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Simultaneous Intracardiac Pressure Measurement to Detect the Origin of Pressure Gradient in a Patient with Coexisting Aortic Stenosis and Asymmetrical Interventricular Septal Hypertrophy

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Female, 64
Final Diagnosis: Aortic stenosis
Symptoms: Short of breath
Medication: —
Clinical Procedure: Aortic valve replacement
Specialty: Cardiology

Objective: Challenging differential diagnosis

Background: Both aortic stenosis (AS) and left ventricular outflow tract (LVOT) obstruction can cause a pressure gradient along the LVOT. The interference caused by these 2 stenotic diseases are still not well understood, which might make echocardiographic evaluation difficult.

Case Report: A 60-year-old female was referred with occasional chest discomfort. Echocardiography revealed AS and asymmetrical hypertrophy of the basal interventricular septum (IVS). Continuous-wave Doppler recordings from the LV apex along a line oriented through the aortic valve showed a high velocity: peak velocity, 4.1 m/s; peak pressure gradient, 67.1 mmHg. Based on echocardiographic findings, the main cause of the pressure gradient was likely AS, but the coexistence of LVOT obstruction could not be ruled out. Therefore, simultaneous intracardiac pressure measurement was performed to detect the precise origin of the pressure gradient. This revealed that AS was the main cause of the pressure gradient. In addition to baseline measurement, measurement during continuous isoproterenol infusion was applied, which denied a latent LVOT obstruction. Elective aortic valve replacement improved the patient's symptoms and decreased IVS thickness.

Conclusions: Simultaneous intracardiac pressure measurement was effective to detect the origin of pressure gradient in a patient with severe AS accompanied by asymmetrical IVS hypertrophy. This experience provides insight into the clinical assessment of coexisting stenotic diseases and the association between AS and asymmetrical IVS hypertrophy.

MeSH Keywords: Aortic Stenosis, Subvalvular • Hemodynamics • Ventricular Outflow Obstruction

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Background

Both aortic stenosis (AS) and left ventricular outflow tract (LVOT) obstruction can cause a pressure gradient along the LVOT [1–3]. To determine the origin of a pressure gradient using echocardiography is difficult when these 2 disorders coexist, and the interference caused by these 2 stenotic diseases is still not well understood. Here, we describe a case of a 60-year-old female patient who presented with AS accompanied by asymmetrical interventricular septum (IVS) hypertrophy. Simultaneous intracardiac pressure measurements revealed that AS was the main cause of the pressure gradient, and denied the coexistence of LVOT obstruction.

Case Report

A 60-year-old female was referred with occasional chest discomfort that tended to occur with physical exertion. The patient had hypertension that was under treatment with amlodipine 5 mg daily and valsartan 80 mg daily. She had undergone a partial gastrectomy to treat gastric cancer at the age of 42 years, and neurosurgical clipping of a ruptured cerebral aneurysm that had caused a subarachnoid hemorrhage at the age of 55 years. A physical examination revealed the following: height of 160 cm; weight of 44 kg; blood pressure of 142/72 mmHg; and a regular pulse of 72 beats per minute. The chest was without rales, but a grade 4 systolic murmur was audible at the right upper sternal border. Laboratory findings showed that N-terminal-pro B-type natriuretic peptide had increased to 456 pg/mL. Chest radiography findings were essentially normal (cardio-thoracic ratio, 45%) (Figure 1A). Electrocardiography revealed sinus rhythm, but voltage criteria indicated left ventricular hypertrophy (Figure 1B).

Transthoracic (TTE) and transesophageal echocardiography (TEE) revealed AS and asymmetrical hypertrophy of the basal IVS (Figure 2A, 2B). The IVS and the LV posterior wall were 18 mm and 11 mm thick, respectively. The area of the aortic valve calculated by planimetry from a TEE image was 0.60 cm^2 (Figure 2C). Continuous-wave Doppler recordings from the LV apex along a line oriented through the aortic valve showed a high velocity wave with a round contour and an earlier peak. The peak velocity was 4.1 m/s, and maximal and mean pressure gradient values were 67.1 mmHg and 41.3 mmHg, respectively (Figure 2D). On the other hand, a mosaic flow pattern was observed in the LVOT, and continuous-wave Doppler recording in a direction slightly shifted by the aforementioned showed dagger-shaped wave with a late systolic peak velocity of 3.0 m/s (Figure 2E). Systolic anterior motion of anterior mitral leaflet was not observed.

