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CASE REPORT
Other

Dynamic improvement of an acute exacerbated subaortic pressure gradient after intravenous propranolol and cibenzoline, recorded using a pressure wire: a case report

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Background Beta-blockers and Class 1A antiarrhythmics decrease the subaortic pressure gradient in hypertrophic obstructive cardiomyopathy. However, real-time monitoring of the pressure gradient transition during intravenous therapy, based on cardiac catheterization, has never been reported.

Case summary A 52-year-old man, with an history of hypertension, was transferred to our hospital, complaining of angina. A 12-lead electrocardiogram showed diffuse ST-segment depression, and transthoracic echocardiography revealed a thickened left ventricular outflow tract (LVOT) septum, resulting in LVOT obstruction which had never been diagnosed. Besides, severe mitral regurgitation (MR) due to systolic anterior motion was detected. Emergent cardiac catheterization revealed normal coronary arteries and severe MR. Simultaneous pressure measurements were taken at the ascending aorta (using a coronary catheter) and left ventricle (using a pressure wire). The subaortic systolic pressure gradient was 147 mmHg: 251 mmHg in the left ventricle and 104 mmHg in the aorta. Intravenous cibenzoline, following propranolol, was administered to ameliorate the pressure gradient, following which his chest pain disappeared immediately; the pressure gradient decreased to 13 mmHg. Further, severe MR was diminished. Oral bisoprolol and cibenzoline administration effectively stabilized his condition after catheterization.

Discussion Monitoring the simultaneous pressure between the left ventricle and aorta with a pressure wire revealed drastic improvement in the subaortic systolic pressure gradient. Owing to the soft, fine structure, the pressure wire allowed recording of the subaortic pressure gradient stably with less frequent premature ventricular contractions. Furthermore, this method could decrease the burden of catheter-related complications by eliminating the need for multiple atrial punctures.

Keywords Hypertrophic obstructive cardiomyopathy • Propranolol • Cibenzoline • Pressure wire • Pharmacological response • Case report

ESC Curriculum 6.5 Cardiomyopathy • 3.4 Coronary angiography

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Learning points

- Intravenous propranolol and cibenzoline are helpful in reducing the subaortic pressure gradient in left ventricular outflow tract obstruction.
- A pressure wire inserted through a coronary catheter can be used to stably quantify the dynamic subaortic pressure gradient, with fewer PVCs than catheter placed in the left ventricle, via a transradial, single-puncture approach.
- This is the first report that clearly visualized the concurrent efficacy of these drugs in reducing pressure gradient by cardiac catheterization.

Introduction

Hypertrophic myocardial septum on the left ventricular outflow tract leads to an increased subaortic pressure gradient and causes chest pain. Beta-blockers and Class 1A anti-arrhythmic drugs have been noted to decrease the subaortic pressure. However, to our knowledge, no studies on the real-time improvement in the subaortic pressure gradient after using these drugs during cardiac catheterization have been reported.

Timeline

Timeline	Event
49 years old	Patient was diagnosed with hypertension and asymptomatic concentric hypertrophy by transthoracic echocardiography (TTE). However, no left ventricular outflow tract (LVOT) obstruction was detected.
52 years old (before admission)	Patient had sudden chest pain three hours after drinking 1000 mL beer.
Upon admission	TTE showed thickened ventricular septum on LVOT. Colour doppler ultrasound showed mosaic flow at LVOT and severe MR due to systolic anterior motion of mitral valve.
At catheter laboratory	Coronary artery disease was not found. Pressures across LVOT were simultaneously recorded by a pressure wire inserted through a coronary angiographic catheter. Intravenous propranolol and cibenzoline were observed to be helpful in decreasing both the subaortic pressure gradient and the mitral regurgitation, which resulted in his chest pain disappearing.
After catheterization	Oral bisoprolol and cibenzoline were prescribed to control the LVOT obstruction.
On hospital Day 7	Follow-up TTE revealed that the pressure gradient was under control and severe MR disappeared
On hospital Day 11	Patient was discharged from our department without any symptoms.

