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CASE REPORT

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MitraClip combined with PTSBME for the treatment of obstructive hypertrophic cardiomyopathy with severe mitral regurgitation: a case report

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Abstract

Background Hypertrophic cardiomyopathy (HCM) is the hereditary cardiomyopathy with the highest incidence rate. Its main pathological changes are ventricular septal myocardial hypertrophy and myocardial disorder, which are prone to fatal arrhythmia and heart failure. If left ventricular outflow tract (LVOT) obstruction is combined, it is called obstructive hypertrophic cardiomyopathy (oHCM). There is currently no report on the use of MitraClip combined with percutaneous transluminal septal branch microsphere embolization (PTSBME) for treating patients with oHCM complicated with severe mitral regurgitation (MR).

Case presentation This report describes a 51-year-old male patient who was admitted to the hospital due to "repeated chest tightness and shortness of breath for 2 years, worsening for 6 months". Ultrasound, left ventricular angiography (LVA), and left cardiac catheterization confirmed oHCM with moderate MR. We used MitraClip combined with PTSBME to relieve the patient's LVOT obstruction and MR simultaneously.

Conclusions Traditionally, both interventricular septal and mitral valve lesions are treated simultaneously through surgical intervention. However, the surgical conditions are relatively strict, and many patients are unable to undergo surgical treatment, resulting in delays in their condition. For such patients, minimally invasive intervention may be used to simultaneously treat interventricular septal and mitral valve lesions, further reducing surgical risks and enhancing surgical efficacy. In this case, MitraClip combined with PTSBME was first performed. After the surgery, the patient's LVOT obstruction and MR were simultaneously relieved, and clinical symptoms improved significantly.

Keywords Obstructive hypertrophic cardiomyopathy, Mitral regurgitation, MitraClip, Percutaneous transluminal septal branch microsphere embolization

Background

Hypertrophic cardiomyopathy (HCM) is the hereditary cardiomyopathy with the highest incidence rate [1]. Its main pathological changes are ventricular septal myocardial hypertrophy and myocardial disorder, which are prone to fatal arrhythmia and heart failure. Echocardiography or magnetic resonance imaging can confirm the presence of left ventricular outflow tract (LVOT) obstruction (LVOT pressure difference ≥ 30 mmHg, 1 mmHg = 0.133 kPa) if the wall thickness in any part of

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the left ventricular end-diastolic phase is ≥15 mm [2]. The focus of its treatment is on how to improve hypertrophic myocardium without affecting surrounding non-hypertrophic myocardium. Percutaneous transluminal septal branch microsphere embolization (PTSBME) is a new surgical technique that has emerged in recent years. It involves intermittent injection of microspheres to accurately occlude the target blood vessel and damage the target myocardium, reducing the damage to the surrounding non-target myocardium caused by leakage. However, in clinical practice, a large number of patients with HCM often have mitral valve disease. For patients with severe mitral valve disease, surgical interventricular septal myocardial resection combined with mitral valve repair is the first choice to improve ventricular septal hypertrophy and mitral valve disease. However, surgical procedures have disadvantages such as high trauma and high risk, which many patients cannot tolerate. The treatment of mitral valve disease through minimally invasive mitral valve clamping has gradually become a trend. However, there are currently no reports of MitraClip combined with PTSBME for the treatment of obstructive hypertrophic cardiomyopathy (oHCM). This article reports the successful treatment of a case of oHCM with severe mitral regurgitation (MR) using MitraClip combined with PTSBME. There is currently no report of the same procedure in domestic and foreign literature searches, aiming to provide a new treatment approach for this disease.

