# Aortic Stenosis With Dynamic Left Ventricular Outflow Obstruction: Diagnostic and Management Challenges—A Case Series

Frank Qian, MD, MPH, Pei-Chun McGregor, MD, Ahmad Nawid Latifi, MD, and Jayashri Aragam, MD, *Boston and West Roxbury, Massachusetts; and New York and Rochester, New York*

## INTRODUCTION

Although rare, aortic valve stenosis (AS) can coexist with hypertrophic cardiomyopathy (HCM) with obstruction. In an estimate from 207,880 patients from the National Inpatient Sample who underwent transcatheter aortic valve (AV) implantation from 2014 to 2018, about 0.38% had concomitant HCM.[1] When these two entities are coexistent, diagnosis and management becomes extremely difficult, as conventional assessment of AS may be inaccurate because of the presence of serial stenoses.[2-4] Current guidelines provide a framework for delineating the driving pathology when encountering patients with both of these conditions, though applying these principles to real-world practice remains challenging.[5,6] We hereby present three patients with concomitant AS and HCM with obstruction to highlight these diagnostic challenges via echocardiography and emphasize potential approaches to overcoming these limitations.

## CASE PRESENTATIONS

### Case 1

An 89-year-old man with previously known AS and HCM with obstruction began experiencing progressive dyspnea on exertion. Physical examination revealed a midpeaking systolic ejection murmur along the right sternal border and an apical holosystolic murmur. Transthoracic echocardiography (TTE) showed asymmetric left ventricular (LV) septal hypertrophy (1.7 cm; Figure 1, Video 1), systolic anterior motion (SAM) of the mitral valve (MV) with posteriorly directed moderate to severe mitral regurgitation (MR; Figure 2, Video 2), and a calcified AV. MR severity was qualitatively determined because of contamination of the accelerated LV outflow tract (LVOT) flow with proximal isovelocity flow acceleration, which prevented estimation of effective regurgitant orifice area. Doppler assessment revealed two distinct continuous-wave Doppler (CWD) velocity spectral displays: a midpeaking symmetric spectral profile with peak and mean gradients of 33 and 21 mm Hg (Figure 3A), respectively, and a second asymmetric late-peaking, "dagger-shaped" spectral profile with peak gradients of 57 mm Hg at rest (Figure 3B) and 106 mm Hg with the Valsalva maneuver (Figure 4B; all calculated using the modified Bernoulli equation). The LVOT peak velocity was 2.1 m/s, with a velocity-time integral of 45 cm (Figure 4A). Although calculated peak transaortic gradient using the full Bernoulli equation was 15 mm Hg, suggesting AV sclerosis, because of worsening dyspnea and uncertainty of the severity of obstruction at the level of the AV, the patient was referred for transesophageal echocardiography (TEE) and cardiac catheterization. TEE revealed normal-appearing right and left coronary cusps but a thickened, calcified noncoronary cusp with limited leaflet excursion (Video 3). AV area (AVA) by planimetry was estimated at 1.6 $cm^2$, suggesting only mild AS (Figure 4C). In addition, we performed quantification of the patient's MR. With a proximal isovelocity flow acceleration radius of 2.0 cm at a peak MR velocity of 7.5 m/s and a Nyquist limit of 25 cm/s, effective regurgitant orifice area was calculated to be 0.23 $cm^2$. Right heart catheterization and coronary angiography showed normal right-sided filling pressures at rest and nonobstructive coronary artery disease. Peak-to-peak gradient between the LV apex and aorta was 120 mm Hg (Figure 4D), but there was no significant gradient across the AV on pull-back. Diagnosis of HCM with obstruction along with mild AS was made, and the patient subsequently underwent successful alcohol septal ablation, with reported symptomatic improvement.

### Case 2

A 74-year-old man with known AS and HCM with obstruction presented with new exertional chest pain and dyspnea. Physical examination revealed a late-peaking 3/6 systolic ejection murmur with absent second heart sound and an apical holosystolic murmur. TTE demonstrated concentric LV hypertrophy with septal prominence, with the interventricular septum measuring 1.4 cm and the posterior wall measuring 1.1 cm (Figure 5), SAM (Figure 6A, Video 4) with likely severe posteriorly directed MR (Figure 6B), preserved LV systolic function, and a thickened AV. CWD across the LVOT demonstrated two distinct spectral displays: a midpeaking symmetric spectral profile with peak and mean gradients of 92 and 55 mm Hg, respectively, and a second asymmetric mid- to late-peaking "dagger-shaped" spectral profile

From the Section of Cardiovascular Medicine, Boston Medical Center and Boston University Chobanian & Avedisian School of Medicine, Boston, Massachusetts (F.Q.); Heartbeat Health, New York, New York (P.-C.M.); Sands-Constellation Heart Institute, Rochester Regional Health, Rochester, New York (A.N.L.); Department of Cardiology, Veterans Affairs Boston Healthcare System, West Roxbury, Massachusetts (J.A.); Harvard Medical School, Boston, Massachusetts (J.A.).

Keywords: Aortic stenosis, Hypertrophic cardiomyopathy, Left ventricular outflow tract obstruction, Echocardiography

Correspondence: Jayashri Aragam, MD, Department of Cardiology, Veterans Affairs Boston Healthcare System, 1400 VFW Parkway, West Roxbury, MA 02132. (E-mail: *jayashri.aragam@va.gov*).



2468-6441

https://doi.org/10.1016/j.case.2024.10.002

## VIDEO HIGHLIGHTS

**Video 1:** Case 1: two-dimensional TTE, parasternal long-axis view, demonstrates normal LV size and function with features of HCM, including asymmetric LV septal hypertrophy and SAM of the MV; also seen is a calcified AV.

**Video 2:** Case 1: two-dimensional TTE, apical four-chamber view without *(left)* and with *(right)* color flow Doppler, demonstrates SAM of the anterior MV leaflet with at least moderate MR and aliasing in the LVOT.

**Video 3:** Case 1: two-dimensional TEE, midesophageal short-axis (43°) view, demonstrates mild trileaflet AS with a calcified, restricted noncoronary cusp.

**Video 4:** Case 2: two-dimensional TTE, parasternal long-axis view, demonstrates normal LV size and function with features of HCM, including asymmetric LV septal hypertrophy with septal wall thickness of 1.4 cm and posterior wall thickness of 1.1 cm.

**Video 5:** Case 2: two-dimensional TEE, midesophageal short-axis (56°) view of the AV, demonstrates severely calcified and restricted AV leaflets suggestive of AS.

**Video 6:** Case 2: two-dimensional TEE, midesophageal long-axis (134°) view of the AV, demonstrates severely calcified and restricted AV leaflets suggestive of severe AS.

