

Case Report

Hypertrophic Cardiomyopathy in a Patient with Subaortic Membrane

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Citation: Ghaderian H, Asl-Fallah S, Mansourian S, Hosseinsabet A. Hypertrophic Cardiomyopathy in a Patient with Subaortic Membrane. Res Heart Yield Transl Med 2026; 21(3):249-252.

 <https://doi.org/10.18502/rhythm.v21i3.22568>

Highlights

- Asymmetric septal hypertrophy is an uncommon and common echocardiographic finding in subaortic membrane and hypertrophic cardiomyopathy, respectively.
- Cardiac magnetic resonance imaging is useful for distinguishing between these 2 conditions.

Article info:

Received: 26 Sep. 2025

Revised: 03 Oct. 2025

Accepted: 14 Oct. 2025

ABSTRACT

Left ventricular outflow tract obstruction is a presentation of subaortic membrane. Asymmetric septal hypertrophy is an uncommon echocardiographic finding in this condition. Nonetheless, it is a common presentation of hypertrophic cardiomyopathy. These 2 conditions can coexist in the same patient. We describe a woman with a significant obstructive subaortic membrane and asymmetric septal hypertrophy, with a final diagnosis of hypertrophic cardiomyopathy confirmed by cardiac magnetic resonance imaging.

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Keywords: Hypertrophic Cardiomyopathy; Subaortic Membrane; Echocardiography; Cardiac Magnetic Resonance Imaging (CMR)

Introduction

Left ventricular outflow tract (LVOT) obstruction has several etiologies.^{1,2} The presence of a congenital subaortic membrane and systolic anterior motion (SAM) of the mitral leaflet are prototypes of fixed and dynamic LVOT obstruction, respectively.³ Hypertrophic cardiomyopathy (HCM) has several echocardiographic features, including asymmetric septal hypertrophy (ASH) and dynamic LVOT obstruction.⁴ Nevertheless, these features are not specific to HCM and are seen in other conditions.^{5,6} Thus, there is an overlap in echocardiographic features among several conditions, and in some cases, 2 pathologies are involved in a single presenting feature. ASH can be a common finding in HCM and an uncommon finding in a subaortic membrane. Still, ASH can be a marker of the coexistence of these 2 conditions. We describe a woman with concomitant HCM and a subaortic membrane.

Case Presentation

A 56-year-old woman presented with exertional dyspnea, classified as New York Heart Association (NYHA) functional class II, and a systolic murmur at the left parasternal border on physical examination. There was no history of hypertension, drug use, or family history of HCM. Electrocardiography showed left axis deviation, increased QRS voltage and duration (120 ms), ST-segment depression in leads I, II, aVF, and aVL, as well as ST-segment elevation in leads V₁–V₃,

and T-wave inversion in leads I and aVL, all suggestive of LV hypertrophy.

Transthoracic echocardiography revealed that the LV had normal size and systolic function (ejection fraction=60%). ASH was present, with septal and inferolateral wall thicknesses measuring 19 mm and 9 mm, respectively (Figure 1A and Video 1). The right ventricle also appeared normal in size and systolic function. Additional findings included mild-to-moderate mitral insufficiency, mild tricuspid and aortic insufficiency, and no systolic anterior motion of the mitral leaflet. A subaortic membrane causing severe subaortic stenosis was identified, with a peak pressure gradient of 130 mm Hg (Figure 1B, Video 1). Transesophageal echocardiography verified the existence of the subaortic membrane, which was attached to the interventricular septum and anterior mitral leaflet, at a distance of 21 mm from the aortic valve. The septal side of the membrane was located on the hypertrophied antero-septal wall of the LV (Figure 1C, D, and Video 2).

Cardiac magnetic resonance imaging (CMR) showed hypertrophy in the basal and mid segments of the interventricular septum and the inferior wall. Late gadolinium enhancement was observed in the hypertrophied segments and at the right ventricular insertion point to the LV (Figure 2 and Video 3). Cardiac catheterization revealed a 110 mm Hg gradient between the LV and the LVOT. During surgery, a subaortic membrane was identified and resected, and a myectomy was performed.

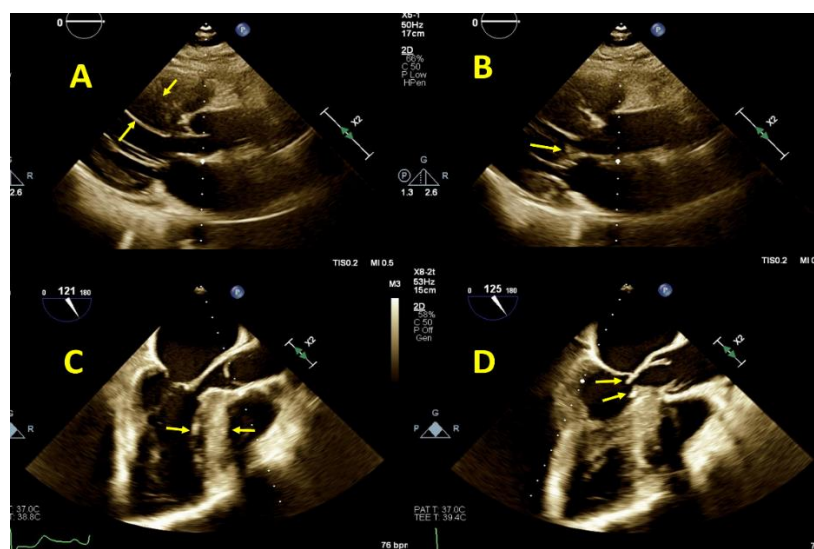


Figure 1. (A) Asymmetric septal hypertrophy (arrows) and (B) a subaortic membrane in the parasternal long-axis view of transthoracic echocardiography (arrow). (C) Asymmetric septal hypertrophy (arrows) and (D) a subaortic membrane (arrows) in the aortic valve long-axis view of transesophageal echocardiography.

