J Cardiol*. 2014;6:1045-8. doi:10.4330/wjc.v6.i9.1045.
7. Gori T. Coronary vasculitis. *Biomedicines*. 2021;9:622. doi:10.3390/biomedicines9060622.
8. Chapman FA, Dhaun N, Dweck M, Mills NL. Coronary vasospasm in eosinophilic granulomatosis with polyangiitis. *Rheumatology (Oxford)*. 2020;59. doi:10.1093/rheumatology/keaa357.
9. 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;42:1289-367. doi:10.1093/eurheartj/ehaa575.

Author Contribution:

AI: Concept, design, data acquisition, analysis, interpretation, drafting, revision, final approval and agreement to be accountable for all aspects of the work.