VALVULAR HEART DISEASE

CLINICAL CASE SERIES

Endocarditis and Hypertrophic Cardiomyopathy



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ABSTRACT

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. The purpose of this case series is to highlight the correlation between endocarditis and HCM, a concern that has also been raised in recent European guidelines. (JACC Case Rep. 2025;30:103087) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

CASE 1: MEDICAL MANAGEMENT OF ENDOCARDITIS IN HYPERTROPHIC CARDIOMYOPATHY

A 55-year-old man presented with low-grade fever, shortness of breath, and weight loss. Obstructive hypertrophic cardiomyopathy (oHCM) was diagnosed 12 years before and managed with an oral beta-blocker.

Transthoracic echocardiography revealed diffuse left ventricular wall thickening and significant

TAKE-HOME MESSAGE

- IE is a rare but serious complication in patients with HCM, with a potentially higher risk because of factors including LVOT obstruction and mitral leaflet abnormalities.
- Further research is needed to better understand risk factors and reassess the balance between the benefits and risks of prophylaxis.

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ABBREVIATIONS AND ACRONYMS

HCM = hypertrophic cardiomyopathy

LVOT = left ventricular outflow tract

oHCM = obstructive hypertrophic cardiomyopathy

SAM = systolic anterior motion

TEE = transesophageal echocardiography

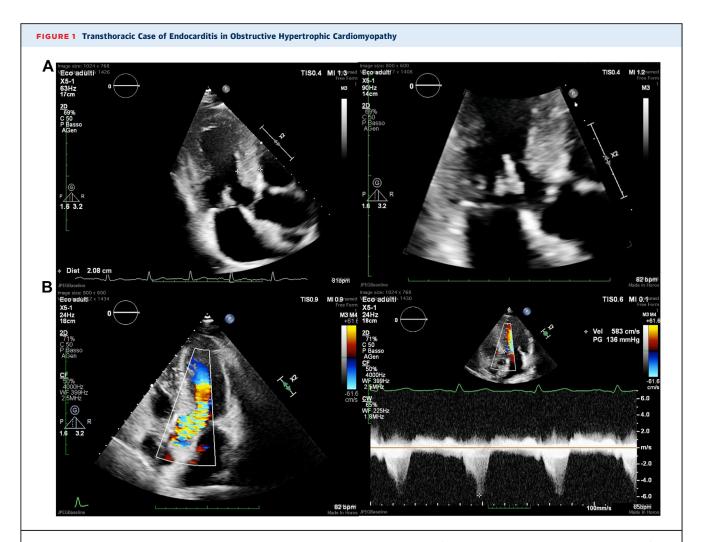
hypertrophy of the interventricular septum (20 mm), with a single vegetation attached to the anterior mitral valve leaflet (Figure 1A), resulting in mild regurgitation (effective regurgitant orifice area 0.2 cm, regurgitant volume 28 mL).

Systolic anterior motion (SAM) of the mitral valve resulted in severe left ventricular outflow tract (LVOT) obstruction, with a resting peak gradient of 153 mm Hg (Figure 1B).

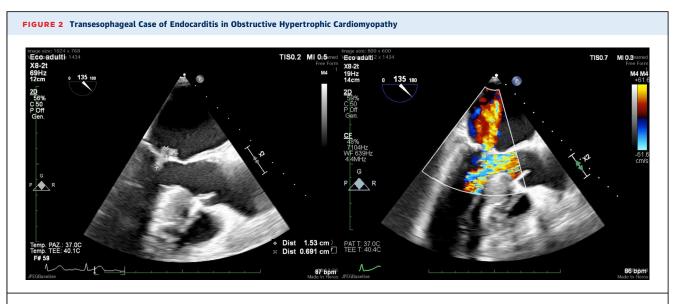
Transesophageal echocardiography (TEE) confirmed a mobile, partially filamentous vegetation (16×4 mm) attached to the anterior mitral leaflet, associated with moderate mitral regurgitation

(effective regurgitant orifice area 0.3 cm², regurgitant volume 32 mL) and a central regurgitant jet (Figure 2, Video 1).