These echocardiographic findings were insufficient to precisely locate the origin of the pressure gradient. Therefore, cardiac catheterization proceeded via a bilateral radial approach. Left ventriculography in the right anterior oblique projection did not reveal any apparent obstruction of the subaortic LVOT (Figure 3A). Intracardiac simultaneous pressure measurements proceeded using two 5-Fr pigtail catheters. Pressure at 2 of the 3 sites (ascending aorta, subaortic LVOT, and LV apex) was

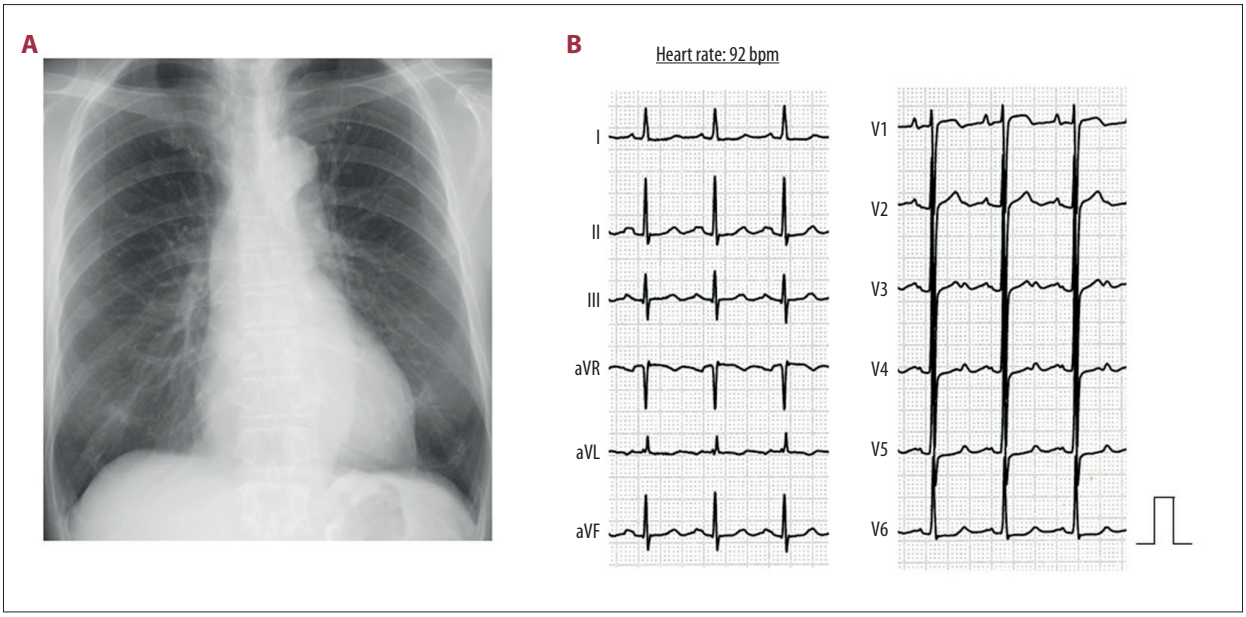

Figure 1. Chest radiography (**A**) and 12-lead electrocardiogram (**B**) at the first patient visit. Chest radiography findings were essentially normal: cardio-thoracic ratio was 45%. Electrocardiography revealed sinus rhythm at a rate of 91 beat per minutes, but voltage criteria indicated left ventricular hypertrophy.