Case report

A 52-year-old man was transferred to our hospital due to chest pain and syncope following excessive alcohol consumption. He had no medical history except for a prescription of valsartan for systemic hypertension, and transthoracic echocardiography (TTE) noted no left ventricular outflow tract (LVOT) obstruction 3 years ago. On admission, his vital signs were as follows: blood pressure of 90/56 mmHg, pulse rate of 93 bpm, and O_2 saturation of 99% on room air. On physical examination, there were no significant findings except for a cold sweat. An initial 12-lead electrocardiogram showed ST-segment depression on the anterior and inferior leads and sinus tachycardia (*Figure 1*). Serum troponin T level was 0.028 ng/mL (normal range: 0–0.014 ng/mL). TTE on admission demonstrated no any regional wall motion abnormality but a hypertrophic myocardial septum on the LVOT (*Figure 2*) and severe mitral regurgitation (MR) with mosaic-like flow due to systolic anterior motion of the mitral valve (see Supplementary material online, *Video S1*). The maximum peak pressure gradient of the LVOT was measured at 139.6 mmHg (*Figure 3A*). An emergent cardiac catheterization was performed to assess and control his persistent chest pain. There was no coronary artery stenosis (*Figure 4A*). Diagnosing the chest pain as an acute exacerbation of the LVOT obstruction, we attempted to measure the pressure gradient between the aorta and left ventricle. Initially, an angiographic diagnostic catheter (Goodtech 5Fr-Judkins Right 4.0, Goodman Medical Ireland Limited, Ireland) was placed in the left ventricle. However, the diagnostic catheter provoked multiple premature ventricular contractions (PVCs) induced by the attachment to the ventricular wall. To avoid them, an angiographic diagnostic catheter and a pressure wire (PressureWire™ X, Abbott, USA) were used to measure aortic and left ventricular pressure (*Figure 4B*) and successfully recorded continuous, simultaneous pressures (*Figure 5A*). The patients initial subaortic systolic pressure gradient was measured at 147 mmHg (104 mmHg, aorta; 251 mmHg, left ventricle) (*Figure 5B*). After administering 4 mg propranolol, his subaortic pressure gradient decreased to 35 mmHg (*Figure 5C*). Despite the reduce in pressure gradient, his chest pain persisted. Therefore, 70 mg cibenzoline was administered and his pressure gradient further reduced to 13 mmHg (*Figure 5D*). A subsequent TTE showed that his MR improved from severe to mild grade (see Supplementary material online, *Video S2*) and the peak subaortic pressure gradient decreased to 24.6 mmHg (*Figure 3B*). Eventually, his chest pain resolved. Based on these findings, oral cibenzoline and bisoprolol were subsequently prescribed. On Day 7, a follow-up TTE showed that the pressure gradient on the LVOT remained to be decreased at 12 mmHg with mild MR. The patient did not have recurrent chest discomfort and was discharged on hospital Day 11.

Discussion

Subaortic pressure gradient fluctuations are frequently observed among patients with left ventricular outflow obstruction; their physical condition influences their subaortic pressure gradients, regardless of the degree of the pressure gradient at rest.[1] Excessive alcohol consumption was assumed to be the main cause of the exacerbated fluctuations observed in this case.[2] To manage the pressure gradient, beta-blockade with Class 1A anti-arrhythmic drugs have been recommended.[3,4] In this case, intravenous propranolol

