Concurrent Acute Ischemic Stroke and Acute Myocardial Infarction in a Patient with Hypertrophic Cardiomyopathy: A Multisystem Emergency

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Abstract

Background: The combination of ischemic stroke and myocardial infarction (MI) in patients with hypertrophic cardiomyopathy (HCM) is unusual and creates diagnostic and treatment difficulties.[1,2]

Case summary: We describe a 53-year-old woman, who was admitted with acute ischemic stroke, and later developed a non-ST elevation MI, followed by stent thrombosis and acute transmural MI. Echocardiography revealed left ventricular hypertrophy (LVH) with systolic anterior motion (SAM) of the mitral valve and dynamic outflow obstruction, consistent with HCM. Coronary angiography revealed significant Left Anterior Descending (LAD) stenosis, treated with a drug-eluting stent (DES). Recurrent thrombosis of this stent led to transmural MI and unstable hemodynamics, requiring urgent re-intervention.

Conclusion: In this patient, HCM-related outflow obstruction and structural changes contributed to both cerebral and myocardial ischemia. Balancing anticoagulation and antiplatelet therapy were particularly difficult. Early recognition of underlying HCM and close coordination between cardiology and neurology teams were essential.

მწვავე იშემიური ინსულტისა და მწვავე მიოკარდიუმის ინფარქტის ერთდროული მიმდინარეობა ჰიპერტროფიული კარდიომიოპათიის მქონე პაციენტში: მულტისისტემური გადაუდებელი შემთხვევა

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შესავალი: იშემიური ინსულტისა და მიოკარდიუმის ინფარქტის (MI) კომბინაცია ჰიპერტროფიული კარდიომიოპათიის (HCM) მქონე პაციენტებში იშვიათია და ქმნის დიაგნოსტიკურ და მკურნალობის სირთულეებს.

შემთხვევის აღწერა: 53 წლის ქალი, რომელსაც მწვავე იშემიური ინსულტის შემდეგ განუვითარდა მიოკარდიუმის ინფარქტი ST სეგმენტის ელევაციის გარეშე, სტენტის თრომბოზი და მწვავე ტრანსმურალური მიოკარდიუმის ინფარქტი. ექოკარდიოგრაფიამ გამოავლინა მარცხენა პარკუჭის ჰიპერტროფია (LVH) მიტრალური სარქვლის სისტოლური წინა მოძრაობით (SAM) და დინამიური გამოსასვლელი ტრაქტის ობსტრუქცია, რომელიც შეესაბამება HCM-ს. კორონაროანგიოგრაფიამ გამოავლინა მარცხენა წინა დაღმავალი (LAD) არტერიის მძიმე სტენოზი, რომელიც ნამკურნალები იყო წამლით დაფარული სტენტით (DES). ამ სტენტის რეციდიულმა თრომბოზმა გამოიწვია ტრანსმურალური მიოკარდიუმის ინფარქტი და ჰემოდინამიკური არასტაბილურობა, რაც საჭიროებდა სასწრაფო ხელახალ ჩარევას. დასკვნა: ამ პაციენტში, HCM-თან ასოცირებულმა გამოსასვლელი ტრაქტის ობსტრუქციამ და სტრუქტურულმა ცვლილებებმა ხელი შეუწყო როგორც ცერებრალური, ასევე მიოკარდიუმის იშემიის განვითარებას. განსაკუთრებით რთული იყო ოპტიმალური ანტიკოაგულანტური და ანტიაგრეგანტული თერაპიის შერჩევა. გადამწყვეტი მნიშვნელობა

ჰქონდა ძირითადი HCM-ის ადრეულ გამოვლენას და კარდიოლოგიისა და ნევროლოგიის გუნდებს შორის მჭიდრო კოორდინაციას.

საკვანძო სიტყვები: იშემიური ინფარქტი, იშემიური ინსულტი. ანტიკოაგულანტური და ანტიაგრეგანტული თერაპია

Clinical Perspective

• What is known?

Hypertrophic cardiomyopathy (HCM) can predispose to thromboembolic complications, including stroke and arrhythmias, but concurrent acute stroke and myocardial infarction is rare.[1,4]

• What does this case add?