Case presentation

A 51-year-old man was admitted to hospital on April 29, 2024 due to worsening chest tightness, shortness of breath and lower limb weakness. The symptoms started over two years ago, were triggered by physical exertion and improved when he rested. In the last six months, the condition worsened considerably, with pronounced shortness of breath and dizziness occurring even with moderate activity and requiring frequent rest. An ultrasonic cardiogram (UCG) performed at an outside hospital revealed left ventricular wall thickening, LVOT obstruction, moderate to severe MR and tricuspid regurgitation. As drug treatment proved ineffective, the patient visited our outpatient clinic. After extensive examinations, the diagnosis of "HCM" was made and the patient was admitted to our department for further treatment. History of coronary heart disease, long-term treatment with oral aspirin and statins; Deny a history of infectious diseases such as hepatitis and tuberculosis. Deny any history of drug or food allergies. Deny surgical history. Deny history of blood transfusion. Deny a family history of HCM. Physical examination: BP 111/68 mmHg, SPO2 98%, Clear breathing sounds in both lungs, no dry or wet rales in both lungs or lung soles. Heart rate is 80 beats per minute, with a regular rhythm. There may be 3/6 grade systolic jet murmurs between the 3–4 intercostal spaces on the left edge of the sternum, and 3/6 grade systolic blowing murmurs in the auscultation area of the mitral valve (MV). There is no edema in both lower limbs. There are no significant abnormalities in BNP, troponin, three major routine tests, liver and kidney function, electrolytes, coagulation function, and thyroid function. Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) are consistent with the sonographic changes of asymmetric oHCM: uneven thickening of the interventricular septum (IVS), obvious basal segment, thickest point about 17.9 mm (Fig. 1A: transthoracic) /18.8 mm (Fig. 1B: transesophageal), protruding to the LVOT, causing LVOT stenosis. The peak pressure difference of LVOT in the resting state is 58 mmHg (Fig. 1C), and the peak pressure difference of LVOT in the stimulation test state is 94 mmHg (Fig. 1D). The length of the anterior leaflet of the MV is 24.6 mm, the length of the posterior leaflet is 10.1 mm, the MV orifice area is 5.43 cm^2, and the MV has moderate regurgitation (Fig. 2A). At the end of systole, the anterior leaflet of the MV is almost completely close to the basal part of the IVS, causing LVOT stenosis. Almost complete obstruction with obvious SAM sign (Fig. 2B), EF 66%. LVA + pressure measurement examination: During systole, the apex and basal ganglia of the left ventricle are significantly compressed, and the pressure difference between the apex and outflow tract of the left ventricle is 80–90 mmHg; Complies with changes in oHCM. Coronary angiography (CA): Localized stenosis of 40–50% in the proximal segment of the anterior descending artery (LAD), no significant stenosis in the left main trunk, circumflex branch, and right coronary artery, with a relatively large first septal branch (Fig. 3). Treatment with drugs such as diltiazem and trimetazidine did not show any improvement in symptoms. After comprehensive evaluation by the structural heart disease team, the main causes of LVOT obstruction and severe MR in this patient were thickening and protrusion of the IVS to the LVOT and elongated anterior leaflet of the MV, as well as SAM syndrome. Therefore, the key to treatment lies in resolving LVOT obstruction and addressing abnormalities in MV structure. At present, the guidelines recommend surgical interventricular septum (IVS) myocardial resection combined with MV repair as the preferred treatment option for such patients. However, due to the clear refusal of the patient and their family to undergo surgical treatment, the heart valve team decided to use MitraClip combined with PTSBME for treatment. MitraClip can clamp the anterior and posterior leaflets of the MV, restrict its movement, relieve the obstruction of the