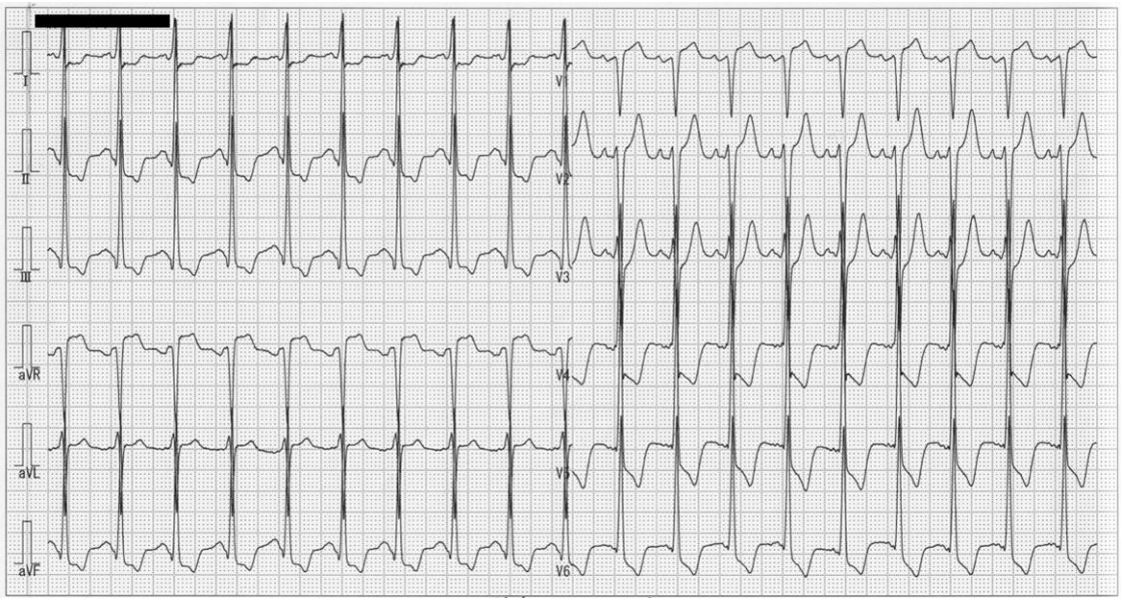

Figure 1 The 12-lead electrocardiogram of the patient on admission. The ST-segment depression on the anterior and inferior leads with sinus tachycardia.

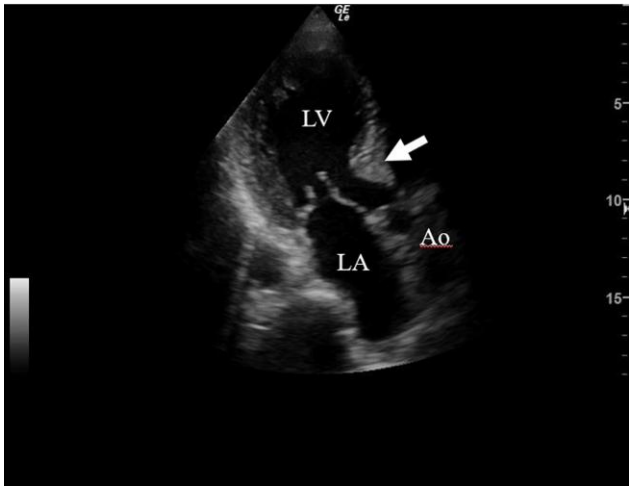

Figure 2 Apical three-chamber view of the initial transthoracic echocardiogram. The hypertrophic myocardial septum on the left ventricular outflow tract with 15.6 mm of its thickness revealed (white arrow). LV, left ventricle; LA, left atrium; Ao, aorta.

and cibenzoline were administered to decrease the pressure gradient of the LVOT, as real-time improvement of the subaortic pressure gradient with these medications has been observed using TTE.[5] No reports have described the concurrent efficacy of intravenous beta-blockade and Class 1A anti-arrhythmic drugs on left ventricular outflow obstruction based on cardiac catheterization recordings. However, in this case, an angiographic catheter with pressure wire showed a visible reduction in the subaortic pressure gradient from the administration of these drugs. Furthermore, improvement in the MR was visualized by TTE after drug administration.

The diagnostic strategy in this case had the following novel features, only one radial arterial puncture and fewer provoked PVCs. Traditionally, two different catheters are used to record pressures across the aortic valve. For example, we would classically place a pigtail catheter in the left ventricle and a coronary angiographic

