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When A Murmur Changes Meaning: Infective Endocarditis in Obstructive Hypertrophic Cardiomyopathy

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Abstract

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disorder, affecting approximately 1 in 500 individuals. Infective endocarditis (IE) is an uncommon but potentially fatal complication in patients affected by hypertrophic cardiomyopathy (HCM). The risk has been described to be significantly higher than in the general population, but the incidence of IE in HCM population remains unknown. The complex pathophysiology of this disease, characterized by structural alterations of the mitral valve apparatus and the presence of turbulent flow that promotes the deposition of microorganisms, could provide a substrate for IE and may, to some extent, explain its higher incidence in this specific population. We report the case of a 54-year-old man with known HCM who presented with progressive dyspnea, irregular fever and weight loss for 3 months. Transesophageal echocardiography demonstrated a mobile vegetation measuring 11 × 4.7 mm attached to the mitral valve, with preserved left ventricular ejection fraction. As patient got multiple antibiotics prior admission, blood culture failed to reveal growth of any organism. He was diagnosed as a case of infective endocarditis on the basis of modifies Duke's criteria. The patient was managed with intravenous antibiotic therapy. This case highlights the uncommon occurrence of IE in patients with HCM and underscores the pivotal role of transesophageal echocardiography in establishing the diagnosis. 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.

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Introduction

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disease, with an estimated prevalence of 1 in 500 individuals worldwide. 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) [1].

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 [2].

Case report: A 54-year-old shopkeeper with a known history of hypertrophic cardiomyopathy (HCM) was admitted to our medicine department for evaluation of progressive shortness of breath, irregular fever, and general fatigue persisting for three months. He denied chest pain, palpitation, leg swelling, chest pain, syncope, or neurological symptoms. Fever was low grade intermittent, highest recorded temperature was 101˚ F. He also lost about 6 kg weight in the given period with reduced appetite. He had history of taking ceftriaxone, levofloxacin on admission, the patient was febrile (100.3 °F), with a heart rate of 90 beats per minute and blood pressure of 140/92 mmHg. A pan systolic murmur was audible at the mitral area with radiation to the left axilla. Pulmonary auscultation revealed bilateral basal fine late inspiratory crackles. Edema was absent, jugular venous pressure was not raised. 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 60 beats per minute, with voltage criteria for left ventricular hypertrophy and repolarization abnormalities (inverted T waves in lead aVL). No conduction disturbances, Q waves, or arrhythmias were noted (Figure 1).

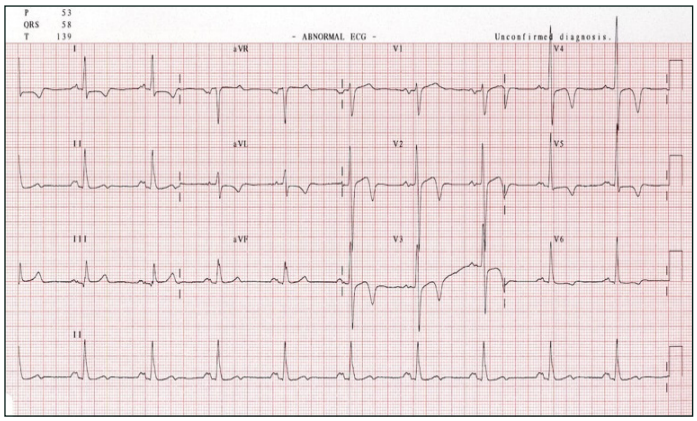
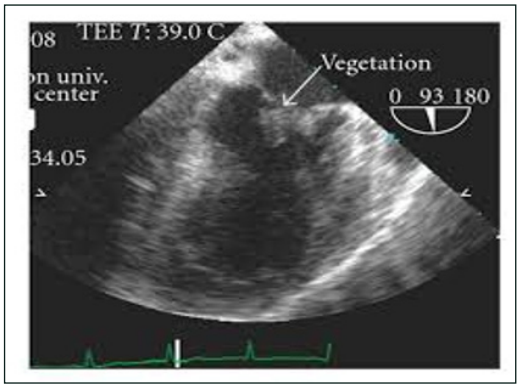


Figure 1: 12 lead ECG Showing Left Ventricular Hypertrophy and Repolarization Abnormalities

Laboratory findings on admission showed hemoglobin at 9.0 g/dL, mean corpuscular volume 89.7 fL, and platelet count 155,000/µL. White blood cell count was 6290/µL, with neutrophils 71%. Inflammatory markers were markedly elevated, with C-reactive protein at 330 mg/L. Renal function was preserved (serum creatinine 0.8 mg/L, estimated GFR 75 mL/min/1.73 m²). Serum sodium and potassium were within normal ranges (Na^+ 140 mmol/L, K^+ 3.9 mmol/L. Urine routine examination showed protein (++), RBC 30-40/HPF with granular casts. Blood cultures failed to reveal growth of any organisms.

