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A Rare Association of Hypertrophic Cardiomyopathy and Infective Endocarditis: Case Report and Literature Review

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Abstract **Case Report**

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disorder, affecting approximately 1 in 500 individuals. Although patients with structural heart disease are considered at increased risk of infective endocarditis (IE), its occurrence in HCM remains exceedingly rare. We report the case of an 84-year-old woman with known HCM who presented with progressive dyspnea, fever, and a new systolic murmur. Transthoracic echocardiography demonstrated a mobile vegetation measuring 10 × 10.3 mm attached to the aortic valve, with preserved left ventricular ejection fraction. Blood cultures were positive for Streptococcus viridans, confirming the diagnosis of IE. The patient was managed with intravenous antibiotic therapy, but her subsequent clinical course could not be fully documented. This case highlights the uncommon occurrence of IE in patients with HCM and underscores the pivotal role of echocardiography and microbiological confirmation in establishing the diagnosis. A brief review of the literature shows that IE in HCM is associated with significant morbidity and mortality, with the mitral valve being most frequently affected, but the aortic valve can also be involved. Our observation adds to the limited number of published cases and emphasizes the importance of early recognition and multidisciplinary evaluation in this challenging clinical scenario.

Keywords: Hypertrophic cardiomyopathy - Infective endocarditis - Aortic valve vegetation - Echocardiography - Cardiac infection - Multidisciplinary management.

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disease, with an estimated prevalence of 1 in 500 individuals worldwide [1]. It is characterized by asymmetric left ventricular hypertrophy, dynamic left ventricular outflow tract obstruction (LVOTO), and diastolic dysfunction. Although HCM has been widely studied in terms of arrhythmias, sudden cardiac death, and heart failure, less attention has been given to its potential association with infective endocarditis (IE).

IE is a life-threatening condition resulting from microbial infection of the endocardial surface, usually involving cardiac valves. The incidence of IE in the general population is approximately 5–7 cases per 100,000 person-years [2] [3]. However, studies suggest that patients with HCM may have an 18- to 28-fold higher risk of developing IE compared with the general population [4]. Predisposing factors include LVOTO, elongated mitral valve leaflets, and left atrial enlargement, which increase the likelihood of endothelial disruption and bacterial colonization [5] [6].

Despite improvements in diagnostic imaging and antibiotic therapy, the prognosis of IE complicating HCM remains poor, with in-hospital and one-year mortality rates reported between 30% and 40% [5]. Streptococcus species, particularly of oral origin, are the most frequently isolated pathogens [5] [6]. Current international guidelines no longer recommend routine antibiotic prophylaxis for HCM, unlike for other high-risk conditions, although this remains a matter of ongoing debate [7] [2].

CASE PRESENTATION

An 84-year-old woman with a known history of hypertrophic cardiomyopathy (HCM) was admitted to our cardiology department for evaluation of progressive shortness of breath, fever, and general fatigue persisting for two weeks. She denied chest pain, syncope, or neurological symptoms.

On admission, the patient was febrile (38.3 °C), with a heart rate of 96 beats per minute and blood pressure of 125/52 mmHg. A new systolic murmur was audible at the aortic area with radiation to the carotids. Pulmonary auscultation revealed bilateral basal crackles. No peripheral stigmata of infective endocarditis, such as Janeway lesions, Osler's nodes, or Roth spots, were observed.

The 12-lead electrocardiogram on admission showed sinus rhythm at 94 beats per minute, with voltage criteria for left ventricular hypertrophy and repolarization abnormalities (flattened T waves in lead aVL). No conduction disturbances, Q waves, or arrhythmias were noted **(Figure 1**).