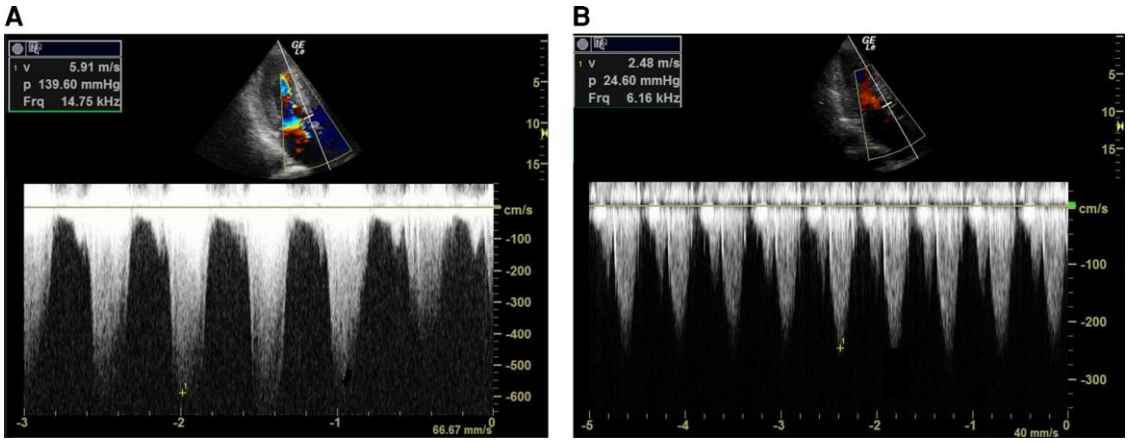

Figure 3 (*A*) The initial pulse wave doppler ultrasound of the left ventricular outflow tract. The maximum peak pressure gradient was measured at 139.6 mmHg. (*B*) The pulse wave doppler ultrasound of the left ventricular outflow tract after intravenous propranolol and cibenzoline. The maximum peak pressure gradient decreased to 24.6 mmHg.

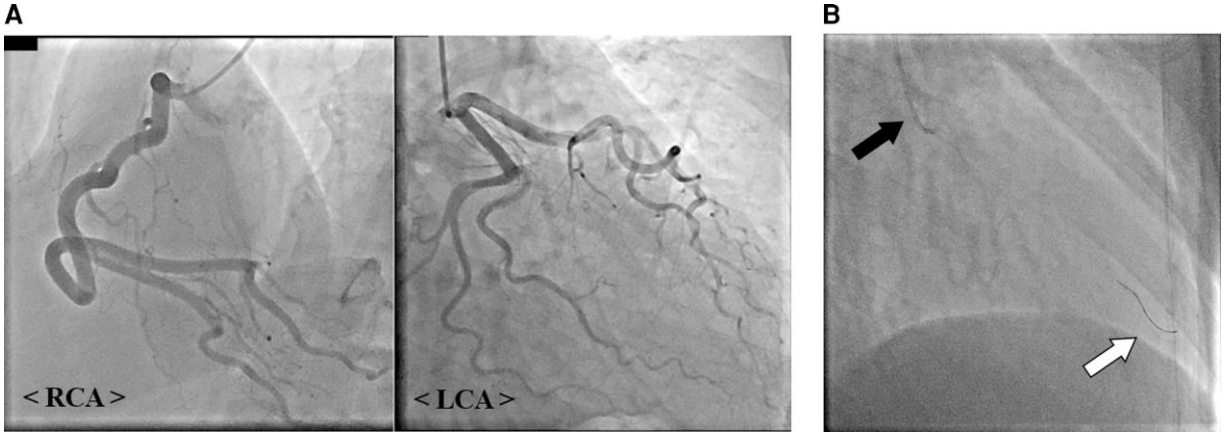

Figure 4 (*A*) The initial coronary angiography. Coronary artery disease was not found. RCA, right coronary artery; LCA, left coronary artery. (*B*) The present method of recording continuous subaortic pressure gradient under fluoroscopic guidance from right anterior oblique view. An angiographic diagnostic catheter (Goodtech 5Fr-Judkins Right 4.0, Goodman Medical Ireland Limited, Ireland: black arrow) was placed above the aortic valve and a pressure wire (PressureWire™ X, Abbott, USA: white arrow) was placed in the left venticle.

catheter in the aorta, and they would be inserted from different arterial puncture sites. In contrast, the present method allowed for completion of catheterization with one radial arterial puncture site. Reduced puncture sites and a radial artery puncture might lead to decreased complications of catheterization.[6] Therefore, the present method may be helpful in preventing complications. Finally, compared with the classical method, the pressure wire provoked fewer PVCs than the catheter placed in the left ventricle. This might be because the pressure wire has a thinner and softer structure, thus applying less stimuli on the myocardium than the catheter. Thus, this method led to a steady complete recording of the subaortic pressure gradient during cardiac catheterization.

