

Case Report

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Anterior ST-Segment Elevation Myocardial Infarction in a 54-Year-Old Male Patient with Uncontrolled Hypertension: A Case Report

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ABSTRACT

Background:

ST-elevation myocardial infarction (STEMI) is the most severe and life threatening form of acute coronary syndrome. It is caused by complete occlusion of an epicardial coronary artery which leads to transmural myocardial necrosis. Rapid diagnosis of STEMI and reperfusion is essential to limit infarct size and reduce mortality. Uncontrolled hypertension is a major modifiable cardiovascular risk factor that causes accelerated atherosclerosis and predisposes to acute coronary events including STEMI.

Case Report:

A 54-year-old male with uncontrolled hypertension was brought with a 15-minute history of acute chest pain radiating to the back and left arm. Blood pressure on arrival was found to be 147/100 mmHg and oxygen saturation was 94% on room air. A 12-lead ECG showed presence of ST-segment elevation in leads V2–V4 which subsequently progressed to extensive ST elevation across V1–V6, I, II, and aVR. Cardiac troponin T rose serially from below the detection threshold to 602 ng/L. Random blood glucose was found to be 438 mg/dL and serum potassium was 3.3 mmol/L. The patient received dual antiplatelet therapy, atorvastatin and sublingual nitrate. Subsequently he was referred to higher centre for primary percutaneous coronary intervention.

Conclusion:

Serial ECG monitoring and cardiac biomarker assessment are essential for diagnosing as well as tracking progression of STEMI. Concurrent diagnosis and management of associated risk factors such as hypertension, hyperglycemia and electrolyte imbalance — alongside prompt reperfusion therapy are important from the point of view of optimal outcomes.

Keywords: Anterior STEMI, Cardiac Troponin, Primary Percutaneous Coronary Intervention, ST-Elevation Myocardial Infarction

INTRODUCTION

Coronary artery disease (CAD) arises from progressive atherosclerosis of the coronary arteries — a process of endothelial injury, lipid deposition, and inflammatory plaque formation that narrows the arterial lumen and impairs myocardial perfusion. This creates an imbalance between myocardial oxygen supply and demand that, over time, produces the broad clinical spectrum of coronary artery disease. Presentation ranges from stable angina, representing fixed flow limitation, to the acute coronary syndromes (ACS), which include unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). The 2025 ACC/AHA guideline for ACS describes the transition to an acute event as triggered by disruption of an atherosclerotic plaque — through rupture or superficial erosion — followed by rapid intracoronary thrombus formation that abruptly reduces or completely occludes coronary blood flow.¹ The 2023 AHA/ACC guideline for chronic coronary disease characterises the longitudinal nature of this condition and the ongoing risk of acute events even in patients on optimal medical therapy.²

Cardiovascular diseases impose a vast global mortality burden. The World Health Organization reports approximately 17.9 million cardiovascular deaths per year, representing around 32% of all global mortality; myocardial infarction and stroke together account for 85% of these deaths.³ Mortality has declined in high-income countries through improved prevention and reperfusion therapy, but more than three-quarters of cardiovascular deaths occur in low- and middle-income countries, where access to timely revascularization remains limited. In Asia, including Indonesia,

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ACS incidence continues to rise alongside increasing rates of hypertension, dyslipidaemia, and diabetes.⁴

Among all acute coronary syndromes, STEMI carries a high immediate risk of death and pump failure. It most commonly results from acute thrombotic occlusion of an epicardial coronary artery. This leads to transmural ischemia and progressive necrosis beginning within 20 minutes of flow interruption to the affected area. The anterior territory, supplied by the left anterior descending artery, is the most frequently affected and places the largest myocardial mass at risk. Myocardial salvage is strongly time-dependent: each additional minute of coronary occlusion translates into further irreversible cell loss, making rapid reperfusion the central priority in management. The Fourth Universal Definition of Myocardial Infarction classifies STEMI by the presence of new ST-segment elevation meeting specific amplitude criteria on the 12-lead ECG, accompanied by a rise and fall in cardiac biomarkers consistent with myocardial necrosis.⁵

Diagnosis rests on the 12-lead ECG, which identifies the infarct territory and triggers immediate activation of the primary percutaneous coronary intervention (PCI) pathway. Serial cardiac troponin measurement confirms necrosis and provides prognostic information, though initial levels may fall below the diagnostic threshold in the earliest hours after symptom onset. Serial ECG recordings and biomarker assessments are therefore both important in the acute diagnostic workup, with ECG changes guiding urgent reperfusion decisions and serial biomarkers providing biochemical confirmation of myocardial injury.

Hypertension is the most prevalent modifiable cardiovascular risk factor and is present in a large proportion of patients with acute MI. Uncontrolled blood pressure accelerates plaque formation and endothelial dysfunction and worsens the acute supply-demand imbalance by raising afterload. Metabolic derangements — including marked hyperglycemia and hypokalemia — are frequently encountered alongside acute MI and carry independent prognostic implications that require concurrent identification and management.

