

Acute Myocardial Injury in the Setting of Hypertensive Emergency: A Case Report

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Abstract

Hypertensive emergency can cause acute myocardial injury characterized by chest pain, elevated cardiac biomarkers, and ischemic electrocardiographic changes in the absence of obstructive coronary artery disease. This presentation reflects a mismatch between myocardial oxygen supply and demand, driven by severe afterload increase, microvascular dysfunction, and subendocardial ischemia. Differentiating this entity from type 1 myocardial infarction is critical, as management priorities differ significantly. Prompt but controlled blood pressure reduction remains the cornerstone of treatment and often leads to rapid clinical and biological improvement. Recognizing this underdiagnosed presentation helps avoid unnecessary invasive procedures and guides appropriate therapeutic strategies.

Categories: Cardiology, Emergency Medicine

Keywords: acute myocardial injury, blood pressure control, ecg changes, hypertensive emergency, troponin elevation, type 2 myocardial infarction

Introduction

Hypertensive emergencies encompass a heterogeneous group of conditions defined by (1) a sudden and severe elevation in blood pressure (BP), typically $\geq 180/120$ mmHg; (2) evidence of acute hypertension-mediated target organ damage; and (3) the need for prompt, context-specific BP reduction to prevent fatal complications [1].

Among the affected organs, the heart is the most frequently involved, accounting for approximately 50% of cases, making cardiac complications the leading form of acute hypertension-mediated organ damage. Although hypertensive emergencies represent only about 0.5% of all emergency department visits, approximately 36% of patients presenting with severely elevated BP are diagnosed with this condition [2].

The major cardiac manifestations include cardiogenic pulmonary edema (32% of cases), acute coronary syndrome (ACS) (18% reported), and aortic dissection (2%). The ACS-like presentations are common and occur in approximately 10-20% of patients [3].

The fourth universal definition of myocardial infarction (MI) consists of a rise and/or fall in cardiac troponin associated with evidence of acute myocardial ischemia (chest pain, electrographic changes, and wall motion abnormalities). Type 1 MI results from atherothrombosis, whereas type 2 MI is caused by myocardial oxygen supply-demand imbalance. In contrast, acute myocardial injury refers to troponin elevation without evidence of acute ischemia. Distinguishing these entities is particularly challenging in hypertensive emergencies, where severe pressure overload may mimic ACS.

ACS has been reported in a significant subset of patients with severe hypertension, and elevated cardiac troponin levels often prompt invasive evaluation [3]. However, myocardial injury related to pressure overload may occur in the absence of obstructive coronary artery disease [4].

We report the case of a 44-year-old male with hypertension, prediabetes, and dyslipidemia, who presented with chest pain, elevated troponin, and T-wave changes on ECG, despite normal coronary arteries. This case represents acute myocardial injury triggered by a hypertensive emergency, where excessive afterload and microvascular dysfunction caused myocardial damage. Prompt recognition and carefully titrated BP reduction are essential to prevent further end-organ damage while avoiding hypoperfusion.

Case Presentation

A 44-year-old man with cardiovascular risk factors presented to the emergency department with prolonged chest pain of anginal characteristics, radiating to the left upper limb and associated with neurovegetative symptoms, including dizziness and diaphoresis. His medical history was notable for a one-year history of arterial hypertension managed with lifestyle measures, prediabetes (HbA1c 6.1%), and dyslipidemia. He

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denied smoking, alcohol consumption, or illicit drug use.

On admission, BP was markedly elevated at 211/121 mmHg symmetrically, with a heart rate of 85 beats per minute. Ophthalmologic evaluation revealed no visual disturbances, and no hypertensive retinopathy was noted. Neurological examination was unremarkable, with no focal neurological deficits or features suggestive of acute hypertensive brain injury. Cardiac auscultation revealed regular heart sounds without murmurs, and there were no signs of heart failure. The initial electrocardiogram (ECG) showed sinus rhythm with T-wave inversion in the septal, apical, and lateral leads (Figure 1).