Transthoracic echocardiography showed a non-dilated aortic root and a non-dilated left ventricle (end-diastolic diameter 32 mm, indexed 22 mm/m²). The left ventricle exhibited concentric hypertrophy, with an indexed LV mass of 94 g/m² and a relative wall thickness of 0.70. Septal and posterior wall thicknesses were 17 mm and 16 mm, respectively, Global systolic function was preserved, with a left ventricular ejection

fraction of 65% as measured by Simpson's biplane method. Mitral regurgitation was present. There was suspected vegetation on anterior mitral valve leaflet resulting in mild to moderate regurgitation. So transesophageal echocardiography was planned which revealed a mobile, partially filamentous vegetation (11 × 4.7 mm mm) attached to the anterior mitral leaflet, associated with moderate mitral regurgitation (effective regurgitant orifice area 0.3 cm2, regurgitant volume 32 mL) and a central regurgitant jet. (Figure 2 and 3).



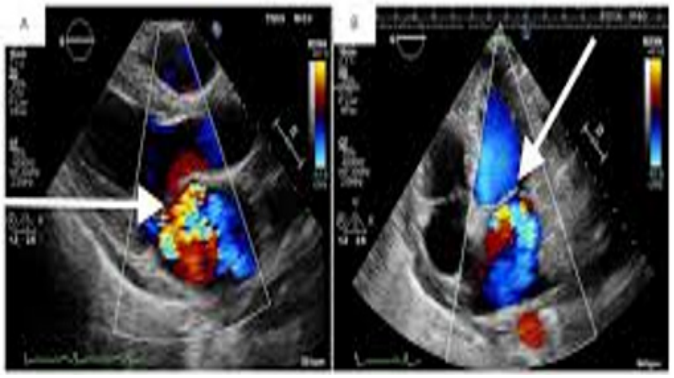


Figure 2 and Figure 3: Transesophageal Echocardiography (TEE) Revealed Vegetation on Anterior Mitral Valve Leaflet and Doppler TEE showed Mitral Regurgitation

The patient was started meropenem, vancomycin and amikacin. With treatment he showed dramatic clinical improvement with improving wellbeing, subsidence of fever and shortness of breath from the 5th day of admission. CRP came down to 58.1 mg/dl on 7th day. Antibiotic was continued for 14 days during hospital stay. As per patients request he was discharged after 2 weeks but advised to continue meropenem for 28 days. On out patient door follow up after 1 month, he was afebrile. There was no shortness of breath. CRP was 9.8 mg/dl, urine routine examination revealed trace proteinuria with no RBC and casts.

After 6 weeks, TEE documented resolution of the vegetation, leading to the choice to avoid surgery. Mitral regurgitation was moderate, with 2 different jets directed posteriorly and laterally; these were the result of the deterioration of the anterior leaflet and the concomitant SAM-related mitral regurgitation. The LVOT gradient remained comparable with the previous examination, which can be explained by the main mechanism of these phenomena linked to the SAM of the anterior mitral leaflet and of the lengthened chordae rather than the presence of the vegetation.

Discussion

Infective endocarditis (IE) is an uncommon but potentially fatal complication in patients affected by HCM. The risk for IE has been described to be significantly higher than in general population, but the incidence of IE in HCM remains unknown, as available information is confined to case reports and very small case series [4,6]. The risk of IE in HCM patients has been reported to be 18- to 28-fold higher than in the general population, 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 [4].

Initially, Spirito et al speculated that OHCM with LVOT obstruction was more likely to cause IE. This higher risk may be related to the damage of valve endocardium due to the systolic mitral septal contact and the high-velocity turbulence of blood flow during ejection. Moreover, patients with HCM usually have elongated mitral leaflets that favor premature erosion and the settlement of microorganisms. Vegetations typically attaching mainly to left-sided valve leaflets support this hypothesis [5].

Streptococcus species, prevalent in the oral cavity, are commonly implicated organisms. IE in HCM manifests mainly as worsening heart failure due to valvular dysfunction, although severe valve incompetence is not always present [4].

Thus, it is possible that persistent sepsis is more common among patients with IE and HCM and treatment typically involves antibiotics and sometimes surgery, though managing surgical complications is challenging because of the heterogeneous cardiac anatomy and labile hemodynamic status in HCM [7]. Literature

assessing surgical outcomes is presently limited to case reports and small case series. Consistent to the finding of published case reports and case series, the overall mortality rate is 22% [5].

The possible cardiac complications include: Heart failure due to valvular insufficiency; Acute myocardial infarction due to coronary artery occlusion; Myocardial abscess may occasionally rupture leading to suppurative pericarditis. Myocarditis caused by infection and inflammation involving the heart muscle.

In the largest multicenter prospective cohort to date, Domínguez et al. identified 34 cases of IE in HCM across 27 Spanish hospitals. 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%. 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 [5].

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. However, prevention measures are strongly encouraged in these patients. Given the highlighted morbidity of IE in HCM and the relevance of oral sources of bacteremia, it seems appropriate to reconsider the real balance between the benefits and risks of IE antibiotic prophylaxis. Large-scale retrospective and prospective studies are urgently needed to further evaluate predisposing factors and provide evidence-based recommendations [8].

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 prolonged fever, dyspnea and new murmurs, particularly in middle aged/elderly populations.

Conflict of Interest: None declared

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