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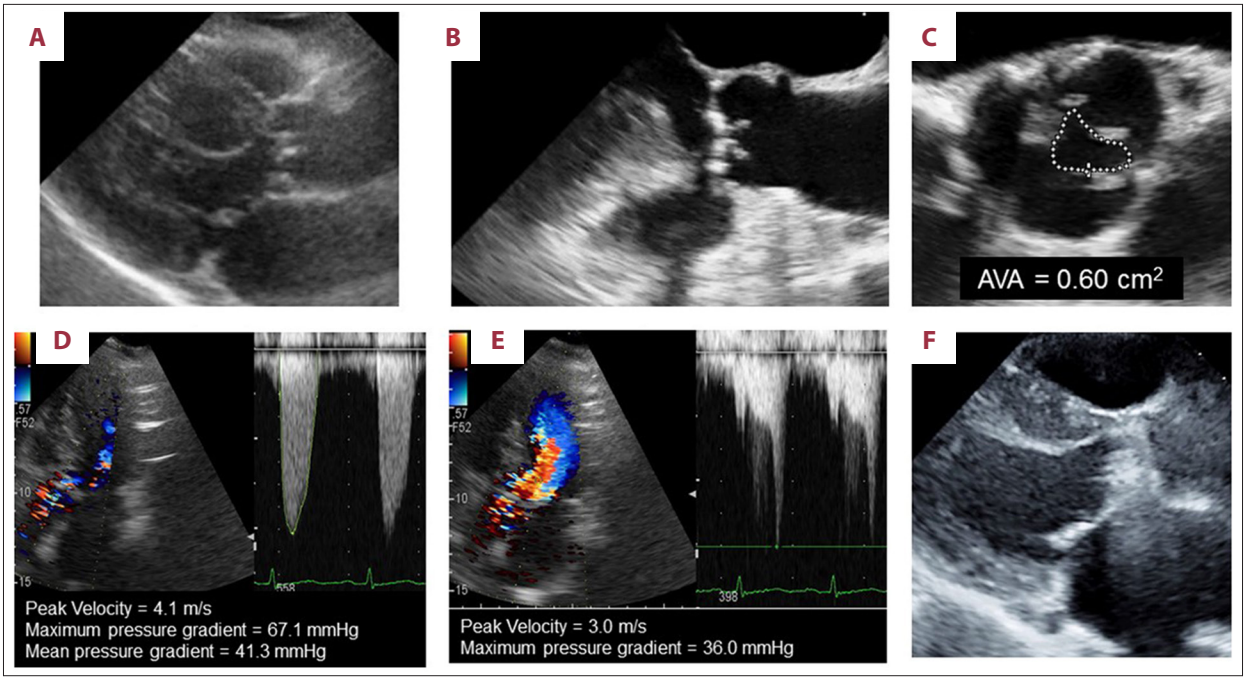

Figure 2. Transthoracic echocardiography of parasternal long-axis view (**A**), and transesophageal echocardiography of mid-esophageal aortic valve long-axis view (**B**), and short-axis view (**C**) before the operation. Continuous-wave Doppler recorded from the LV apex with the interrogation being directed through the left ventricular tract (**D, E**). Transthoracic echocardiography of parasternal long-axis view after the operation (**F**). There was a LVOT narrowing due to asymmetrical hypertrophy of basal intraventricular septum (**A, B**). The AVA was calculated as 0.60 cm^2 by planimetry (**C**). The continuous-wave Doppler recordings from the LV apex along a line oriented through the aortic valve showed a peak velocity of 4.1 m/s, maximum pressure gradient of 67.1 mmHg, and mean pressure gradient of 41.3 mmHg (**D**). The continuous-wave Doppler recordings in a direction slightly shifted by that in D showed dagger-shaped wave with a late systolic peak velocity of 3.0 m/s (**E**). After the operation, the LVOT disappeared (**F**). LVOT – left ventricular outflow tract; AVA – aortic valve area.

simultaneously measured at baseline and again during continuous isoproterenol infusion. Continuous isoproterenol infusion was applied considering a possibility of coexistence of latent LVOT obstruction. One catheter was placed in the subaortic LVOT, and the other was placed in the ascending aorta at baseline. The catheter in the ascending aorta was then moved to the LV apex. During continuous isoproterenol infusion, the catheter in the subaortic LVOT was moved to the ascending aorta. We found peak-to-peak and maximal instantaneous pressure gradients of 45 mmHg and 80 mmHg, respectively, between the subaortic LVOT and the ascending aorta, and a peak-to-peak pressure gradient of 12 mmHg between the subaortic LVOT and the LV apex. The peak-to-peak pressure gradient during continuous isoproterenol infusion was 20 mmHg between the subaortic LVOT and the LV apex, whereas peak-to-peak and maximal instantaneous pressure gradients were 90 mmHg and 125 mmHg between the LV apex and the ascending aorta, respectively (Figure 3B). These results confirmed that AS was the main cause of the pressure gradient and LVOT obstruction was not accompanied. Elective aortic valve replacement proceeded using a mechanical valve. The patient's postoperative course was uneventful, and she remained free of chest discomfort even with physical exertion. At 12 months after the procedure, TTE showed that the thickness of the IVS had decreased from 18 mm to 11 mm (Figure 2F). Dagger-shaped Doppler wave was not observed anymore.