Conclusion

Dynamic improvement in the acutely increased subaortic pressure gradient was treated with intravenous propranolol and cibenzoline and was clearly recorded by cardiac catheterization. This method required using a pressure wire through an angiographic catheter and allowing for completion of catheterization with a single arterial puncture led to a

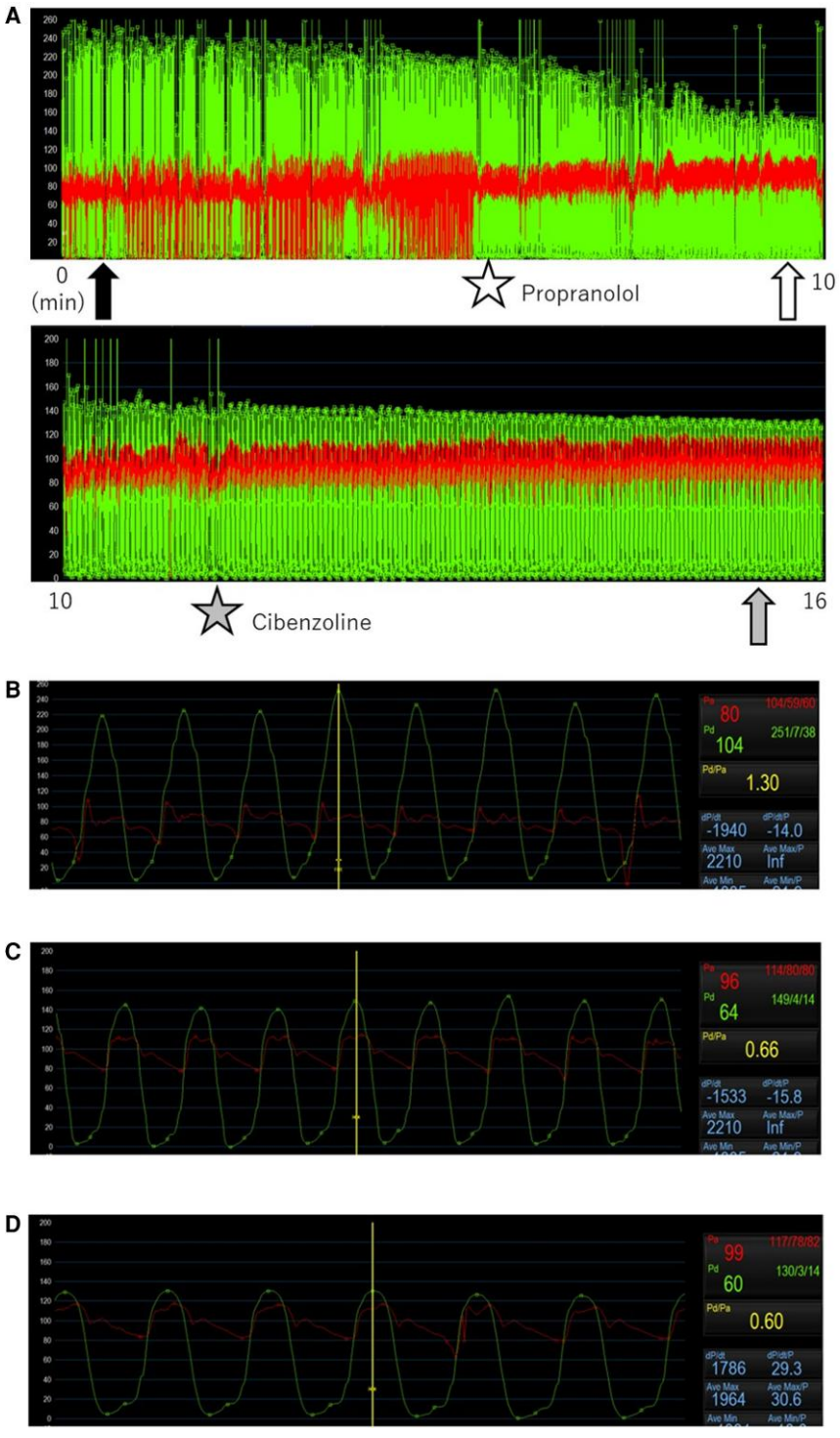

Figure 5 (*A*) The whole, stable, and simultaneous recording of pressures at the aorta (red wave) and the left ventricle (green wave) with an angiographic diagnostic catheter and a pressure wire. Initially, a high subaortic pressure gradient was observed as shown in *Figure 5B* (black arrow). After administering intravenous propranolol (white star), the pressure gradient was reduced to ~30 mmHg as shown in *Figure 5C* (white arrow). Additional cibenzoline was administrated (grey star), decreasing the pressure gradient further as shown in *Figure 5D* (grey arrow). (*B*) Pressures at the aorta (red wave) and the left ventricle (green wave) simultaneously recorded by an angiographic diagnostic catheter and a pressure wire. The representative point (on the yellow line) showed an aortic pressure of 104/59 mmHg (red figures) and a left ventricular pressure of 251/7 mmHg (green figures). (*C*) The simultaneous pressures after administering 4 mg propranolol. The representative point (on the yellow line) showed that the subaortic pressure gradient decreased to 35 mmHg (114 mmHg, aorta; 149 mmHg, left ventricle). (*D*) The simultaneous pressures after administering 70 mg cibenzoline. The representative point (on the yellow line) showed that the subaortic pressure gradient decreased to 13 mmHg (117 mmHg, aorta; 130 mmHg, left ventricle).

steady recording of the subaortic pressure gradient; therefore, it may be advantageous over traditional procedures.

Lead author biography