This case highlights the potential for dynamic left ventricular outflow tract obstruction in HCM to contribute to both cerebral and myocardial ischemia, and underscores the challenge of managing anticoagulation in the setting of dual vascular events.[2,3,5]

• How might this impact clinical practice?

Clinicians should consider early cardiac evaluation in stroke patients with suggestive features, and anticipate the need for multidisciplinary management when HCM and concurrent vascular events are present. [4]

Introduction

Hypertrophic cardiomyopathy is an inherited disease in which the heart muscle, most often the septum, becomes abnormally thick. This may narrow the left ventricular outflow tract and change the way blood leaves the heart. Patients with HCM are more likely to develop arrhythmias, progressive heart failure, thromboembolic complications, and in some cases sudden cardiac death.[1]

While HCM is well recognized for causing arrhythmias and embolic stroke, the occurrence of both ischemic stroke and myocardial infarction in close succession is unusual. [1] When these events overlap, decisions around treatment become particularly complex. [2,3,4] This case demonstrates the need for early recognition of HCM when patients present with both neurological and cardiac ischemia. [4]

Case Presentation

T.K. is a 53-year-old woman who was admitted to our hospital on December 30, 2024. According to the patient, her symptoms began at 6:00 AM the same day. She reported weakness in her left limbs, difficulty speaking, and facial asymmetry. Her family contacted emergency services, and she was initially admitted to regional clinical center, before being transferred to our facility for further evaluation.

On admission, her vital signs were: heart rate 81 bpm, blood pressure 146/107 mmHg, respiratory rate 18/min, SpO_2 98%, and temperature 36.8°C. Neurological examination revealed that the patient was conscious, oriented, and interactive. She had decreased strength in the left limbs, dysarthria, and facial asymmetry, without pathological reflexes. Her skin and mucosa appeared normal. Pulmonary examination revealed weakened vesicular breath sounds. Cardiovascular exam was unremarkable.

Medical History:

• Essential (primary) hypertension for 20 years; peak BP of 200/100 mmHg. Medications include amlodipine and bisoprolol.
• Insulin-dependent diabetes mellitus managed with metformin; blood glucose levels periodically elevated.

• Family history: Both father and uncle died young from sudden cardiac death.

Initial investigations included laboratory tests and an ECG, which showed sinus rhythm without specific abnormalities. A brain MRI revealed a hyperintense lesion on DWI and hypointense on ADC in the caudal part of the right lentiform nucleus, consistent with acute ischemia. The patient was diagnosed with an acute ischemic stroke (ICD-10 I63.5) and admitted to the neurology department.

Neurological status initially showed an NIHSS score of 10. Symptomatic therapy and secondary stroke prevention were initiated. Her neurological condition gradually improved with NIHSS decreasing to 3, with only mild left-sided hemiparesis.

On January 3, 2025, she experienced an episode of tachycardia and sweating during a routine check-up. ECG showed ischemic T-wave inversions in leads I, aVL, and V2–V5, indicating subendocardial MI (Fig. 1). Cardiac biomarkers showed elevated High-sensitivity Troponin I (hs-TnI) - 668 ng/L. She was transferred to the cardiac ICU for further evaluation.

Echocardiography revealed marked left ventricular hypertrophy (LVH) in an S-shaped configuration, with systolic anterior motion (SAM) and left ventricular outflow tract (LVOT) obstruction. Systolic function was moderately reduced (EF - 42%) due to apical hypokinesis. Mild mitral regurgitation with eccentric flow was observed.

Coronary angiography revealed:

• LAD: Diffuse mid-segment damage with 80% stenosis; large diagonal branch 50% stenosis.

• Posterior Descending Artery: 60% stenosis.

• Left Main Artery, Right Coronary Artery and Circumflex Artery: No significant stenosis.

A drug-eluting stent was implanted in the LAD with good angiographic outcome (Fig. 2). She remained under anticoagulation - enoxaparin sodium 4000 IU (40 mg) OD, with aspirin (acetylsalicylic acid) 75 mg OD, and additional antiplatelet therapy (ticagrelor 90 mg BID), and neurologist supervision.