**Video 7:** Case 2: two-dimensional TEE with color flow Doppler, midesophageal oblique long-axis (51°) view, demonstrates normal LV size and function with eccentric and posteriorly directed MR.

**Video 8:** Case 3: two-dimensional TTE, parasternal long-axis view, demonstrates normal LV size and function with features of HCM, including asymmetric LV septal hypertrophy, SAM of the MV, and a severely calcified and restricted AV.

**Video 9:** Case 3: two-dimensional TEE, midesophageal biplane images, demonstrates a trileaflet AV with thickening, calcification and restricted leaflet motion.

**View the video content online at www.cvcasejournal.com.**

with peak gradients of 74 mm Hg at rest and 212 mm Hg with the Valsalva maneuver (Figure 7). LVOT peak velocity was 2.2 m/s. Peak transaortic gradient using the full Bernoulli equation was 70 mm Hg. Subsequent TEE showed a severely calcified and restricted AV with planimetry AVA of 0.74 $cm^2$ (Figure 8, Videos 5 and 6), elevated gradients (Figure 9A), and moderate to severe eccentric and posteriorly directed MR (Video 7). Preoperative cardiac catheterization confirmed transaortic mean gradient of 83 mm Hg and AVA of 0.5 $cm^2$ (Figure 9B) with nonobstructive coronary artery disease. With severe AS, HCM with obstruction, and MR, the patient underwent successful replacement of the AV and MV with septal myectomy, complicated by complete heart block requiring permanent pacemaker implantation. During surgery, degenerative MV pathology was found, consistent with components of both primary and secondary MR and preventing an attempt at MV repair. The patient has since recovered well.

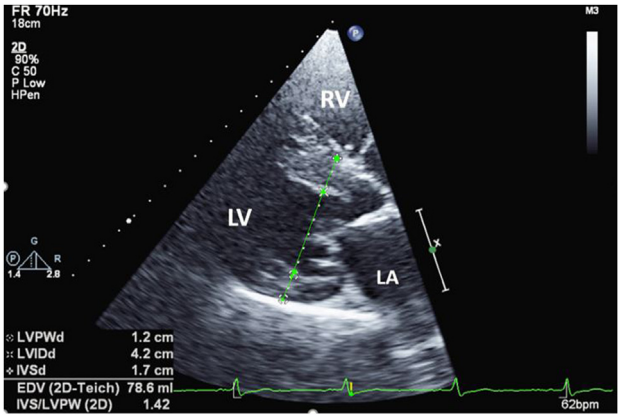


Figure 1 Case 1: two-dimensional TTE, parasternal long-axis diastolic view, demonstrates HCM with asymmetric left ventricular septal hypertrophy. *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

### Case 3

A previously healthy 54-year-old man was referred to the valve clinic for evaluation of AS after experiencing intermittent, nonexertional chest pain. The patient denied dyspnea, orthopnea, or lower extremity edema and was euvolemic on physical examination. Initial TTE revealed asymmetric LV septal hypertrophy (Video 8), SAM without significant MR, and a calcified AV with restricted leaflet excursion. There were two distinct CWD spectral displays across the LVOT: a midpeaking symmetric spectral profile with peak velocity of 3.9 m/s along with another asymmetric late-peaking "dagger-shaped" spectral profile demonstrating peak gradients of 27 mm Hg at rest and 66 mm Hg with the Valsalva maneuver (Figure 10). Diagnosis of latent HCM with dynamic obstruction was made along with moderate AS, with a calculated AVA of 1.4 $cm^2$ using LVOT peak velocity of 1.1 m/s and a velocity-time integral of 36 cm. One year later, the patient had syncope in the setting of rapid atrial fibrillation alternating with long ventricular pauses. Repeat TTE revealed no significant changes in gradients or LV function. The decision was made to place an automated implantable cardioverter-defibrillator because of syncope and a family history of sudden death. One year later, the patient began reporting exertional dyspnea. Repeat TTE showed stable transaortic gradients, so the patient was referred for TEE and cardiac catheterization to better characterize AS and LVOT obstruction. No significant SAM or LVOT obstruction was observed on TEE, but the AV was noted to be calcified with restricted leaflets and planimetry AVA of 1.1 $cm^2$ (Figure 11, Video 9). Cardiac catheterization revealed normal coronary arteries and a peak-to-peak LV-aortic gradient of 30 mm Hg (Figure 12A). Upon device interrogation, there were multiple episodes of atrial fibrillation with rapid ventricular rates, which were thought at the time to contribute to the symptoms. With up-titration of $\beta$-blocker, symptoms were alleviated until 1 year later, when the patient presented with worsening dyspnea. TTE revealed transaortic peak and mean gradients of 84 and 51 mm Hg (Figure 12B), respectively, and a calculated AVA of 0.7 $cm^2$ without significant LVOT obstruction. The patient subsequently underwent successful AV replacement, septal myectomy, and a maze procedure, with reduction of symptoms.