Yoshihiro Harano is a general cardiologist who graduated from Gifu University School of Medicine, Gifu, Japan, in 2018. Since 2021, he has worked at Gifu Heart Center (Gifu, Japan) as a senior resident with specific interest in catheter intervention. Memberships: 2019 The Japanese Society of Internal Medicine, 2019 The Japanese Heart Rhythm Society, 2020 The Japanese Circulation Society, and 2021 The Japanese Association of Cardiovascular Intervention and Therapeutics.

Supplementary material

Supplementary material is available at the *European Heart Journal – Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementarydata.

Consent: The authors confirm that written consent for submission and publication of this case report, including images and associated text, has been obtained from the patient in line with COPE guidance.

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References

1. Maron BJ, Rowin EJ, Udelson JE, Maron MS. Clinical Spectrum and management of heart failure in hypertrophic cardiomyopathy. *JACC Heart Fail* 2018;**6**:353–363.
2. Paz R, Jortner R, Tunick PA, Sclarovsky S, Eilat B, Perez JL, Kronzon I. The effect of the ingestion of ethanol on obstruction of the left ventricular outflow tract in hypertrophic cardiomyopathy. *N Engl J Med* 1996;**335**:938–941.
3. Sherrid MV, Shetty A, Winson G, Kim B, Musat D, Alviar CL, Homel P, Balaram SK, Swistel DG. Treatment of obstructive hypertrophic cardiomyopathy symptoms and gradient resistant to first-line therapy with β-blockade or verapamil. *Circ Heart Fail* 2013;**6**:694–702.
4. Sherrid MV, Barac I, McKenna WJ, Elliott PM, Dickie S, Chojnowska L, Casey S, Maron BJ. Multicenter study of the efficacy and safety of disopyramide in obstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2005;**45**:1251–1258.
5. Tokudome T, Mizushige K, Ueda T, Sakamoto S, Matsuo H. Effect of disopyramide on left ventricular pressure gradient in hypertrophic obstructive cardiomyopathy in comparison with propranolol–a case report. *Angiology* 1999;**50**:331–335.
6. Bhat FA, Changal KH, Raina H, Tramboo NA, Rather HA. Transradial versus transfemoral approach for coronary angiography and angioplasty—A prospective, randomized comparison. *BMC Cardiovasc Disord* 2017;**17**:23.