On January 8, the patient presented with severe chest pain radiating to the scapula, BP 250/120 mmHg, HR 130 bpm. ECG showed 10 mm ST elevation in precordial leads, confirming acute transmural MI (Fig. 3). hs-TnI was 4713 ng/L. Repeat angiography showed thrombotic occlusion of the LAD stent (subacute stent thrombosis).

Balloon dilatation and thrombo-aspiration were performed (Fig. 5), leading to partial recanalization and improved flow. Despite residual thrombus in the distal LAD, the final result was satisfactory (Fig. 4). The patient was transferred to the ICU in critical condition requiring norepinephrine and continuous heparin infusions. Neurologic deterioration was noted, with NIHSS score 6 and CT confirming a new ischemic lesion in the right basal ganglia (Fig. 7).

By January 10, vasoactive support was tapered and discontinued. Heparin infusion was stopped midway due to mild hematuria. ECG showed persistent 1 mm ST elevation (Fig. 6). Echocardiography demonstrated dynamic improvement. The patient's condition stabilized with ongoing cardiac and neurologic monitoring.

Timeline

Date	Clinical Event / Intervention

December 30, 2024	Onset of stroke symptoms; transferred from regional clinic; brain MRI confirmed acute ischemia; NIHSS 10.
January 1–2, 2025	Neurological improvement observed; NIHSS score decreased to 3.
January 3, 2025	Episode of tachycardia and sweating; NSTEMI diagnosed (hs-TnI 668 ng/L); cardiac ICU transfer; HCM with SAM and LVOT obstruction identified.
January 4, 2025	Coronary angiography showed LAD stenosis; drug-eluting stent implanted.
January 8, 2025	Severe chest pain; ECG confirmed transmural MI; subacute stent thrombosis detected; thrombo-aspiration performed.
January 9, 2025	Neurological deterioration with new ischemic lesion; NIHSS score 6.
January 10, 2025	Stabilization achieved; vasoactive support discontinued; heparin stopped due to mild hematuria.

Discussion

This case demonstrates how hypertrophic cardiomyopathy can predispose to both cerebral and cardiac ischemia. The structural abnormalities of HCM - including septal hypertrophy, outflow obstruction, and atrial enlargement - promote thrombus formation and impair coronary blood flow.[1] In our patient, the combination of underlying LAD disease and the additional burden of microvascular dysfunction related to HCM likely contributed to myocardial injury.[1,2]

A major complication was stent thrombosis despite antiplatelet therapy.[2,3] Managing stroke and myocardial infarction together required balancing anticoagulation, dual antiplatelet therapy, and blood pressure targets, while responding quickly to bleeding events such as hematuria. [2,3,5]

This experience emphasizes that when stroke patients show features suggestive of structural heart disease, early echocardiographic evaluation is warranted. [1,4] A multidisciplinary approach - involving neurologists, cardiologists, and intensive care physicians - is necessary when HCM coexists with acute vascular events. [1,2,4]

Conclusion

This patient's presentation shows how hypertrophic cardiomyopathy can contribute to both acute stroke and myocardial infarction within days of each other.[1] The presence of outflow obstruction and ventricular hypertrophy complicated both diagnosis and management.[1,2] Careful coordination across specialties and close monitoring of antithrombotic therapy were key to stabilizing the patient. [3,4,5]

Clinical Implication: Clinicians should maintain a high index of suspicion for underlying structural heart disease in patients presenting with simultaneous neurologic and cardiac ischemia.

Figure Legends

Figure 1. Electrocardiogram taken on January 3, 2025, showing deep ischemic T-wave inversions in leads I, aVL, and V2–V5, suggestive of subendocardial MI

