Alcohol septal ablation (ASA) has a high rate of residual left ventricular outflow tract obstruction (LVOTO) in patients with hypertrophic obstructive cardiomyopathy (HOCM). We successfully treated a patient with severe residual LVOTO after ASA by percutaneous intramyocardial septal radiofrequency ablation (PIMSRA), or Liwen procedure, showing favorable gradient decline, functional recovery, and no relevant complications after procedure. This intervention may provide a new possibility for treating HOCM with failed ASA.

A 53-year-old woman complained of recurrent dizziness and chest pain while climbing stairs or walking fast; the symptoms had been present for more than 2 years and had worsened in the last 3 months. Two years ago, the patient was diagnosed with HOCM and underwent ASA at our hospital. After administration of metoprolol, her resting heart rate (HR) remained under 60 bpm. Echocardiography showed that the thickest interventricular septum (IVS) was 19.6 mm, and maximal left ventricular outflow tract (LVOT) pressure gradient (LVOT-PGmax) was 31 mm Hg in a resting state (HR = 57 bpm) and 76 mm Hg under Valsalva maneuver (HR = 55 bpm), which were similar to the echocardiographic performance before ASA with thickest IVS 18.0 mm and LVOT-PGmax 76 mm Hg at rest (HR = 72 bpm). Treadmill exercise echocardiography found the LVOT-PGmax even reached 158 mm Hg. Myocardial contrast echocardiography (MCE) further demonstrated that the myocardium of the middle and upper IVS was thickened and the myocardial activity was normal. After a flash with high MI (MI = 1.22, flash frame = 10), abundant reperfusion signals appeared rapidly in the IVS.

The process of ablation: adjust the direction of the needle to the electrical impedance of the myocardium. The heated myocardium around the tip presents a hyperechoic mass as indicated by the arrows.

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septal contact location during mitral systolic anterior motion (Figure 1). In addition, we reviewed the previous ASA from 2 years ago and found the operator injected 1.8 mL alcohol into the distal segment of the second septal branch but that there was extensive collateral circulation. In sum, it could be concluded that the ASA procedure failed to induce sufficient infarcted IVS area, resulting in persistent severe LVOTO.

The patient refused to undergo surgical myectomy; therefore we decided to perform PIMSRA to alleviate her symptoms. Under the guidance of real-time echocardiography, a radiofrequency electrode needle gently punctured the right ventricular apex of the chest wall and into the basal segment of the IVS (Video 2). To describe the ablation area more accurately, the IVS was artificially divided into zones 1, 2, 3, and 4 counterclockwise and evenly in the short-axis view.

Figure 1 (A) Pre-PIMSRA echocardiography showed thickened IVS on the parasternal long-axis view. (B) The LVOT-PGmax at Valsalva maneuver was 76 mm Hg. (C) The LVOT-PGmax under maximal treadmill exercise tolerance was 158 mm Hg. (D) The pre-PIMSRA CCTA showed large septal branches. (E) The CMR demonstrated nearly transmural (certainly >75%) late gadolinium enhancement with an endocardial base on the right ventricular side of the septum. The location is distal to the mitral-septal contact location during systolic anterior motion. (F) The MCE demonstrated abundant ventricular septal blood supply on the apical 4-chamber view.

Figure 2 The process of PIMSRA. (A) Lower the color signal scale to ensure the guideline avoids the coronary artery and the right ventricular cavity. (B) The needle was inserted along the guideline to the central part of IVS (arrows indicate the needle artifact). The posterior (zones 3(C) and 4(D)) and anterior (zones 1(E) and 2(F)) septum were ablated successively from the basal part to the middle part (arrows indicate the needle artifact).
from the junction with the anterior wall. The posterior (zones 3 and 4) and anterior (zones 1 and 2) septa were ablated successively from the basal to the middle part, with an ablation power of 25 to 40 W (Video 3). After 2 hours of operation, the ablation area and surrounding edema reached approximately 30 to 40 mm along the long axis (extending distally to 10 mm beyond the mitral-septal contact point) and 30 to 40 mm wide along the short axis (accounting for 2/3 of the thickness of the IVS; Figure 2). Immediately after ablation, the LVOT-PGmax decreased to 35 mm Hg. There was no obvious bundle branch block, atrioventricular block, or ventricular arrhythmias, and MCE showed a 4.49 cm² filling defect along the long axis of the IVS (Figure 3, Video 4).

During a 1-month follow-up visit, a repeat transthoracic echocardiogram demonstrated the septal thickness had shrunk to 16.0 mm and LVOT-PGmax had also dropped to 5 mm Hg at rest (HR = 66 bpm) and 7 mm Hg at Valsalva (HR = 53 bpm; Figure 3). We made a telephone follow-up at 6 months after PIMSRA and learned that the patient was in good health and denied chest pain, syncope, or movement restrictions. An electrocardiogram showed no arrhythmia, and echocardiography did not report an increase in LVOT velocity, although no measurements were provided.