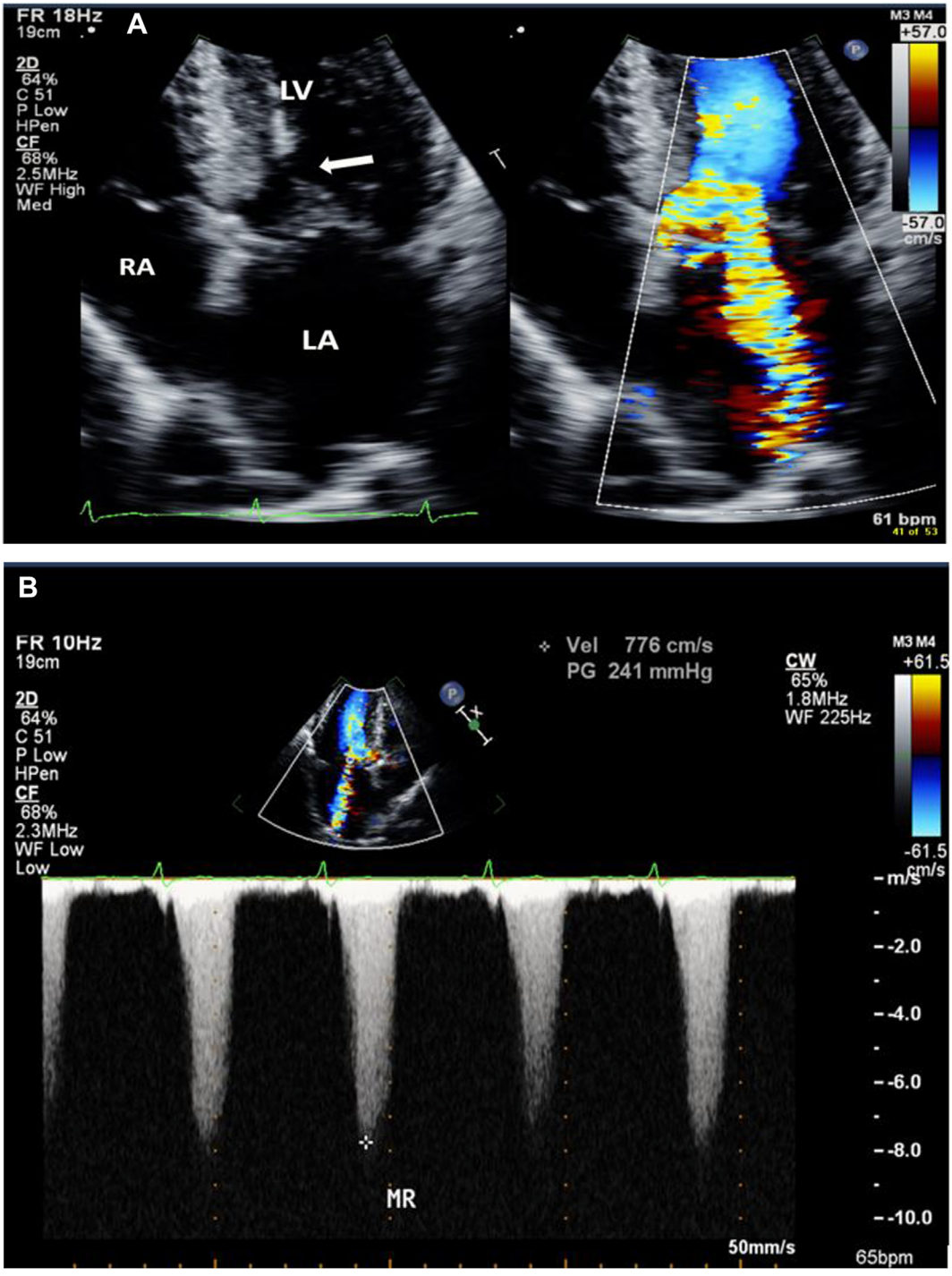


Figure 2 Case 1: **(A)** Two-dimensional TTE, apical four-chamber systolic view without *(left)* and with *(right)* color flow Doppler, demonstrates SAM of the anterior MV leaflet *(arrow)* with at least moderate MR and aliasing in the LVOT. **(B)** CWD across the MV demonstrates a spectral display with a high-velocity, late-peaking spectral profile from the MR jet (7.8 m/s, 241 mm Hg). *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

## DISCUSSION

The epidemiology of concomitant HCM with obstruction and AS is not well studied, because of the relative rarity of these two conditions coexisting. However, it is conceivable that with greater contemporary use of echocardiography and other multimodality imaging, there is expected to be greater recognition of these simultaneously occurring conditions. Existing guidelines provide a relatively limited framework for the diagnostic workup and management for these patients. A 2021 study identified 191

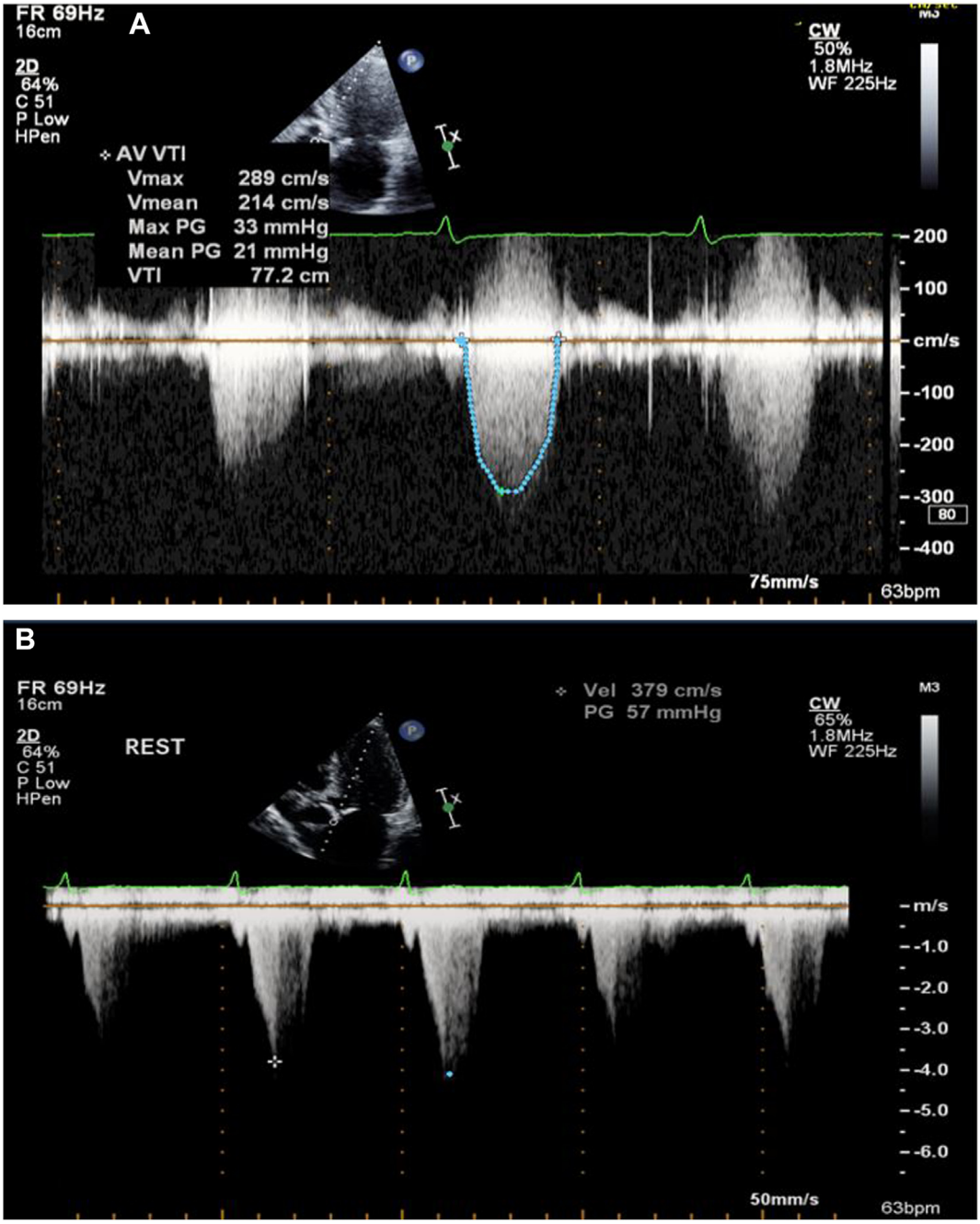


Figure 3 Case 1: two-dimensional TTE, apical five-chamber view, CWD across the AV, demonstrates a spectral display with two distinct spectral profiles: **(A)** midpeaking spectral profile (2.9 m/s, 33 mm Hg) and **(B)** late-peaking spectral profile (3.8 m/s, 57 mm Hg).

consecutive patients with concomitant HCM with obstruction and AS who were referred for myectomy and AV replacement over a 16-year period.[7] Of note, all patients underwent TEE as well as invasive hemodynamic testing to confirm the severity of AS, given challenges in applying the continuity equation with the presence of dual-obstructive physiology. In terms of demographics, patients were found to be generally older than patients with HCM with obstruction alone and did not exhibit any sex predominance (approximately 52% of the cohort were women). Interestingly, despite greater operative complexity, in-hospital mortality was low at 1.5%, as opposed to the 5% predicted mortality on the basis of Society of Thoracic Surgeons risk scoring.