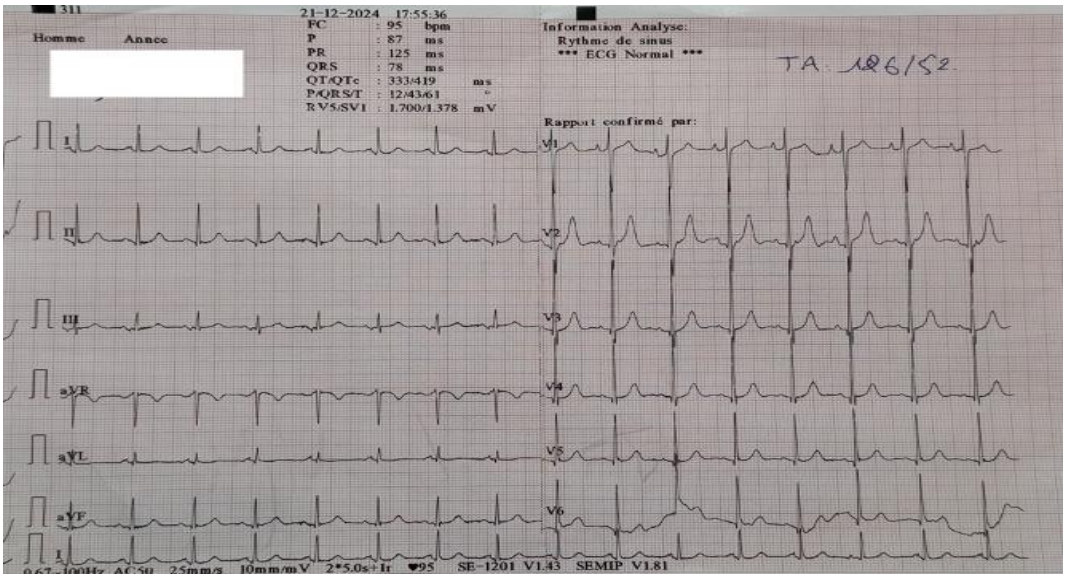

Figure 1: Standard 12-lead electrocardiogram on admission showing sinus rhythm with signs of left ventricular hypertrophy and repolarization abnormalities (flattened T wave in lead aVL)

Transthoracic echocardiography (TTE)

Transthoracic echocardiography showed a non-dilated aortic root and a non-dilated left ventricle (end-diastolic diameter 34 mm, indexed 24 mm/m²). The left ventricle exhibited concentric hypertrophy, with an indexed LV mass of 104 g/m² and a relative wall thickness of 0.74. Septal and posterior wall thicknesses were 17 mm and 16 mm, respectively, Global systolic function was preserved, with a left ventricular ejection fraction of 69% as measured by Simpson's biplane method.

The left atrium was dilated (area 25 cm², volume 78 mL, indexed 55 mL/m²), while the right atrium was not dilated (area 11 cm²). The right ventricle was slightly enlarged (RV/LV ratio 0.8) but with preserved systolic function (TAPSE 20 mm, S′ 11 cm/s).

The mitral valve was thin and mobile, without stenosis or regurgitation. The aortic valve was tricuspid and mildly calcified, without baseline stenosis or regurgitation. Importantly, a mobile echogenic mass measuring 10 × 10.3 mm was visualized, consistent with a vegetation attached to the ventricular side of the aortic valve (Figure 2).

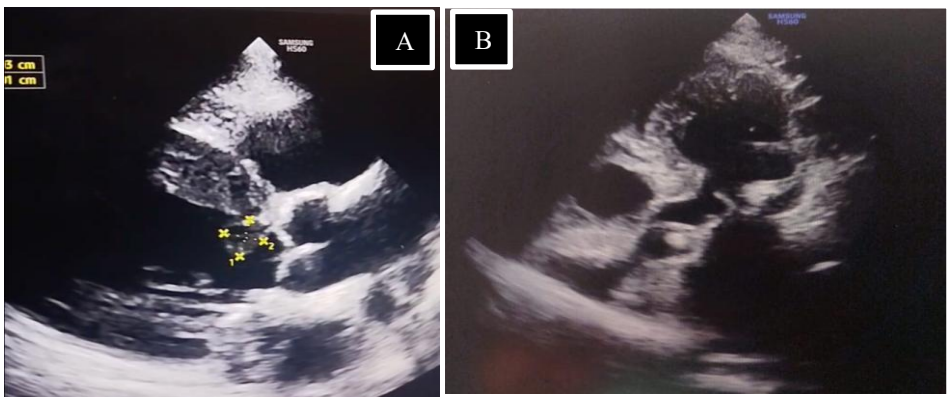

Figure 2: Transthoracic echocardiography showing vegetations attached to the aortic valve. (A) Parasternal long-axis view demonstrating a mobile echogenic vegetation measuring approximately 10 × 10.3 mm (yellow markers). (B) Apical 5-chamber view confirming the presence of the vegetation on the aortic valve

Doppler analysis revealed impaired relaxation with elevated LV filling pressures (E/A ratio 0.75; E wave 116 cm/s; septal e′ 6.7 cm/s; lateral e′ 10.1 cm/s; average E/e′ 6.8). The estimated pulmonary artery systolic pressure was 40 mmHg, assuming a right atrial pressure of 3 mmHg, with a tricuspid regurgitant jet velocity of 3 m/s. The inferior vena cava was not dilated and remained compliant. No pericardial effusion was detected.