Discussion

Doppler echocardiography is useful to evaluate cardiac stenotic disease, but it has several limitations [4]. Although pulsed-Doppler ultrasonography can determine direction and flow velocity at a specific point, its maximal recordable velocity is generally 1.5–2 m/s due to the Nyquist limit. In contrast, continuous-wave Doppler is not constrained by velocity limits and it can record velocities that exceed those of pulsed Doppler. However, the precise location of the maximal velocity must be deduced by integrating the interrogation line direction with known cardiac anatomy. Thus, the coexistence of 2 stenotic diseases on the same interrogation line interferes with Doppler echocardiographic evaluations of the severity of both diseases. AS can also be evaluated using direct 2-dimensional planimetry [5], but LVOT obstruction would affect aortic valve opening, causing inaccuracies. In the present case, the main cause of the pressure gradient was likely AS on the basis of smooth contour

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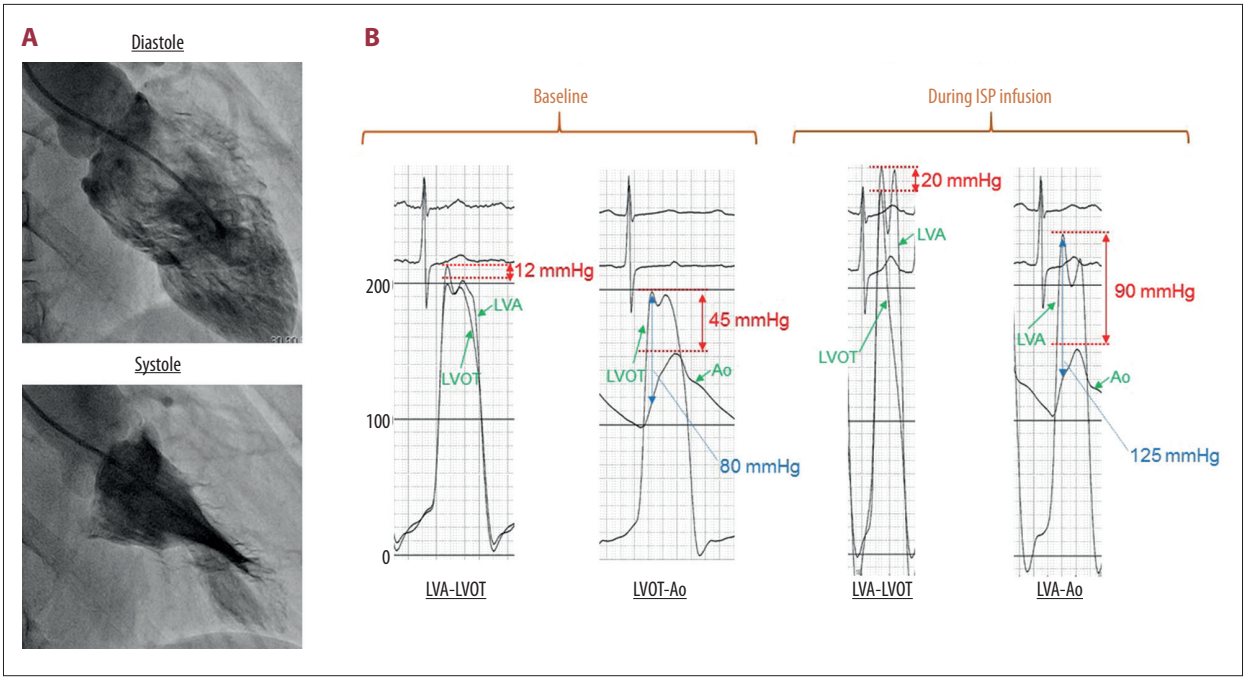

Figure 3. Left ventriculogram in the right anterior oblique projection (**A**) and simultaneous pressure recordings (**B**).There was no apparent obstruction in the subaortic LVOT. We found peak-to-peak and maximum instantaneous pressure gradients of 45 mmHg and 80 mmHg, respectively between subaortic LVOT and Ao, and a peak-to-peak pressure gradient of 12 mmHg between subaortic LVOT and LV apex. The peak-to-peak pressure gradient during continuous isoproterenol infusion was 20 mmHg between the subaortic LVOT and the LV apex, whereas peak-to-peak and maximal instantaneous pressure gradients were 90 mmHg and 125 mmHg between the LV apex and the Ao, respectively. LVOT – left ventricular outflow tract; Ao – ascending aorta.

with a mid-systolic peak, however, dagger-shaped contour was also observed, suggesting a possibility of coexistence of LVOT obstruction.