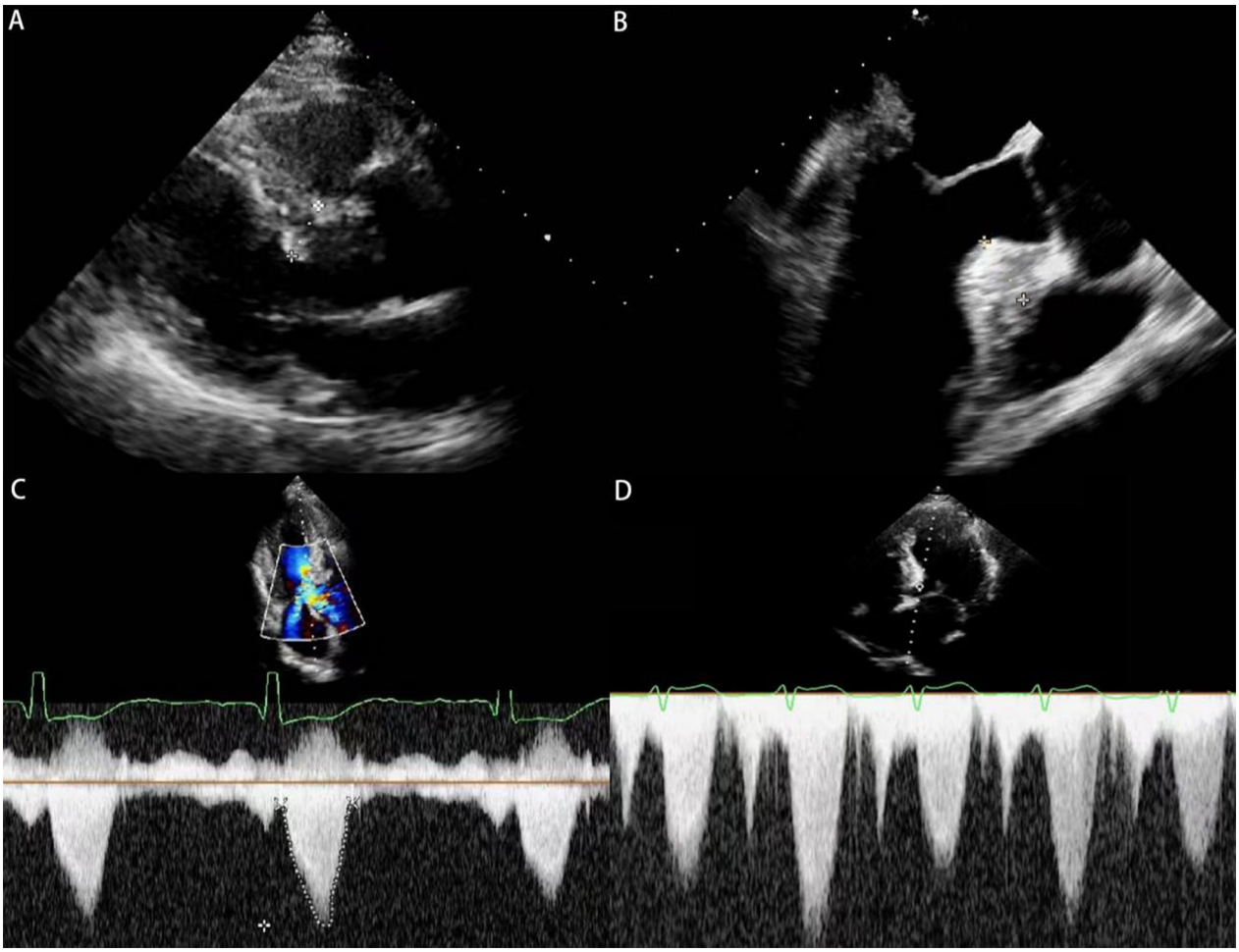

Fig. 1 Preoperative evaluation of interventricular septal branch and LVOT **A** Measurement of basal thickness of interventricular septal branch by TTE **B** Measurement of basal thickness of interventricular septal branch by TEE **C** Peak LVOT pressure difference at rest 58 mmHg **D** Peak LVOT pressure difference at stimulation test 94 mmHg