DISCUSSION

Severe LVOTO not only causes symptoms such as chest pain and dyspnea but is a strong independent predictor of mortality and progression to heart failure. Even though ASA has recently become an alternative treatment for surgical myectomy because of its minor invasive and less experiential dependence, it still has a high rate of reintervention due to residual LVOTO. We presented a case of reintervention following ASA by PIMSRA, providing a new possibility for treating such patients.

Alcohol septal ablation works by inducing iatrogenic myocardial infarction of the septum related to LVOTO, the so-called target septal zone. However, the blood supply of the septum is so abundant and highly variable and includes all other septal branches as well as the posterior descending artery, leading to an unsatisfactory infarcted area that is inadequate to cover the target septal zone. In this case, alcohol failed to be injected into the other septal branches, and there was rich collateral circulation. After ASA, the thick septal branches on CCTA, abundant perfusion on MCE, and unsatisfactory delayed enhancement region on CMR all implied that ASA was not adequate to eliminate blood supply to the basal septum. Moreover, mitral valve abnormalities are frequently observed in patients with HOCM, which may cause persistent systolic anterior motion and gradients after ASA, as in our patient.

Patients experience sternal pain after thoracotomy and are at risk for cognitive impairment from prolonged extracorporeal circulation procedures and often defer to less invasive options. Consequently, we decided to adopt PIMSRA for the patient, with full informed consent, which is a novel, minimally invasive, well-received, and effective procedure. We insert the radiofrequency needle through the chest wall tissue directly into the central myocardium of the septum, which avoids the conduct system, and perform ablation under the direct guidance of echocardiography, which allows a sufficient ablated area. A previous animal study showed that 12 months after the PIMSRA procedure, the thickness of the ablated myocardium decreased significantly as did the motion amplitude and the systolic wall thickening rate. A similar reduction in IVS thickness was also observed in HOCM patients 3 months after PIMSRA, with gradient decline, improvement in New York Heart Association classification, and acceptable complication rates. In our patient, the operation was successful, with favorable clinical effects and no significant complications, causing us to be optimistic about the long-term clinical outcome.

CONCLUSION

We successfully treated a patient with residual LVOTO after ASA by PIMSRA, with postoperative favorable gradient decline, functional recovery, and no relevant complications, indicating that PIMSRA may be an optional treatment for reintervention following failed ASA.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2022.06.003.

REFERENCES

1. Maron MS, Olivotto I, Betocchi S, Casey SA, Lesser JR, Losi MA, et al. Effect of left ventricular outflow tract obstruction on clinical outcome in hypertrophic cardiomyopathy. N Engl J Med 2003;348:295-303.
2. Liebregts M, Vriesendorp PA, Ten BJ. Alcohol septal ablation for obstructive hypertrophic cardiomyopathy: a word of endorsement. J Am Coll Cardiol 2017;70:481-8.
3. Fifer MA, Sigwart U. Controversies in cardiovascular medicine. Hypertrophic obstructive cardiomyopathy: alcohol septal ablation. Eur Heart J 2011;32:1059-64.

4. Spirito P, Rossi J, Maron B. Alcohol septal ablation: in which patients and why? Ann Cardiothorac Surg 2017;6:369-75.

5. Yang Q, Zhu C, Cui H, Tang B, Wang S, Yu Q, et al. Surgical septal myectomy outcome for obstructive hypertrophic cardiomyopathy after alcohol septal ablation. J Thorac Dis 2021;13:1055-65.

6. Sherrid MV, Adams DH. The mitral valve in hypertrophic cardiomyopathy: other side of the outflow tract. J Am Coll Cardiol 2020;76:2248-51.

7. Liu L, Liu B, Li J, Zhang Y. Percutaneous intramyocardial septal radiofrequency ablation of hypertrophic obstructive cardiomyopathy: a novel minimally invasive treatment for reduction of outflow tract obstruction. Eurointervention 2018;13:e2112-3.

8. Liu F, Fu J, Hsi D, Sun C, He G, Hu R, et al. Percutaneous intramyocardial septal radiofrequency ablation for interventricular septal reduction: an ovine model with 1-year outcomes. Cardiology 2020;145:53-62.

9. Zhou M, Ta S, Hahn RT, Hsi DH, Leon MB, Hu R, et al. Percutaneous intramyocardial septal radiofrequency ablation in patients with drug refractory hypertrophic obstructive cardiomyopathy. JAMA Cardiol 2022;7:529-38.