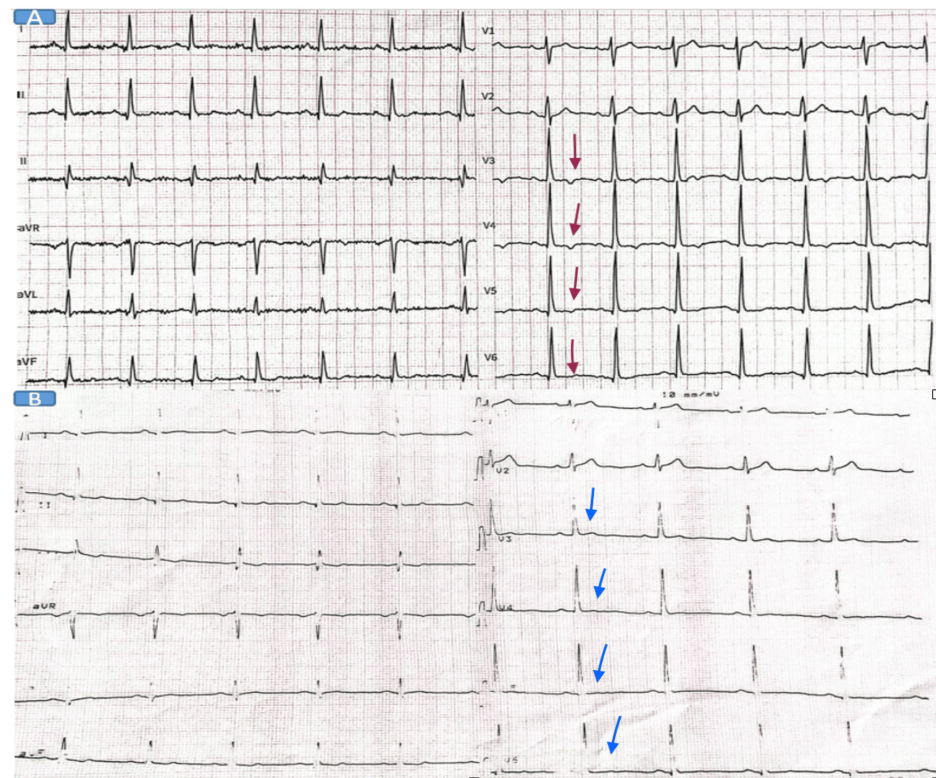


FIGURE 1: Electrocardiogram

Panel A: ECG at admission showing negative T waves in the septal, apical, and lateral leads (arrows). Panel B: The ECG performed 3 hours later showed T-wave normalization in the septal and apical leads and T-wave flattening in the lateral leads (arrows).

Given the presence of ongoing chest pain, ischemic electrocardiographic changes, and severe hypertension, an ACS in the setting of hypertensive emergency was initially suspected. Potential diagnoses included type 1 non-ST-elevation MI, type 2 MI secondary to oxygen supply-demand imbalance, acute myocardial injury related to pressure overload, and MI with non-obstructive coronary arteries, particularly coronary vasospasm.

The patient was admitted to a monitored unit and treated with anticoagulation and dual antiplatelet therapy. Intravenous antihypertensive treatment was initiated with labetalol (50 mg bolus followed by continuous infusion at 4 mg/min) and nitroglycerin starting at 5 µg/min, with dose titration every 5 minutes, achieving a target level of 160/100 mmHg before coronarography, corresponding to an initial 20% reduction in mean arterial pressure.

Laboratory evaluation showed a mildly elevated high-sensitivity troponin level of 39 ng/L (reference range: 2-34 ng/L), with all other routine laboratory parameters within normal limits (Table 1). Serial measurements at 1 and 3 hours demonstrated stable values (42 ng/L and 44 ng/L, respectively), without a significant rise or fall. A repeat ECG performed 3 hours later demonstrated T-wave normalization in the septal and apical leads, with persistent flattening in the lateral leads (Figure 1). Transthoracic echocardiography revealed a non-hypertrophied left ventricle with preserved global and regional systolic function and a left ventricular ejection fraction of 65%.