Gram-positive *Streptococcus mitis* grew in 2 sets of blood cultures, and the patient was administered antibiotic therapy with piperacillin/tazobactam and daptomycin. After 6 weeks, TEE documented resolution of the vegetation, leading to the choice to avoid surgery (Video 2). 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



(A) Transthoracic apical 4-chamber view showing severe interventricular septal hypertrophy (20.8 mm) (left) and a vegetation attached to the anterior mitral leaflet (right). (B) Systolic aliasing confirming the diagnosis of hypertrophic cardiomyopathy with systolic anterior motion-related outflow tract obstruction (resting peak gradient 135 mm Hg).



Midesophageal long-axis view showing a vegetation measuring 15.3×6.9 mm attached to the anterior mitral valve leaflet (left), resulting in moderate regurgitation with combined systolic aliasing by left ventricular outflow tract obstruction (right).

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.

CASE 2: SEVERE ENDOCARDITIS IN HYPERTROPHIC CARDIOMYOPATHY

An 83-year-old woman with oHCM diagnosed 4 years earlier was admitted with fever and worsening of general condition.

Transthoracic echocardiography showed LVOT obstruction with a severe peak gradient of 160 mm Hg due to SAM of the anterior leaflet, severe mitral regurgitation, and a moderate mitral stenosis (mean gradient 10 mm Hg, area 1.5 cm²) (Figure 3B). A voluminous mass was reported on the posterior mitral leaflet, highly suspicious for endocardial vegetation (Figure 3A, left). Subsequently, TEE confirmed a voluminous, mobile, and partially calcified mass (10 \times 25 mm) on the coaptation of the posterior mitral leaflet, generating double-jet mitral regurgitation with a perforation in the middle of the leaflet (Video 3).

Empirical antibiotics were started while awaiting blood culture results. A surgical approach was proposed, encompassing mitral valve replacement and septal myectomy, but the patient refused. Unfortunately, the severe septic shock caused by *Staphylococcus aureus* perpetuated and ultimately led to the patient's death.

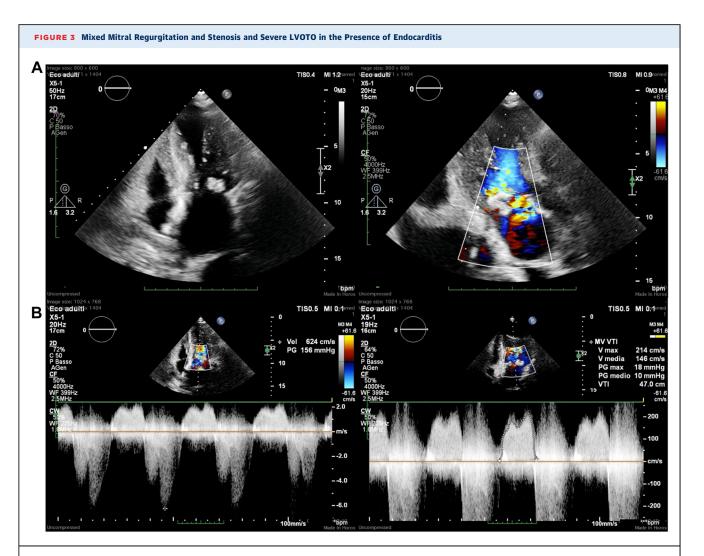
DISCUSSION

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. 1,2

Initially, Spirito et al¹ 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.³

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.¹

Thus, it is possible that persistent sepsis is more common among patients with IE and HCM,³ 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



(A) Transthoracic baseline evaluation, apical 4-chamber view (interventricular septal thickness 19 mm) with systolic anterior motion (left), and 3-chamber view showing moderate to severe mitral regurgitation (right). (B) Continuous-wave Doppler spectral curves showing mitral regurgitation and dagger-shaped intraventricular acceleration with a peak gradient of 156 mm Hg (left) and moderate to severe mitral stenosis (mean gradient 10 mm Hg) resulting from the vegetation (right).

HCM.⁴ 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%.³

The 2023 European Society of Cardiology guidelines² classify patients with HCM as having intermediate risk for IE, recommending that IE antibiotic prophylaxis not be routinely administered and may be considered on an individual basis. However, prevention measures are strongly encouraged in these patients.⁵ 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.

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KEY WORDS endocarditis, HOCM, LVOTO, mitral valve

APPENDIX For supplemental videos, please see the online version of this paper.