HCM should be suspected when significant LV hypertrophy is present. These hypertrophic patterns are usually asymmetric, with a septal predominance, but can involve any location or even manifest as concentric hypertrophy.[8] Other supportive two-dimensional findings for HCM include a narrow LVOT and the presence of SAM of the MV with associated posteriorly directed MR.[9] In general, LV wall thickness of ≥15 mm at any site within the left ventricle is indicative of HCM, though 13 to 14 mm may

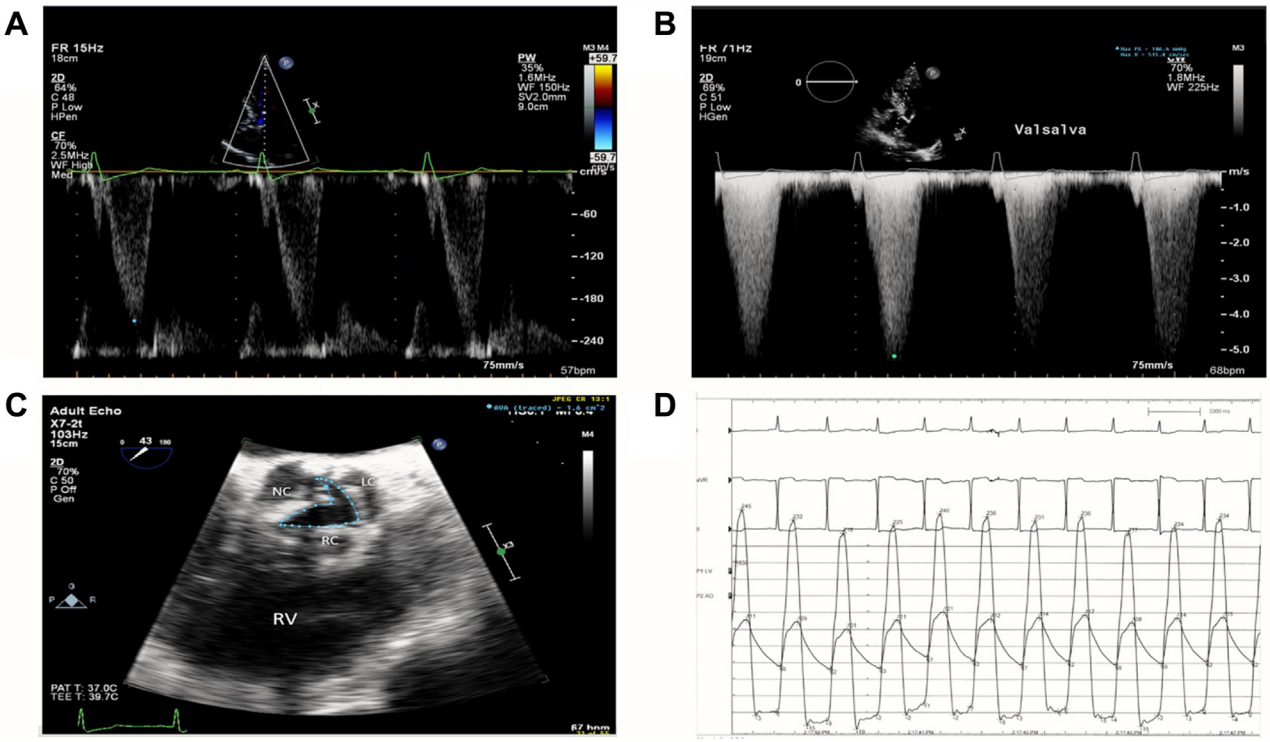


Figure 4 Case 1: two-dimensional TTE, apical five-chamber view, pulsed-wave Doppler **(A)** and CWD **(B)** spectral displays, demonstrates a maximal LVOT velocity of 2.1 m/s (18 mm Hg) and maximal late-peaking aortic velocity during Valsalva of 5.2 m/s (106 mm Hg), respectively. **(C)** Two-dimensional TEE, midesophageal short-axis (43°) systolic view, demonstrates mild AS with AVA of 1.6 $cm^2$ by planimetry. **(D)** Left heart catheterization pressure tracing from the LV apex and the aorta demonstrates an estimated peak-to-peak gradient of 120 mm Hg. *LC*, Left coronary cusp; *NC*, noncoronary cusp; *RC*, right coronary cusp

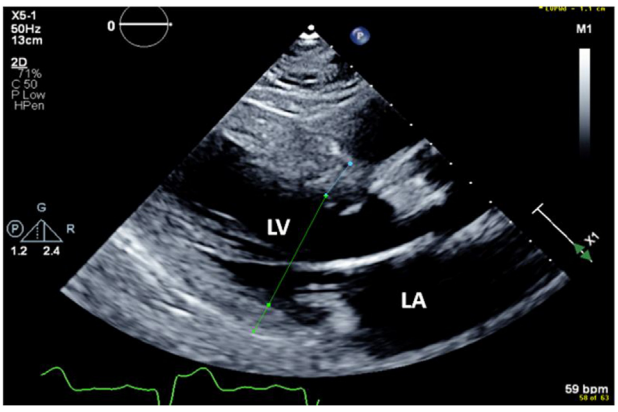


Figure 5 Case 2 two-dimensional TTE, parasternal long-axis end-diastolic view, demonstrates asymmetric left ventricular hypertrophy with septal wall thickness of 1.4 cm and posterior wall thickness of 1.1 cm). *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

also be considered diagnostic when combined with a family history of HCM, dynamic LVOT obstruction, or pathologic electrocardiographic findings.[10] Careful application of the aforementioned criteria is needed to ensure accurate diagnosis of HCM, given that other systemic conditions may also lead to significant LV hypertrophy, which may lead to misdiagnosis or potential overdiagnosis of HCM. Other echocardiographic mimickers of HCM, including cardiac amyloidosis, Danon disease, Fabry disease, hypertensive heart disease, and athlete's heart, can often be excluded on the basis of the presence of other associated clinical phenotypes or through additional imaging modalities, such as cardiovascular magnetic resonance. When evaluating AS, as alluded to previously, the continuity equation cannot be reliably used to calculate the AVA. The degree of AV calcification and excursion can be important clues along with AVA by planimetry on TEE. However, planimetry may be inaccurate when significant calcification causes shadowing or reverberation artifacts.[11] Cardiac computed tomography has emerged as a new imaging modality to assess the severity of AS, particularly in the presence of discrepant echocardiographic findings, through the assessment of the AV calcium score, which has been demonstrated to have significant adverse prognostic importance, as well as allow direct estimates of AVA through planimetry.[12] These alternative imaging modalities can be used for AV planimetry to better estimate stenosis severity in the noninvasive setting.[13]