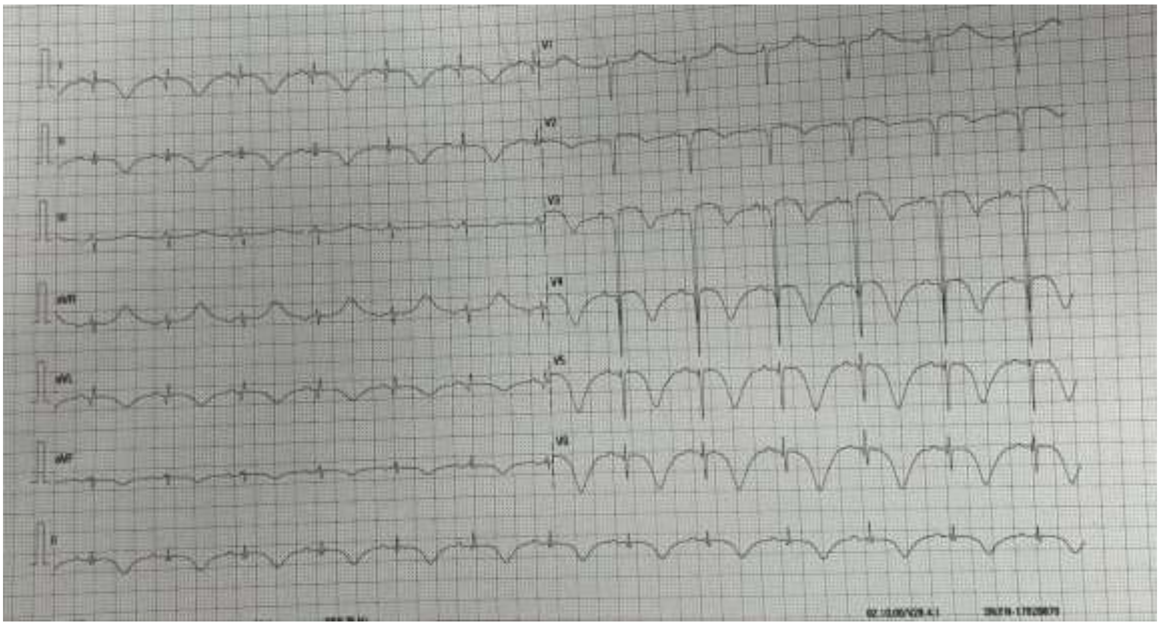

Figure 2. Coronary angiography on January 4, 2025. Mid-segment LAD stenosis before and after stent placement

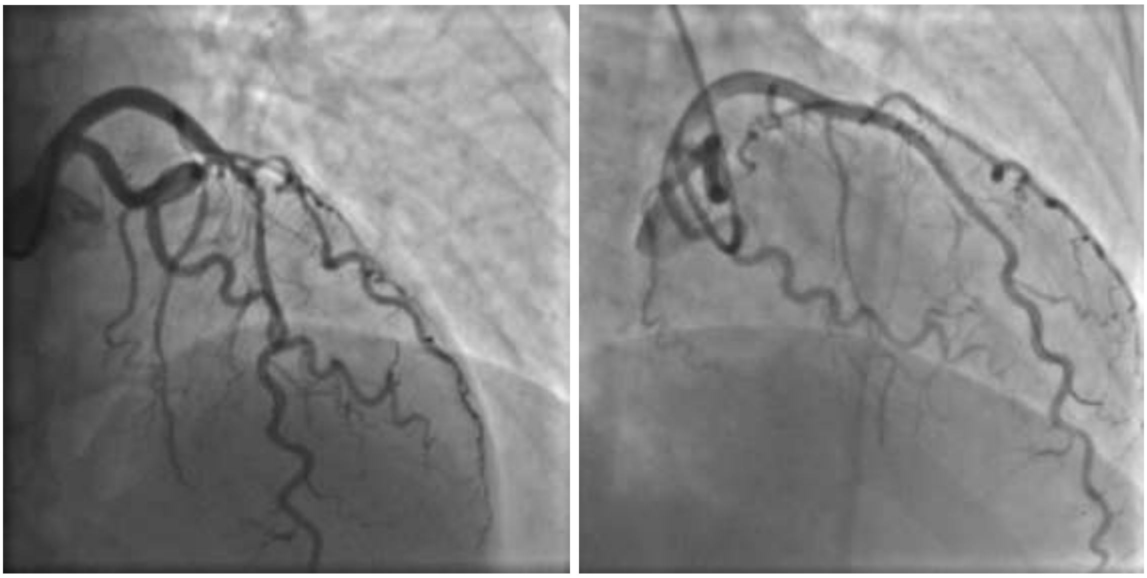

Figure 3. Electrocardiogram taken on January 8, 2025, showing ST segment elevations in precordial leads suggestive of acute transmural MI of anterior wall