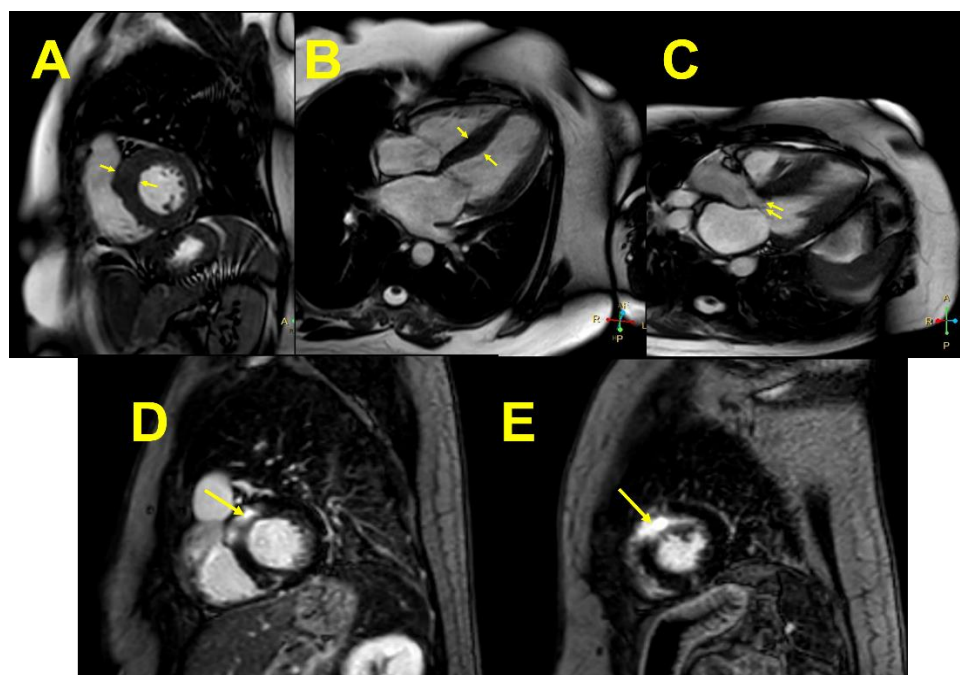


Figure 2. Cardiac magnetic resonance imaging. Steady-state free-precession sequence showing mid-short-axis (A) and apical 4-chamber (B) views, which show asymmetric septal hypertrophy (arrows); in addition, the 3-chamber view demonstrates a subaortic membrane (arrows) (C). The phase-sensitive inversion recovery sequence in basal and mid short-axis views demonstrates dense mid-wall late gadolinium enhancement, especially at the right ventricular insertion points (arrow), a typical pattern suggestive of hypertrophic cardiomyopathy (D, E).

Discussion

Approximately 1% of patients with HCM also have a subaortic membrane, and roughly half of these cases do not show SAM of the mitral leaflet at rest.⁷ In some patients with a hemodynamically significant subaortic membrane, ASH has been observed, although this is not a common feature.⁸ Conversely, ASH is frequently associated with HCM.⁷ Therefore, the coexistence of HCM and a subaortic membrane should be considered in patients with ASH, regardless of whether SAM is present.^{7,9,10} CMR helps distinguish between an isolated subaortic membrane and the coexistence of HCM and a subaortic membrane.¹¹ The presence of late gadolinium enhancement supports a diagnosis of HCM and suggests that both conditions coexist.¹⁰ Detecting HCM in these patients is clinically important because it requires further management, including monitoring for syncope, evaluating family history of sudden cardiac death, and performing Holter monitoring. Therefore, in patients with both a subaortic membrane and ASH, CMR may be required to make an accurate diagnosis. Moreover, differentiating dynamic from fixed LVOT obstruction in patients with ASH is important because each condition requires distinct treatment

that may be detrimental to the other. For example, administering high doses of beta-blockers, calcium channel blockers, or mavacamten to a patient with fixed LVOT obstruction may precipitate heart failure and pulmonary congestion.¹²

Conclusions

ASH is an echocardiographic finding with various etiologies, such as HCM and subaortic membrane. Because multiple etiologies can coexist in patients with ASH, meticulous investigation is necessary, as identifying the underlying cause has important clinical implications.

Supplement

Video 1. Transthoracic echocardiography. The parasternal long-axis view demonstrated asymmetric septal hypertrophy and a subaortic membrane with a mosaic pattern in the left ventricular outflow tract, suggestive of left ventricular outflow tract stenosis.

Video 2. Transesophageal echocardiography. Asymmetric septal hypertrophy and a subaortic membrane with a mosaic pattern in the left ventricular outflow tract were confirmed, suggestive of left ventricular outflow tract stenosis.

Video 3. Cardiac magnetic resonance imaging. Cine steady-state free-precession sequence in 3-chamber, 4-chamber, and short-axis views. Four-chamber and short-axis views show hypertrophied left ventricular segments and asymmetric septal hypertrophy, and the 5-chamber view demonstrates a circumferential subaortic membrane that leads to significant flow acceleration in the left ventricular outflow tract.

Declarations:

Ethical Approval

General written informed consent was obtained at hospital admission for the anonymous use of patient data in research.

Funding

According to the authors, this article has no financial support.

Conflict of Interest

The authors report no conflict of interest.

Acknowledgments

The authors have no acknowledgement to disclose.

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