Classically described spectral Doppler display for HCM with obstruction is a "dagger-shaped" spectral profile with peak velocity in late systole. Pulsed-wave Doppler is important to identify sites of obstruction. This spectral Doppler pattern is distinct from the parabolic pattern seen with valvular AS, which also demonstrates early (to mid) peak velocity in mild (or moderate) AS and late peak velocity in severe AS.[6] These spectral Doppler displays can also be "contaminated" by the presence of MR, which starts at the

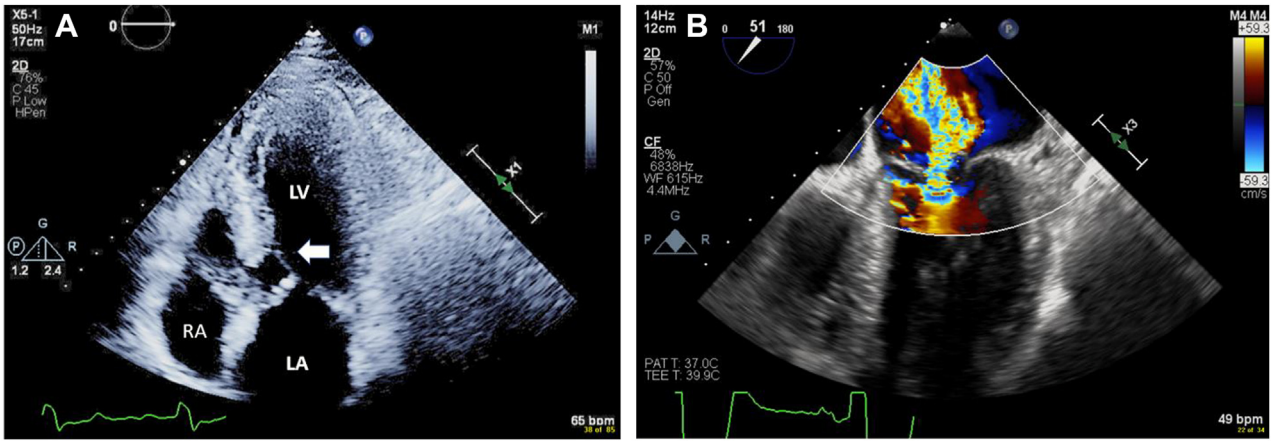


Figure 6 Case 2: **(A)** Two-dimensional TTE, apical four-chamber systolic view, demonstrates SAM of the anterior MV leaflet *(arrow)* and a dilated left atrium (LA). **(B)** Two-dimensional TEE with color flow Doppler, midesophageal long-axis (51°) view, demonstrates at least moderate MR with significant proximal flow acceleration and vena contracta. *LV*, Left ventricle; *RA*, right atrium.

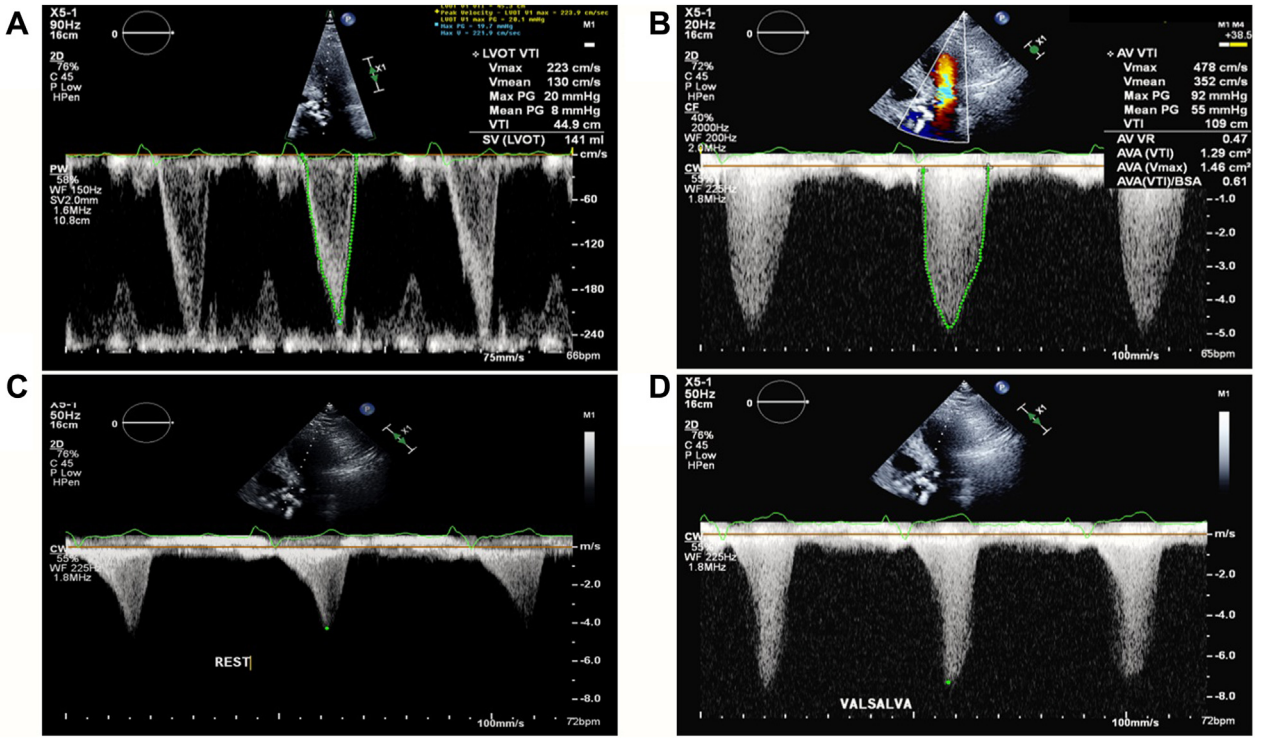


Figure 7 Case 2: Two-dimensional TTE, apical five-chamber view, pulsed-wave Doppler **(A)** and CWD **(B-D)** spectral displays, demonstrates a Doppler profile that peaks in late systole (**A**; 2.2 m/s), a fixed, severe AS Doppler profile that peaks in midsystole (**B**; 4.8 m/s), a late-peaking, functional Doppler profile at rest (**C**; 4.3 m/s), and a late-peaking dynamic LVOT obstruction Doppler profile during the Valsalva maneuver (**D**; 7.3 m/s).