Simultaneous pressure measurements provide valuable information when 2 stenotic diseases are suspected. Pressure gradients are commonly determined by measuring peak-to-peak gradients. However, a peak-to-peak gradient between the ascending aorta and the subaortic LVOT has no physiological basis, because maximal left ventricular pressure and aortic pressure are rarely simultaneous [6,7]. The maximal instantaneous pressure gradient reflects the severity of AS more accurately than the peak-to-peak gradient. The pull-back technique using a single catheter is more convenient than simultaneous pressure measurements, but the maximal instantaneous pressure gradient cannot be measured.

On the basis of echocardiography and simultaneous pressure recording at baseline, the diagnosis of severe AS was made. However, a dynamic intraventricular pressure gradient is one of the most critical features of LVOT obstruction. Many patients with LVOT obstruction do not have a systolic pressure gradient at rest, but this can be provoked using inotropic agents, the Valsalva maneuver, an extrasystole, and potent systemic dilators. We applied continuous infusion of isoproterenol during simultaneous pressure measurement considering a possibility of latent LVOT obstruction, because whether LVOT obstruction is accompanied or not is very important to determine an operation strategy: aortic valve replacement only or aortic valve replacement plus myectomy. The standard indication for myectomy is symptomatic severe LVOT pressure gradient ≥50 mmHg with or without provocation maneuvers. In the present case, a small pressure gradient between the subaortic LVOT and LV apex was observed even during isoproterenol infusion, which denied a latent LVOT obstruction and the need for myectomy.

Flow acceleration due to AS could cause a non-obstructive pressure gradient between the subaortic LVOT and the LV apex [7]. In this regard, the mild pressure gradient between the LVOT and LV apex in our patient might have been attributable to flow acceleration due to AS rather than LVOT narrowing.

Dweck et al. found asymmetrical LV hypertrophy (LVH) in 27% of patients with AS and it was most frequently located in the septum at the basal and mid-cavity [8]. The findings of that study suggested that a detailed hemodynamic analysis is required for some patients with AS to determine the origin of pressure gradient, as was the case for our patient. Although

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whether or not hypertrophic cardiomyopathy has caused asymmetrical LVH is difficult to determine morphologically, this was ruled out in our patient because her LVH regressed after aortic valve replacement.

Rader et al. reported that the LV mass decreases in the range of 17% to 31% within the first year after surgical aortic valve replacement in patients with AS [9]. Based on this evidence, we simply replaced the aortic valve and did not provide any intervention for the IVS hypertrophy. As expected, the thickness of the IVS decreased after this procedure.

Conclusions

Simultaneous intracardiac pressure measurement was effective to detect the origin of pressure gradient in a patient with severe AS accompanied by asymmetrical IVS hypertrophy. This experience provides insight into the clinical assessment of coexisting stenotic diseases and the association between AS and asymmetrical IVS hypertrophy.

Conflict of interest

None.

References:

1. Yoshinaga M, Yoshikawa D, Ishii H et al: Clinical characteristics and long-term outcomes of hypertrophic cardiomyopathy. Int Heart J, 2015; 56: 415–20
2. Saikrishnan N, Kumar G, Sawaya FJ et al: Accurate assessment of aortic stenosis. Circulation, 2014; 129: 244–53
3. Maron BJ, Maron MS, Wigle D, Braunwald E: The 50-year history, controversy, and clinical implications of left ventricular outflow tract obstruction in hypertrophic cardiomyopathy. J Am Coll Cardiol, 2009; 54: 191–200
4. Anavekar NS, Oh JK: Doppler echocardiography: A contemporary review. J Cardiol, 2009; 54: 347–58
5. Hoffmann R, Flachskampf FA, Hanrath P: Planimetry of orifice area in aortic stenosis using multiplane transesophageal echocardiography. J Am Coll Cardiol, 1993; 22: 529–34
6. Currie PJ, Hagler DC, Seward JB et al: Instantaneous pressure gradient: A simultaneous Doppler and dual catheter correlative study. J Am Coll Cardiol, 1986; 7: 800–6
7. Pasipoularides A. Clinical assessment of ventricular ejection dynamics with and without outflow obstruction. J Am Coll Cardiol, 1990; 15: 859–82
8. Dweck MR, Joshi S, Murigu T et al: Left ventricular remodeling and hypertrophy in patients with aortic stenosis: Insights from cardiovascular magnetic resonance. J Cardiovasc Magn Reson, 2012; 14: 50
9. Rader F, Sachdev E, Arsanjani R, Siegel RJ: Left ventricular hypertrophy in valvular aortic stenosis: mechanism and clinical implications. Am J Med, 2015; 128: 344–52

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