Laboratory findings on admission

showed hemoglobin at 12.0 g/dL, mean corpuscular volume 88.7 fL, and platelet count 165,000/µL. White blood cell count was 8,190/µL, with neutrophils 6,880/µL. Inflammatory markers were markedly elevated, with C-reactive protein at 334 mg/L. Renal function was preserved (serum creatinine 7.8 mg/L, estimated GFR 75 mL/min/1.73 m²), while liver enzymes were mildly elevated (AST 58 U/L, ALT 57 U/L, GGT 66 U/L). Serum sodium and potassium were within normal ranges (Na^+ 142 mmol/L, K^+ 3.8 mmol/L). Cardiac biomarkers showed troponin at 30 ng/L (five times the upper normal limit) and markedly increased D-dimer levels (2,731 ng/mL).

Blood cultures were positive for Streptococcus viridans, confirming the diagnosis of infective endocarditis. Intravenous antibiotic therapy was initiated according to international guidelines and adapted to the sensitivity profile.

Surgical intervention was not considered due to advanced age and frailty.

Discussion

Infective endocarditis (IE) is a rare but serious complication in patients with hypertrophic cardiomyopathy (HCM). While the prevalence of HCM is estimated at 1 in 500 individuals [1], only a limited number of cases of IE have been described in this population [5] [4] [6]. The risk of IE in HCM patients has been reported to be 18- to 28-fold higher than in the general population [4], most likely due to turbulent blood flow, systolic anterior motion of the mitral valve, and left ventricular outflow tract obstruction, which predispose to endothelial damage and microbial colonization.

In the largest multicenter prospective cohort to date, Domínguez et al. identified 34 cases of IE in HCM across 27 Spanish hospitals [5]. The majority of cases involved the mitral valve (71%), while the aortic valve was less commonly affected (35%). Streptococcus species, particularly viridans group streptococci, were the most frequently isolated pathogens. Our case is consistent with these findings, as the patient developed native aortic valve endocarditis with Streptococcus viridans. The clinical outcome of IE in HCM is often poor. In the GAMES cohort, in-hospital mortality reached 32%, and one-year mortality approached 41% [5]. Similarly, historical series and recent reports confirm that morbidity and mortality remain high, especially in elderly patients and those with advanced heart failure. In our case, although surgical intervention was not considered due to advanced age and frailty, the patient responded initially to medical therapy. This conservative approach is also reflected in other case series, where the anatomical complexity of HCM and the hemodynamic consequences of LVOTO complicate surgical decision-making.

The question of antibiotic prophylaxis remains debated. Current European guidelines classify HCM as an intermediate-risk condition and do not recommend routine antibiotic prophylaxis, unlike for prosthetic valves or previous endocarditis [7]. However, given the increased risk of IE reported in HCM and the strong association with oral streptococci, some authors suggest reconsidering targeted prophylaxis in selected patients [5]

This case therefore illustrates several key points. First, it reinforces that IE can complicate HCM even in the absence of significant outflow tract obstruction. Second, it highlights the diagnostic value of echocardiography, which remains central to case detection and follow-up. Finally, it adds to the limited body of literature describing IE on the aortic valve in patients with HCM, an association that is less common than mitral involvement but well documented in prospective cohorts.

Conclusion

Infective endocarditis is an uncommon but serious complication of hypertrophic cardiomyopathy. Although the mitral valve is most frequently affected, our case illustrates that the aortic valve can also be involved. Echocardiography remains the cornerstone for diagnosis, and microbiological confirmation provides essential guidance for therapy. This report adds to the limited literature on this rare association and emphasizes the importance of maintaining a high index of suspicion for endocarditis in patients with HCM presenting with fever and new murmurs, particularly in elderly populations.

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