onset of systole but intensifies during late systole with the development of outflow obstruction because SAM creates MV malcoaptation. The maximal velocity is typically higher with MR than AS, depicting the higher gradient between the left ventricle and left atrium. Concomitant AS and HCM with obstruction may reveal two distinct CWD spectral displays. Failure to recognize subaortic obstruction before AV replacement may result in adverse surgical outcomes.[14,15] Although two-dimensional and Doppler imaging can identify LVOT obstruction and distinguish it from AS, accurate assessment of AS is challenging in serial stenoses because assumptions from the simplified Bernoulli equation are no longer valid and may lead to overestimation of AS.[2,16] When proximal velocity is >1.5 m/s, using the full Bernoulli equation ($P = 4V_2^2 - V_1^2$, where $P$ is the instantaneous pressure gradient, $V_1$ is the velocity

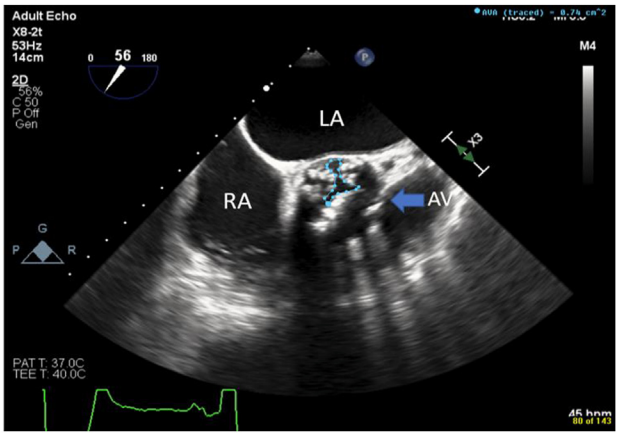


Figure 8 Case 2: two-dimensional TEE, midesophageal short-axis (56°) systolic view of the AV, demonstrates severe calcific AS *(arrow)* with a planimetered valve area of 0.74 $cm^2$. *LA*, Left atrium; *RA*, right atrium.

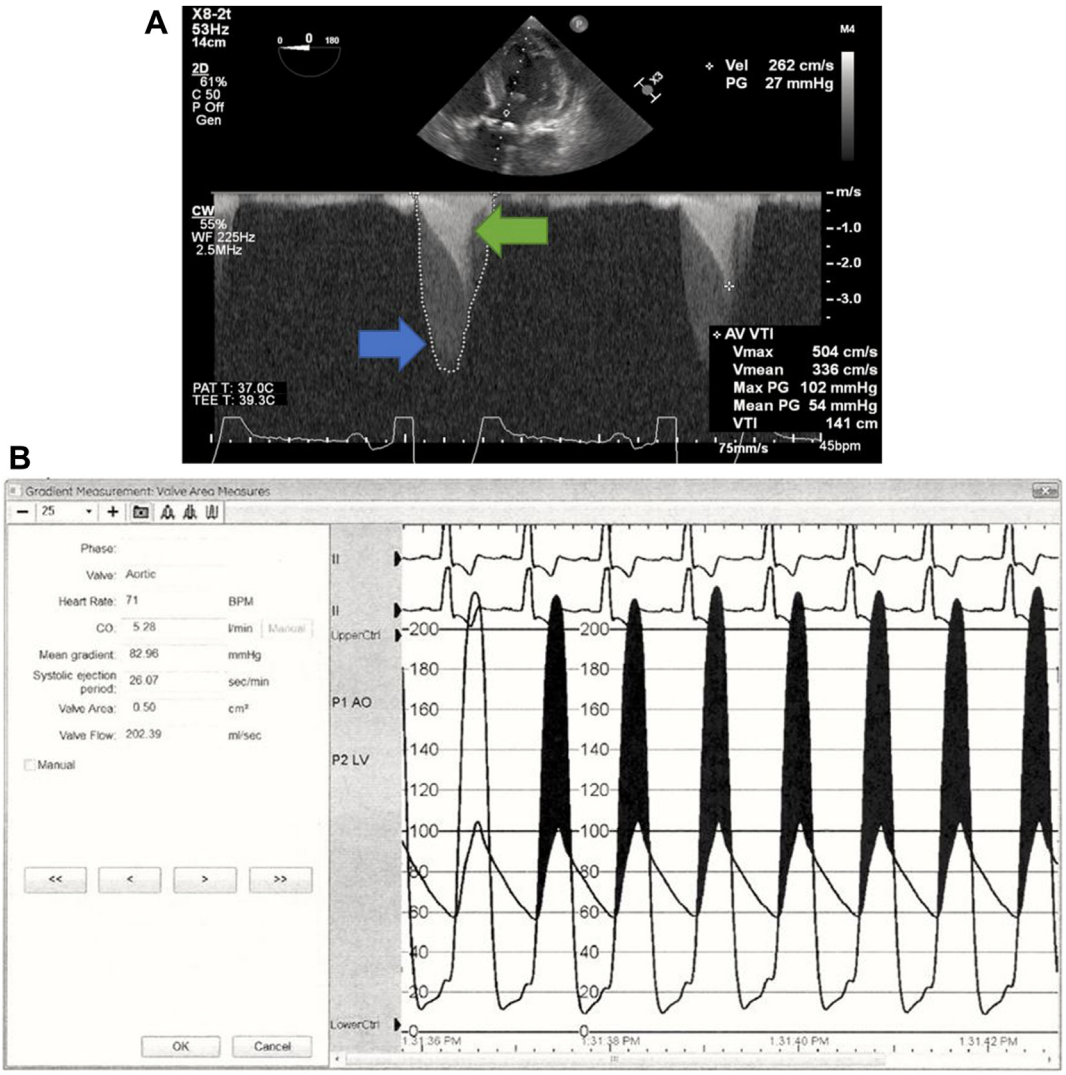


Figure 9 Case 2: **(A)** Two-dimensional TEE, midesophageal long-axis (0°) view, CWD spectral display, demonstrates two distinct spectral profiles across the AV **(A)**; the gray, midpeaking, higher velocity (5.0 m/s) profile represents the valvular AS *(blue arrow)*, and the whiter, ''dagger-shaped,'' lower velocity (2.6 m/s) profile represents the functional obstruction from HCM *(green arrow)*. **(B)** Left heart catheterization pressure tracing from the left ventricle and the aorta demonstrates a transaortic mean gradient of 83 mm Hg and AVA of 0.5 $cm^2$.