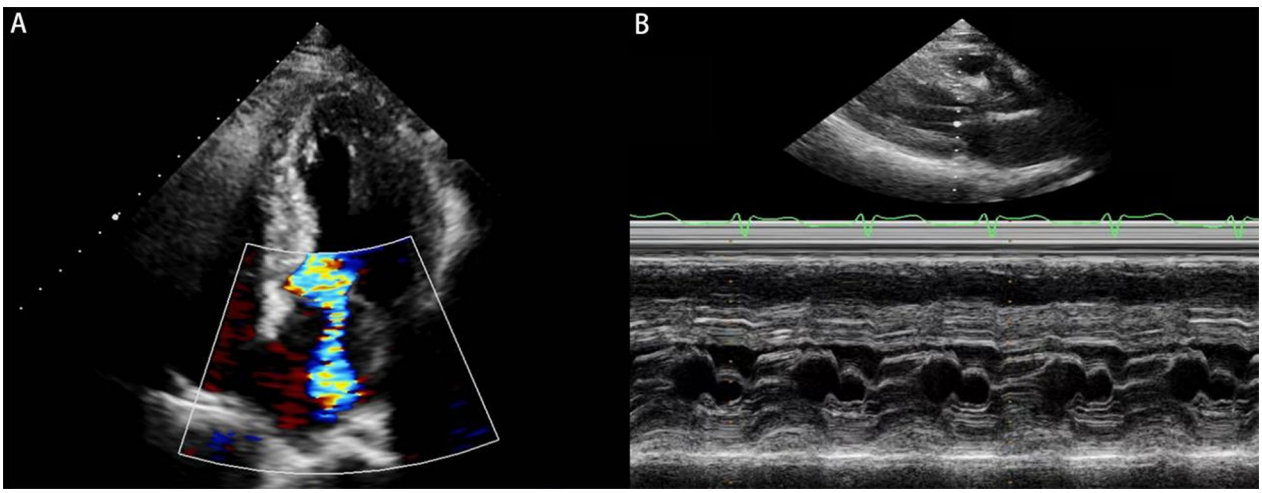

Fig. 2 Preoperative evaluation of MR A Moderate MR B SAM sign

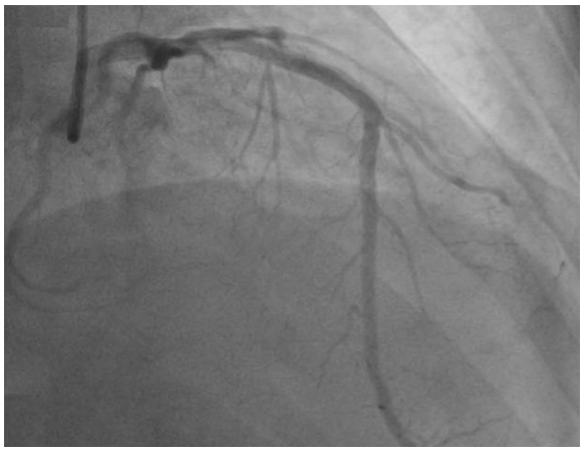

Fig. 3 Mild coronary artery disease with a relatively large first septal branch

LVOT during systole, and reduce the area of the MV orifice. PTSBME, under the guidance of selective contrast-enhanced ultrasound, embolizes the first septal branch to reduce the amplitude of interventricular motion and local IVS thickness, thereby treating LVOT obstruction and severe MR.

After general anesthesia and tracheal intubation, the patient inserts TEE for backup and first uses microsphere particles for IVS branch embolization. CA shows a relatively large first IVS branch. The SION blue guidewire was successfully delivered to the distal segment of the thick IVS, and a microcatheter was inserted along the guidewire to the middle segment of the IVS branch. Under the guidance of TEE, selective contrast-enhanced ultrasound was performed through the microcatheter to confirm the effectiveness of the proposed embolization of the first IVS branch. Intravenous infusion of isoproterenol to achieve a heart rate of over 100 beats per minute for stimulation test: blood pressure significantly decreased to 60/40 mmHg, and LVOT peak pressure difference was 110 mmHg. A microcatheter was used to deliver a 2.0 * 20 mm OTW balloon to the proximal segment of the IVS branch after exchanging with a SION blue guidewire. After 8 atm expansion of the OTW balloon, approximately 6 ml of microsphere fluid was injected into the OTW balloon for embolization. After embolization, TEE re-measured a significant decrease in outflow velocity, and CA showed normal blood flow in the anterior descending branch and grade 0 blood flow in the IVS branch. Selective ultrasound contrast showed reduced amplitude of hypertrophic myocardial motion and no contrast agent echo, confirming the effectiveness of embolization. Esophageal ultrasound showed significant MR and the SAM sign did not disappear. An intravenous infusion of isoproterenol was administered to achieve a heart rate of 100 beats per minute or above for stimulation testing. Blood pressure significantly decreased to 60/40 mmHg, and LVOT peak pressure was 90 mmHg. After discussion with the structural heart disease team, further MV clamping was decided. Under the guidance of TEE, the atrial septum is punctured and the MitraClip XTR shaping clip delivery system is used for MV clamping. The MitraClip XTR shaping clip delivery system is adjusted under ultrasound guidance to point towards the most obvious MV regurgitation and can move vertically. The arms of the MitraClip shaping clip are opened to 120°, and the MitraClip XTR shaping clip is adjusted under the guidance of TEE to be located in the middle of the anterior and posterior leaflets of the MV. The MitraClip XTR shaping clip is further adjusted to the outer boundary of the MV annulus under real-time 3D ultrasound surgical field of view, and the arms are placed at 12:30 and 6:30. During diastole, the MitraClip XTR is slowly withdrawn into the ventricular cavity. Form the clamp and place both leaflets on the two arms of the MitraClip forming clamp. Operate the MitraClip XTR forming clamp to clamp both valve tips, Repeated confirmation of minimal MR via TEE and subsequent stimulation tests confirmed an effective heart rate of over 100 beats per minute, a blood pressure of around 90/60 mmHg, and a transvalvular pressure gradient of 50 mmHg. Finally, release the MitraClip XTR forming clip. Further, CA showed no significant abnormalities in the LAD and right coronary artery, and the bedside electrocardiogram confirmed no significant conduction block or acute myocardial infarction in the anterior and inferior walls.