Parameter	Result	Reference range
Hemoglobin (g/dL)	14.2	13.0-17.0
White blood cell count (×10 ⁹ /L)	7.5	4.0-10.0
Platelet count (×10 ⁹ /L)	250	150-400
Serum creatinine (mg/dL)	0.9	0.7-1.3
Potassium (mmol/L)	4.2	3.5-5.0
Sodium (mmol/L)	139	135-145
Fasting glucose (mg/dL)	108	70-110
High-sensitivity troponin (ng/L)	39	2-34

TABLE 1: Laboratory findings on admission
High-sensitivity troponin is a cardiac biomarker used to detect myocardial injury. All other laboratory parameters were within normal limits.

Coronary angiography was subsequently performed and demonstrated normal coronary arteries (Figure 2). In the absence of obstructive coronary disease, with preserved ventricular function and modest troponin elevation in the context of severe hypertension, a diagnosis of acute myocardial injury secondary to hypertensive emergency was established. Antithrombotic therapy was therefore discontinued.

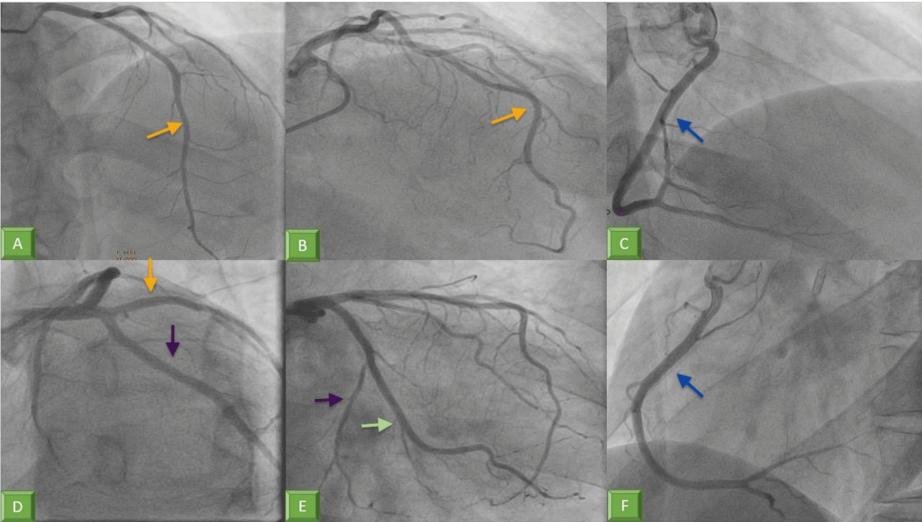


FIGURE 2: Coronary angiography
Panel A: Cranial frontal view demonstrating a lesion-free left anterior descending artery. Panel B: Cranial right anterior oblique projection showing a smooth, stenosis-free course of the left anterior descending artery. Panel C: Right anterior oblique view revealing a right coronary artery without any detectable lesions. Panel D: Caudal left anterior oblique view showing no evidence of occlusion in the left anterior descending (upper arrow) or circumflex arteries (lower arrow). Panel E: Caudal right anterior oblique projection illustrating the absence of stenosis in the circumflex (upper arrow) and marginal arteries (lower arrow). Panel F: Left anterior oblique view confirming a patent right coronary artery and its bifurcations, without signs of thrombosis.

Intravenous antihypertensive therapy was maintained for approximately 18 hours until sustained BP control was achieved (157/97 mmHg), after which the patient was transitioned to oral antihypertensive treatment. The patient was discharged the following day on amlodipine 10 mg and ramipril 10 mg once daily, with a BP of 127/73 mmHg. Given the patient’s relatively young age and severe presentation, secondary causes of hypertension were investigated. Screening for primary aldosteronism, including plasma aldosterone concentration and aldosterone-to-renin ratio, was unremarkable. Evaluation for pheochromocytoma with plasma and urinary metanephrines was negative. In addition, renal artery imaging showed no evidence of renal artery stenosis. At one-month follow-up, he remained asymptomatic, with well-controlled BP on the same dual therapy, and normalization of electrocardiographic findings, including positive T-waves in the

anterior and lateral leads (Figure 3).