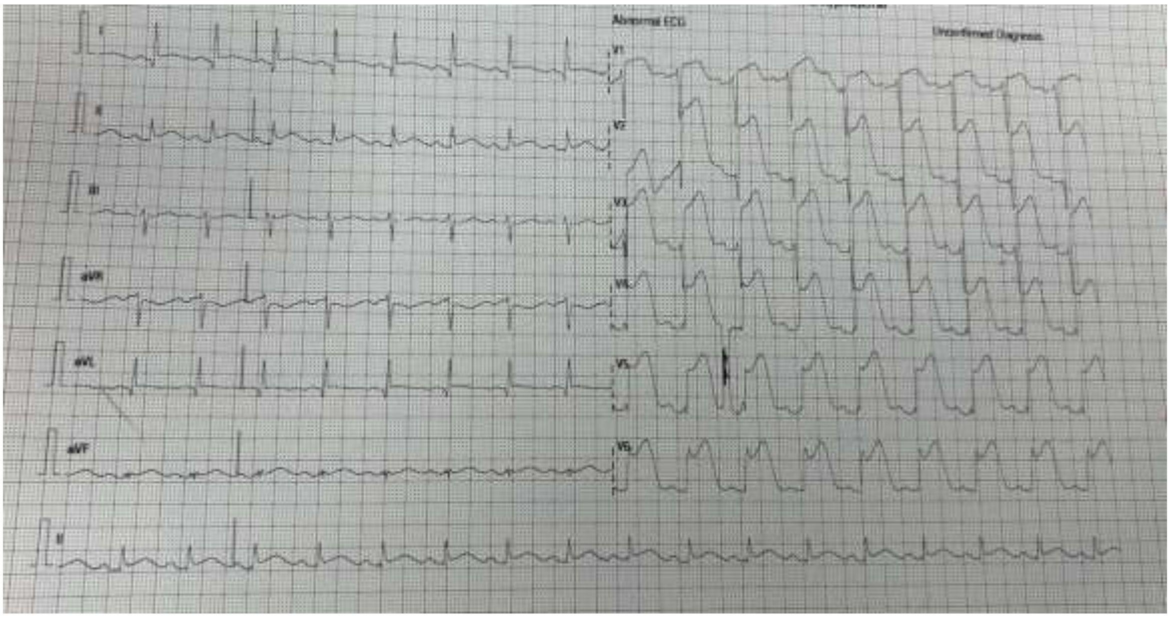

Figure 4. Repeat coronary angiography on January 8, 2025. Occluded LAD stent (subacute stent thrombosis) and post-thromboaspiration images showing partial recanalization with residual thrombus