After one month follow-up after discharge, the patient's chest tightness and shortness of breath symptoms improved compared to before, heart function improved compared to before, activity tolerance increased compared to before, and the six-minute walking test increased from 240 m before surgery to 480 m. Re-examination of UCG showed a significant reduction in thickening of the IVS base, approximately 14 mm (Fig. 4A), with a peak LVOT pressure difference of 14 mmHg (Fig. 4B). Cardiac MRI confirmed a reduction in IVS hypertrophy (Fig. 4C), and ultrasound showed a decrease in MR compared to before (Fig. 4D), with an EF of 66%.

Discussion

HCM is an autosomal dominant genetic disease characterized by asymmetric left ventricular hypertrophy [1]. According to the presence or absence of LVOT obstruction, it is divided into oHCM and non-oHCM. HCM is the hereditary cardiomyopathy with the highest incidence rate at present. In the 1990s, foreign statistics found that the prevalence of HCM in adults was about

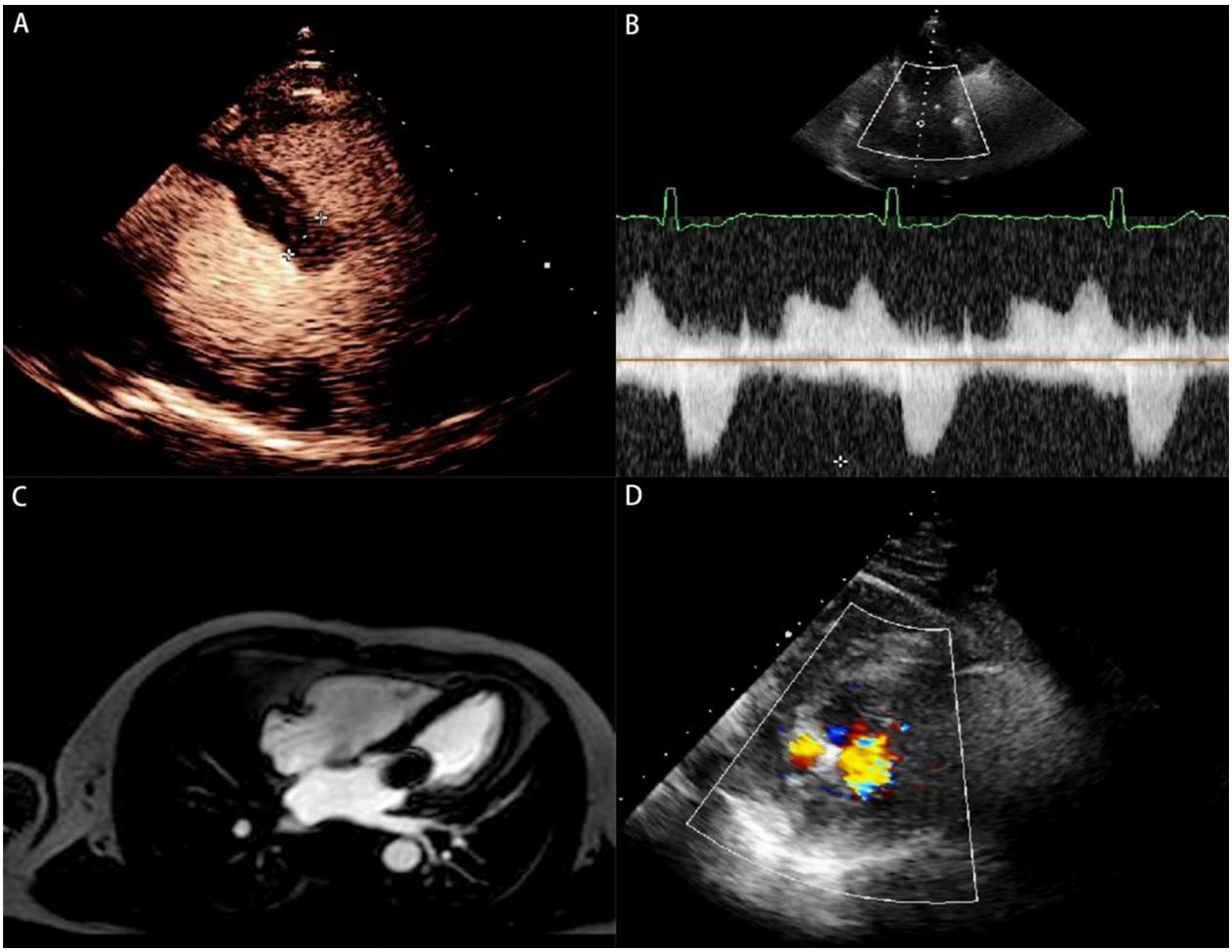

Fig. 4 Postoperative evaluation of interventricular septal thickness, LVOT obstruction and MR. **A** UCG shows significant reduction in basal interventricular septal hypertrophy. **B** Resting LVOT peak pressure drops to 14 mmHg. **C** MRI evaluation shows significant reduction in basal interventricular septal hypertrophy. **D** UCG shows significant reduction in MR

1/500. In recent years, with the development of clinical and molecular genetics, as well as the improvement of diagnostic techniques, the estimated prevalence of HCM is at least 1/200 [2]. The main pathological changes are IVS myocardial hypertrophy and disordered myocardial arrangement, which can easily lead to fatal arrhythmias and heart failure, and are a common cause of sudden cardiac death in adolescents [3]. Excessive IVS hypertrophy leads to LVOT obstruction and systolic MV shift (SAM), further exacerbating left ventricular burden [4]. Therefore, traditional treatments mainly target myocardial hypertrophy. Including drug therapy and surgical treatment. For oHCM with poor drug efficacy, surgical treatment is often used to relieve outflow tract obstruction [5]. The traditional surgery is partial resection of the