We present a case of extensive anterior STEMI in a 54-year-

old male with uncontrolled hypertension, marked hyperglycemia, and hypokalemia to illustrate the clinical presentation, electrocardiographic evolution, and initial management of this cardiovascular emergency.

CASE REPORT

A 54-year-old male was brought to the emergency department with a 15-minute history of sudden-onset left-sided chest pain. Pain was described as a heavy pressure sensation radiating to the back as well as left arm. Onset was abrupt and accompanied by profuse cold sweating, nausea, and two episodes of vomiting en route to hospital. This was his first episode of chest pain. He denied palpitations, dyspnea, fever, and cough. Past medical history included uncontrolled hypertension for which he was not taking any regular medication. There was no family history of cardiovascular disease, hypertension, or diabetes mellitus, and no known drug allergies.

On examination, the patient was moderately ill and fully conscious. On arrival blood pressure was 147/100 mm of Hg, heart rate was 51 beats per minute, respiratory rate 22 breaths per minute, temperature 36.6°C, and oxygen saturation was found to be 94% on room air. Cardiac examination showed the apex beat at the fifth intercostal space in the midclavicular line. First and second heart sounds were normal. There was no murmur or third heart sound. Chest auscultation revealed bilateral rhonchi without wheeze. Jugular venous pressure was not raised and there was no peripheral oedema.

A 12-lead ECG was immediately done which showed sinus rhythm at 57 bpm with presence of ST-segment elevation in V2–V4 leads (Figure 1). These findings were consistent with anterior STEMI. A repeat ECG was performed within the hour which demonstrated more extensive ST-segment elevation compared with the initial tracing (Figure 2). These dynamic changes supported evolution of the anterior STEMI. A portable anteroposterior chest radiograph showed a cardiothoracic ratio below 50%. Lung fields were clear bilaterally and sharp costophrenic angles with no signs of pulmonary oedema or cardiomegaly (Figure 3).

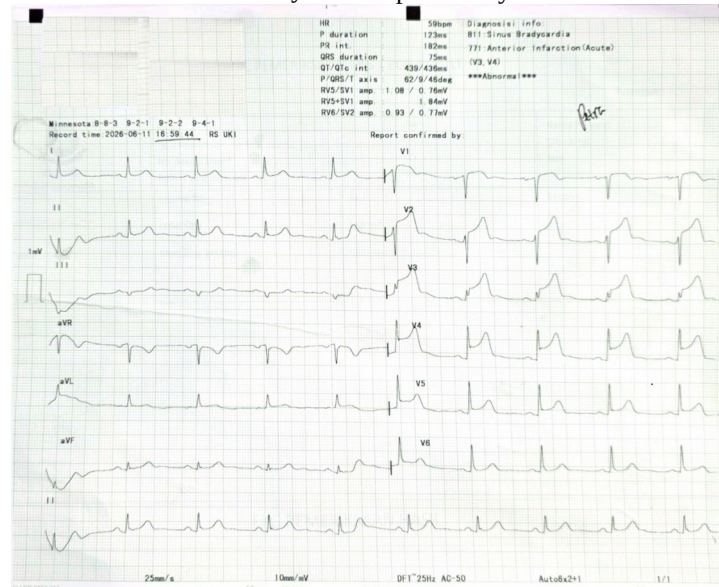


Figure 1: Initial 12-lead ECG showing sinus rhythm at 57 bpm with ST-segment elevation in leads V2–V4, consistent with anterior STEMI.

HR 69b Diagnosis info:
 P duration 118ms 800 Sinus Rhyth
 PR int 181ms 734 Suspect Anteroseptal Infa
 QRS duration 77ms ction? (V1, V2)
 QT/QTc int 410/439ms 772 Lateral Infarction (Anterol)
 P/QRS/T axis 72/19/61deg V5, V6
 RV5/SV1 amp. 0.99 / 0.62mV 131 Low Voltage (Limb Leads)
 RV5/SV1 amp. 1.61mV 611 Flat T (V4)
 RV5/SV2 amp. 0.91 / 0.65mV

Report confirmed by ****Abnormal****

25mm/s 10mm/mV DF125Hz AC-50 AutoRx2x1 1/1

showed respiratory alkalosis (pH 7.50 with PaCO₂ 28 mmHg). Urine analysis showed 4+ glycosuria and 1+ ketonuria. Despite ketonuria, the arterial blood gas showed alkalemia rather than metabolic acidosis, arguing against diabetic ketoacidosis. Serum creatinine was within normal limits, while blood urea was mildly elevated (Table 1).

The diagnosis of extensive anterior STEMI was established. Pericarditis, Prinzmetal angina, and left ventricular aneurysm

were considered in the differential diagnosis and were considered less likely on the basis of the clinical presentation and dynamic ECG evolution.