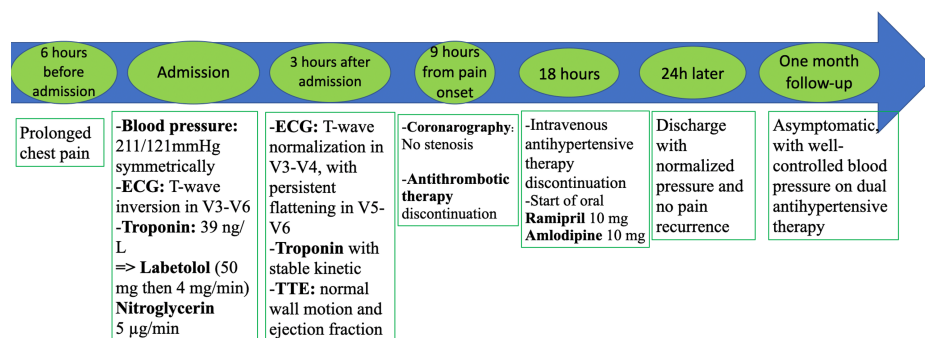


FIGURE 3: Timeline

This timeline table summarizes the entire case for a quick reference of events.

Discussion

This case describes a hypertensive emergency presenting with features suggestive of non-ST-elevation ACS, highlighting a common diagnostic and therapeutic challenge in acute cardiovascular care. Despite the modest troponin elevation typically observed in hypertensive crises, the persistence of chest pain associated with ischemic electrocardiographic changes and the patient's cardiovascular risk profile prompted the performance of coronary angiography.

Angina may occur in the absence of clinically or angiographically evident coronary artery disease in the setting of severe hypertension [5]. A sudden increase in BP leads to a marked rise in afterload and myocardial wall stress, resulting in increased myocardial oxygen demand. At the same time, elevated left ventricular end-diastolic pressure and reduced diastolic perfusion impair coronary blood flow. This imbalance between oxygen supply and demand can produce subendocardial ischemia even in the absence of obstructive coronary lesions [6].

In addition, endothelial dysfunction and microvascular injury play a central role [7]. Excessive shear stress damages the vascular endothelium and disrupts normal coronary autoregulation. This process promotes vasoconstriction, increased vascular permeability, and microthrombotic phenomena within the coronary microcirculation, contributing to myocardial injury and troponin release despite normal epicardial arteries [8].

Neurohormonal activation further exacerbates myocardial stress. Activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system increases systemic vasoconstriction, afterload, and myocardial oxygen consumption, thereby amplifying the mismatch between supply and demand [9].

In a hypertensive emergency, troponin elevation reflects acute myocardial injury related to increased wall stress and microvascular dysfunction rather than plaque rupture or coronary thrombosis. Electrocardiographic changes may be transient or nonspecific, and imaging typically shows preserved ventricular function without regional wall-motion abnormalities. In contrast, type 2 MI is defined by a supply-demand mismatch resulting in true ischemia. In the present case, the combination of chest pain and transient electrocardiographic changes in the absence of wall motion abnormalities supported the diagnosis of acute myocardial injury secondary to hypertensive emergency.

Current recommendations emphasize a cautious reduction in BP, with an initial decrease of no more than 20-25% within the first hour, followed by gradual normalization to prevent worsening end-organ ischemia [10]. In this patient, treatment with intravenous nitroglycerin and labetalol allowed effective BP control while reducing myocardial oxygen demand. However, secondary hypertension, although excluded in this patient, remains an important differential diagnosis. In addition, careful BP monitoring and appropriate long-term antihypertensive management are essential, as ongoing outpatient follow-up is required.

Conclusions

This case highlights the complexity of clinical decision-making in patients presenting with a hypertensive emergency and features suggestive of ACS. Severe hypertension may lead to acute myocardial injury that mimics MI, even in the presence of cardiovascular risk factors and ischemic electrocardiographic changes. Recognizing this presentation is essential to avoid misclassification and inappropriate management.

Early identification of myocardial injury related to hypertensive emergency allows for a more targeted therapeutic approach focused on controlled BP reduction. This strategy helps prevent unnecessary escalation of antithrombotic therapy and reduces the risk of procedure-related and iatrogenic complications. Increased awareness of this entity may improve diagnostic accuracy and optimize patient outcomes in acute care settings.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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