IVS. With the advancement of technology, minimally invasive intervention therapy for IVS has gradually become a trend. At present, common techniques include alcohol septal ablation (ASA), percutaneous intramyocardial septal radiofrequency ablation (PIMSRA), Stereotactic cyber knife treatment, surgical septal myectomy (SSM), et al. In 1995, the use of anhydrous alcohol for chemical ablation of the interventricular septal branch was first reported. Injecting anhydrous alcohol into the interventricular septum caused local coagulative necrosis of the myocardium. As the myocardial tissue was absorbed, the septal myocardium became thinner, thereby reducing the outflow tract pressure gradient [6]. Subsequently, ASA became one of the main surgical procedures for the treatment of HCM. However, due to its liquid nature, anhydrous alcohol is difficult to control leakage and can easily damage non-target areas of the myocardium. Particle ball embolization is a new type of material that has emerged in recent years, which uses intermittent projectile injection to keep it in the target blood vessel,

reducing complications caused by leakage and thus reducing surgical risks [7].

In this case, a relatively large septal branch was first identified under CA. Under the guidance of TEE, selective contrast-enhanced ultrasound was performed through a microcatheter to further confirm the target ventricular septal branch. During the operation, TEE was used to measure the outflow tract velocity, and Isoproterenol was used for the provocation test. Then, about 2 ml of microsphere particle solution was injected into the septal branch for embolization. The LVOT velocity was significantly reduced by retesting, confirming the effectiveness of the treatment. However, subsequent repeated measurements using ultrasound still indicated moderate MR, significant SAM sign, and a high-pressure gradient in the outflow tract, suggesting that the anterior leaflet of the mitral valve moved forward during systole, exacerbating outflow tract obstruction. After Mitral Clip clamp treatment, the pressure gradient significantly decreased, MR disappeared, and postoperative follow-up symptoms improved significantly. UCG and magnetic resonance imaging also showed good results.