Treatment comprised aspirin 160 mg orally, clopidogrel 300 mg orally, atorvastatin 40 mg orally, sublingual isosorbide dinitrate 5 mg (repeatable every five to ten minutes up to three doses), omeprazole 40 mg intravenously, ondansetron 8 mg intravenously, and sucralfate 15 mL orally. Continuous ECG monitoring was established and intravenous access secured. These measures constituted the initial treatment before transfer. Given the diagnosis of STEMI and the availability of a primary PCI-capable referral centre, the patient was transferred urgently for coronary angiography and primary PCI.

DISCUSSION

STEMI results from acute thrombotic occlusion of an epicardial coronary artery, most commonly following rupture of an atherosclerotic plaque with thrombus formation. Anterior STEMI, caused by left anterior descending artery occlusion, places the largest ventricular mass at risk and carries the greatest burden of myocardial injury among all STEMI subtypes.⁶

This patient's presentation — sudden heavy chest pressure radiating to the back and left arm, with diaphoresis, nausea, and vomiting — is a classic symptom complex for acute myocardial ischemia. Immediate 12-lead ECG acquisition is mandatory in this setting, as ECG diagnosis of STEMI triggers the reperfusion pathway without delay.⁶ The initial recording showed ST elevation in V2–V4; serial monitoring within the hour demonstrated more extensive ST-segment elevation compared with the initial tracing, reflecting evolution of the ischemic process. This ECG evolution underscores why serial monitoring — rather than a single recording — is important: dynamic changes demonstrate progression of the electrocardiographic abnormalities and help define the evolving extent of myocardial involvement. Cardiac troponin T was undetectable on initial sampling, which is expected in very early presentations. Troponin does not reliably rise above the detection threshold until one to two hours after irreversible cell death begins. The serial rise to 602 ng/L provided biochemical confirmation of necrosis consistent with the ECG diagnosis. Current guidelines recommend serial high-sensitivity troponin measurements at presentation and two to three hours later in suspected ACS.⁶ Uncontrolled hypertension represented an important background risk in this patient. Chronic elevation of arterial pressure damages the endothelium, accelerates lipid deposition, and promotes plaque vulnerability to rupture. Mills et al. identified hypertension as a major contributor to global cardiovascular mortality and ischaemic heart disease burden.⁷ In the acute phase, elevated afterload from uncontrolled hypertension may worsen the myocardial supply-demand mismatch in the ischemic territory.

The marked hyperglycemia — with glucose values above 400 mg/dL — carries independent prognostic risk even in patients with no previous diagnosis of diabetes mellitus. Capes et al. in a systematic review found that hyperglycemia at the time of MI was associated with significantly higher in-hospital mortality in both diabetic as well as non-diabetic patients.⁹ In the present patient, the marked hyperglycemia may have represented stress hyperglycemia or previously undiagnosed diabetes mellitus; this distinction could not be established from the available data. Concurrent hypokalemia (potassium 3.3 mmol/L) also required active attention. Nordrehaug et al. showed that hypokalemia at the time of acute MI was

independently associated with increased risk of ventricular fibrillation.¹⁰ Both hyperglycemia and hypokalemia require monitoring and appropriate correction alongside the primary reperfusion strategy.

Primary PCI is the preferred reperfusion strategy for STEMI when delivered within guideline-specified time windows.⁶ De Luca et al. established that increasing delay from symptom onset to balloon inflation is associated with increased mortality, supporting the priority of urgent transfer in this case.⁸ Keeley et al., in a meta-analysis of 23 randomised trials, confirmed that primary PCI achieves higher rates of complete reperfusion and reduces re-infarction and death compared with fibrinolysis.¹¹ The dual antiplatelet pretreatment administered here — aspirin and clopidogrel — is consistent with guideline-directed initial management. Sabatine et al. demonstrated in the CLARITY-TIMI 28 trial that adding clopidogrel to aspirin significantly reduced the rate of an occluded infarct artery and major adverse cardiac events in STEMI.¹² Ibanez et al., in the 2017 ESC STEMI guidelines, further specify that systematic management of metabolic complications — including hyperglycemia and electrolyte abnormalities — is an integral part of comprehensive STEMI care.¹³

CONCLUSION

STEMI is a life threatening cardiovascular emergency in which timely diagnosis and prompt reperfusion determine myocardial salvage. This case demonstrates the importance of serial ECG monitoring and cardiac biomarker assessment in demonstrating electrocardiographic evolution and providing biochemical confirmation of myocardial necrosis. In this patient coexistence of uncontrolled hypertension, marked hyperglycemia and hypokalemia underscores the need for concurrent identification and management of associated cardiovascular and metabolic abnormalities. Early guideline-directed pharmacological management and referral to higher centre for primary PCI are the cornerstones of patients presenting with STEMI.

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