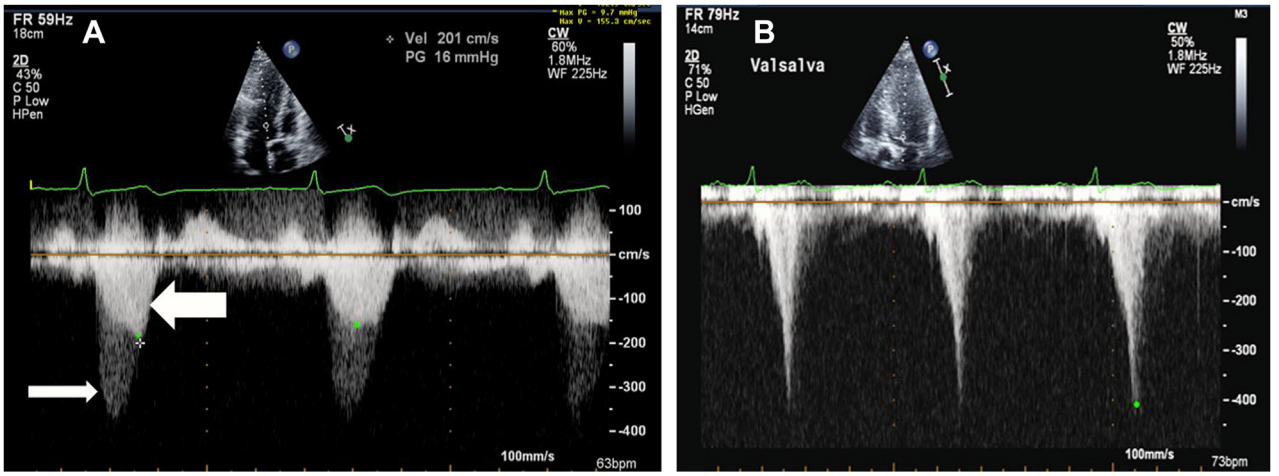


**Figure 10** Case 3: two-dimensional TTE, apical long-axis **(A)** and five-chamber **(B)** views with CWD through the AV, demonstrates two distinct profiles; a midsystolic peak flow *(thin arrow)* suggestive of AS and a late systolic peak flow *(thick arrow)* suggestive of dynamic LVOT obstruction at rest (10 mm Hg) and with the Valsalva maneuver (66 mm Hg).

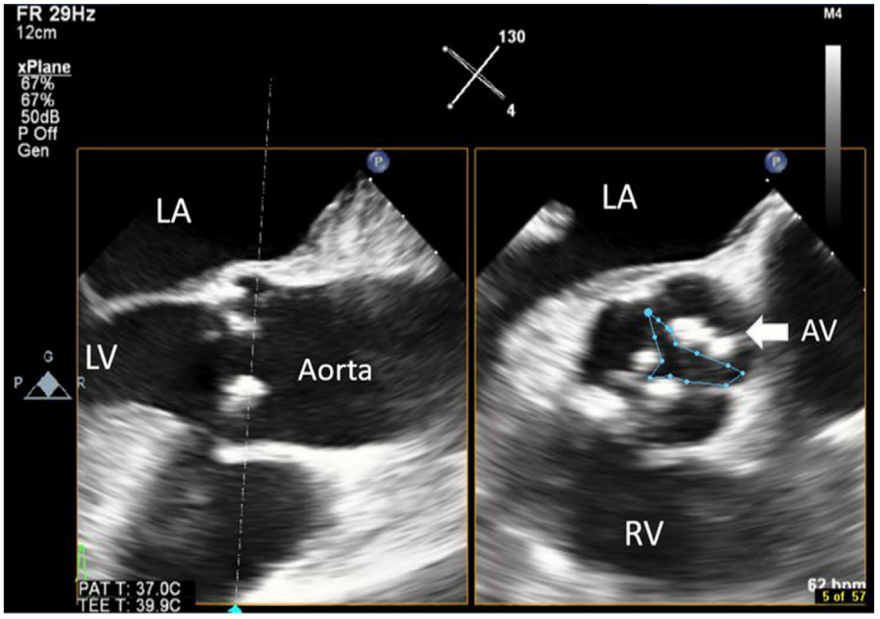


**Figure 11** Case 3: two-dimensional TEE, midesophageal biplane systolic images, demonstrates a trileaflet AV with thickening and calcification *(arrow)*; long axis–aligned *(left)* AV short-axis *(right)* planimetry demonstrates an AVA of 1.1 $cm^2$. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

proximal to the stenosis, and $V_2$ is the velocity distal to the stenosis) is recommended.[2,6,11] Although peak gradient can be calculated in this manner, mean gradient cannot be calculated. Invasive catheterization is of value when diagnostic uncertainty remains despite thorough noninvasive assessment, namely, by estimating the pressures at each level of obstruction, as seen in case 3.[2,15,16] These distinctions will become increasingly important in the era of transcatheter-based therapies, in which determining the principal site of obstruction will be key to selecting patients for combined surgical treatments (i.e., myectomy and AV replacement) vs transcatheter-based treatments (transcatheter AV implantation and alcohol septal ablation).

## CONCLUSION

Serial stenoses with AS and HCM with obstruction are rare and can pose diagnostic challenges when assessing hemodynamic significance at each level of obstruction. The initial diagnostic modality of choice is TTE. If diagnostic dilemma persists, TEE and invasive hemodynamics can be helpful.