The mechanism of mitral valve disease in patients with HCM is not fully understood. The current mainstream view is that it is caused by a combination of factors such as abnormal left ventricular hemodynamics and primary mitral valve disease [8]. Firstly, LVOT stenosis and increased blood flow velocity create relative negative pressure in the outflow tract, leading to anterior displacement of the mitral valve; Secondly, the mitral valve, including lesions below the chordae tendineae, also plays an important role, such as disordered arrangement of the papillary muscles, compression of the chordae tendineae by the interventricular septal branch during myocardial contraction, migration of the anterior leaflet of the mitral valve, leading to SAM sign and MR. Obstruction of the LVOT promotes MR, while mitral valve disease in turn exacerbates LVOT obstruction. Research has shown that anterior papillary muscle displacement directly contacts the interventricular septal branch during left ventricular contraction, leading to LVOT obstruction; Abnormal insertion of papillary muscles and chordae tendineae into the leaflets can also obstruct by pulling the mitral valve to protrude into the LVOT [9]. Therefore, the treatment focus of HCM is to alleviate LVOT obstruction and MR. According to reports, sufficient interventricular septal volume reduction is sufficient to improve LVOT obstruction and SAM signs in the absence of severe primary mitral valve disease [10]. For patients with severe MR, mitral valve surgery can be used to improve MR [11]. In clinical practice, for patients with moderate to severe MR, many surgeons tend to perform mitral valve surgery based on interventricular septal branch resection. Afanasyeva et al. conducted a meta-analysis and found that the combined treatment of mitral valve repair for HCM with MR has significant advantages in reducing the risk of patient mortality, thromboembolism, postoperative mitral valve dysfunction, and reoperation [12]. However, surgical procedures are difficult for elderly and high-risk populations. The emergence of transcatheter edge-to-edge repair (TEER) surgery provides a new option for patients with HCM, reducing MR and outflow tract obstruction by restricting mitral valve activity [13]. In 2014, Schafer et al. reported a case of oHCM with recurrent LVOT obstruction and moderate MR 2 years after ventricular septal myocardial resection combined with mitral valve repair. Mitral Clip was used to reduce MR while limiting the anterior leaflet systolic shift of the mitral valve, thereby eliminating SAM and relieving LVOT obstruction [14]. This also confirms that improving MR can alleviate LVOT obstruction. Nakano et al. reported a case of an HCM patient undergoing ASA, which resulted in severe myocardial edema with mitral valve pseudoprolapse and severe MR after surgery. Subsequently, their team used MitralClip surgery to improve MR and achieved good therapeutic effects [15].

It can be seen that many patients with HCM who undergo interventricular septal surgery still have mitral valve surgery pointers after postoperative reassessment. Traditionally, both interventricular septal and mitral valve lesions are treated through surgical intervention. However, the surgical conditions are relatively strict, and many patients are unwilling to undergo surgical treatment, resulting in delayed treatment. For such patients, minimally invasive intervention may be used to simultaneously treat interventricular septal and mitral valve lesions, further reducing surgical risks and enhancing surgical efficacy. However, there are currently no reports of MitraClip combined with PTSBME for the treatment of HCM through interventional surgery.

Conclusion

This case first used PTSBME, and intraoperative monitoring showed relief of outflow tract obstruction, while SAM and MR still existed. After repeated evaluation, MitraClip surgery was used to improve MR. The patient's left ventricular LVOT obstruction and MR were relieved simultaneously after surgery, and clinical symptoms improved significantly. MitraClip combined with PTSBME is an effective solution for oHCM with moderate MR.

However, the current use of MitraClip for the treatment of MR in HCM is mostly a small sample study, and the longest follow-up time is only over 20 months, with unclear long-term efficacy. Therefore, when determining surgical strategies, it is necessary to repeatedly evaluate

the indications. And further exploration of larger sample clinical studies is needed in the future.

Abbreviations

HCM	Hypertrophic cardiomyopathy
LVOT	Left ventricular outflow tract
oHCM	Obstructive hypertrophic cardiomyopathy
PTSBME	Percutaneous transluminal septal branch microsphere embolization
MR	Mitral regurgitation
LVA	Left ventricular angiography
UCG	Ultrasonic cardiogram
TR	Tricuspid regurgitation
MV	Mitral valve
TTE	Transthoracic echocardiography
TEE	Transesophageal echocardiography
IVS	Interventricular septum
CA	Coronary angiography
LAD	Anterior descending artery
ASA	Alcohol septal ablation
PIMSRA	Percutaneous intramyocardial septal radiofrequency ablation
SSM	Surgical septal myectomy
TEER	Transcatheter edge-to-edge repair

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Author contributions
XW and ZL wrote the main manuscript and prepared figures,ML and SH drafted the initial manuscript, GP completed Critical review and revision. All authors reviewed the manuscript.

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Availability of data and materials
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
The patient agreed the doctors could use and publish her disease related article with personal information deleted.

Competing interest
The authors declare no competing interests.

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