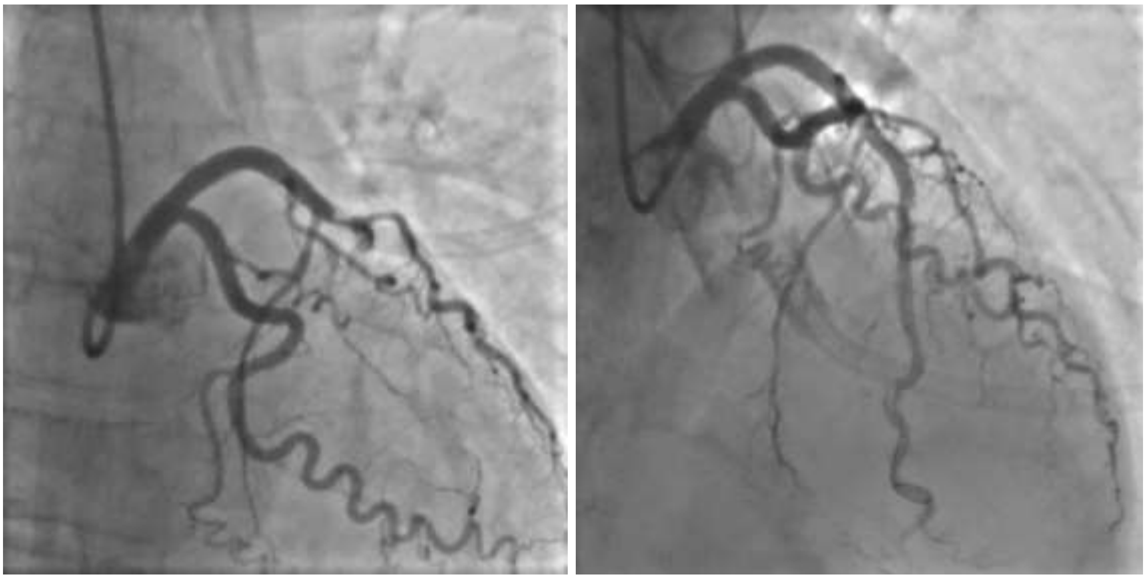

Figure 5. Thrombus retrieved during second coronary angiography on January 8, 2025

Figure 6. Electrocardiogram taken on January 8, 2025, 8 hours after stent implantation, showing persistent ST elevations precordial leads

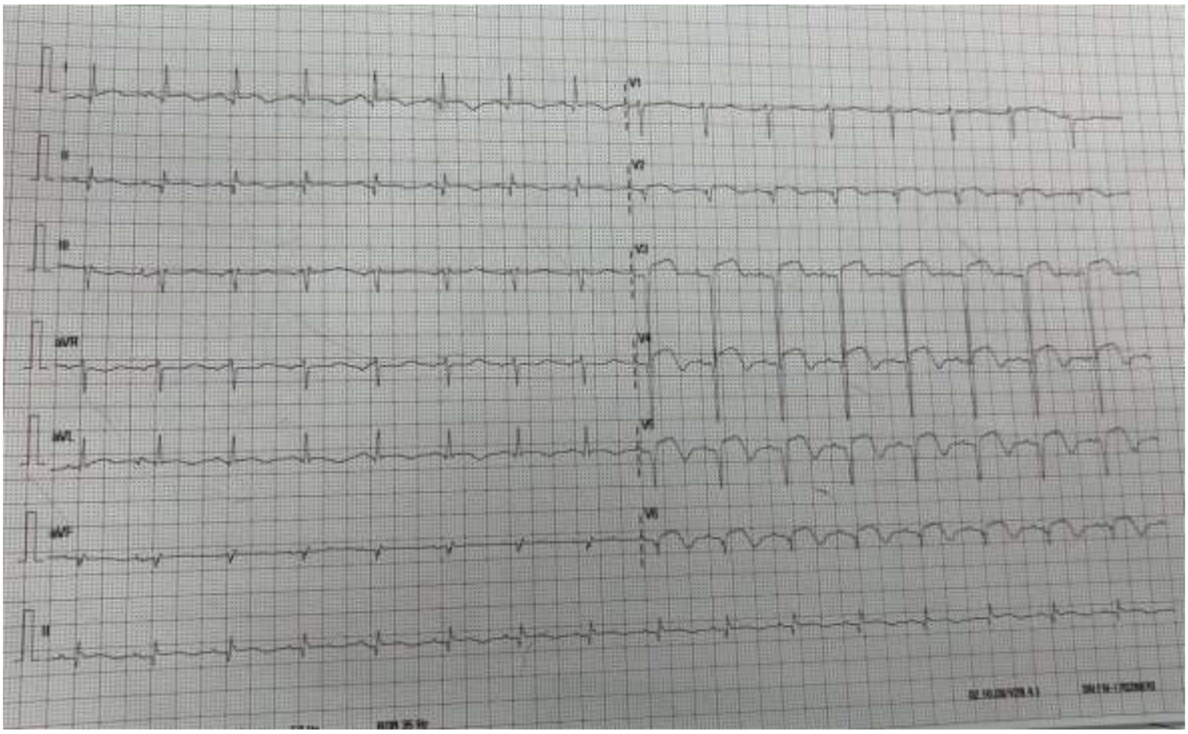

Figure 7. Follow-up brain CT scan on January 9, 2025. New ischemic lesion in the right basal ganglia near the lateral ventricle

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