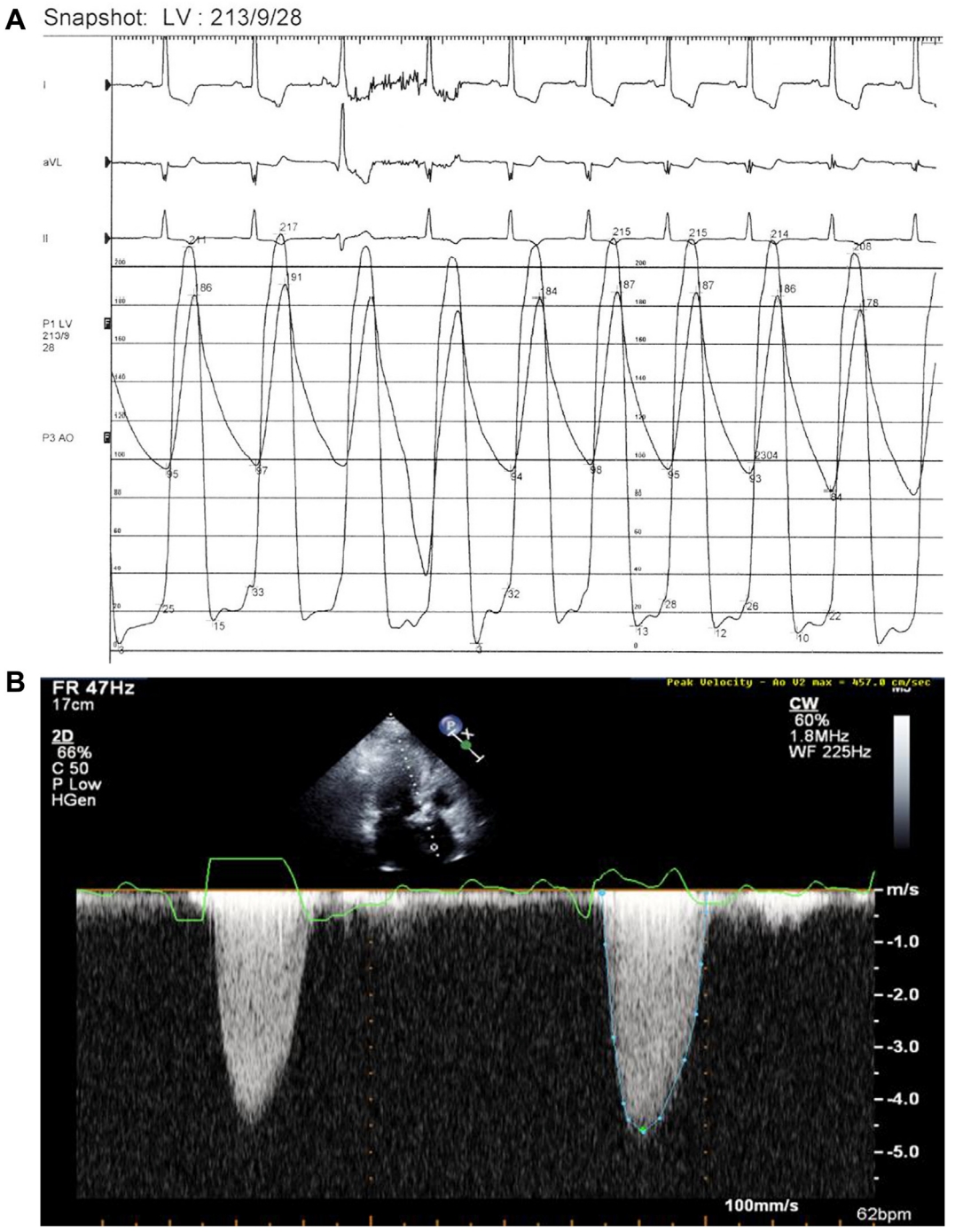


Figure 12 Case 3: **(A)** Left heart catheterization pressure tracing from the left ventricle and the aorta demonstrates a transaortic peak-to-peak gradient of 30 mm Hg. **(B)** Two-dimensional TTE, apical long-axis view with CWD through the AV, demonstrates a spectral Doppler display profile consistent with severe AS (peak gradient 83 mm Hg, mean gradient 51 mm Hg).

## ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

## CONSENT STATEMENT

The authors declare that since this was a non-interventional, retrospective, observational study utilizing de-identified data, informed consent was not required from the patient under an IRB exemption status.

## FUNDING STATEMENT

The authors declare that this report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## DISCLOSURE STATEMENT

The authors report no conflict of interest.

## SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2024.10.002.

## REFERENCES

1. Barasch E. Aortic stenosis associated with hypertrophic cardiomyopathy: a double trouble. Cardiology 2023;148:287-8.
2. Scantlebury DC, Geske JB, Nishimura RA. Limitations of Doppler echocardiography in the evaluation of serial stenoses. Circ Cardiovasc Imaging 2013;6:850-2.
3. Vanichsarn C, Siegel RJ. Fool me once, fool me twice: hypertrophic cardiomyopathy with aortic stenosis. Am J Med 2015;128:1076-9.
4. Kansal MM, Mookadam F, Tajik AJ. Double-trouble Doppler: reduction in aortic flow due to combined left ventricular outflow tract obstruction and severe aortic stenosis, complete diagnosis by transthoracic Doppler echocardiography. J Am Soc Echocardiogr 2011;24:471.e1-4.
5. Ommen SR, Mital S, Burke MA, Day SM, Deswal A, Elliott P, et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy: a report of the American College of Cardiology/American Heart Association joint committee on clinical practice guidelines. Circulation 2020;142:e558-631.
6. Baumgartner H, Hung J, Bermejo J, Chambers JB, Edvardsen T, Goldstein S, et al. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. J Am Soc Echocardiogr 2017;30:372-92.
7. Desai MY, Alashi A, Popovic ZB, Wierup P, Griffin BP, Thamilarasan M, et al. Outcomes in patients with obstructive hypertrophic cardiomyopathy and concomitant aortic stenosis undergoing surgical myectomy and aortic valve replacement. J Am Heart Assoc 2021;10:e018435.
8. Williams LK, Frenneaux MP, Steeds RP. Echocardiography in hypertrophic cardiomyopathy diagnosis, prognosis, and role in management. Eur J Echocardiogr 2009;10:iii9-14.
9. Nagueh SF, Phelan D, Abraham T, Armour A, Desai MY, Dragulescu A, et al. Recommendations for Multimodality cardiovascular imaging of patients with hypertrophic cardiomyopathy: an update from the American Society of Echocardiography, in collaboration with the American Society of Nuclear Cardiology, the Society for Cardiovascular Magnetic Resonance, and the Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr 2022;35:533-69.
10. Maron BJ, Desai MY, Nishimura RA, Spirito P, Rakowski H, Towbin JA, et al. Diagnosis and evaluation of hypertrophic cardiomyopathy: JACC state-of-the-art review. J Am Coll Cardiol 2022;79:372-89.
11. Jenni R, Martin RP. Letter by Martin and Jenni regarding article, "Limitations of Doppler echocardiography in the evaluation of serial stenoses". Circ Cardiovasc Imaging 2014;7:211.
12. Pawade T, Sheth T, Guzzetti E, Dweck MR, Clavel MA. Why and How to measure aortic valve calcification in patients with aortic stenosis. JACC Cardiovasc Imaging 2019;12:1835-48.
13. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, et al. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr 2019;32:1-64.
14. Susini G, Zucchetti M, Sisillo E, Bortone F, Salvi L, Ceriani R, et al. Diagnostic pitfalls with the combination of a hypertrophic cardiomyopathy and aortic valvular stenosis. J Cardiothorac Vasc Anesth 1991;5:66-8.
15. Ishimura M, Takayama M, Saji M, Takamisawa I, Umemura J, Sumiyoshi T, et al. A case of hypertrophic obstructive cardiomyopathy with aortic stenosis. J Cardiol Cases 2014;9:129-33.
16. Nanda NC, Gramiak R, Shah PM, Stewart S, DeWeese JA. Echocardiography in the diagnosis of idiopathic hypertrophic subaortic stenosis co-existing with aortic valve disease. Circulation 1974;50:752-7.