LATE-ONSET LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION FOLLOWING MITRAL VALVE REPLACEMENT IN HYPERTROPHIC CARDIOMYOPATHY: DIAGNOSTIC AND THERAPEUTIC CHALLENGES

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Abstract

Hypertrophic obstructive cardiomyopathy is a genetic disease of the myocardium, characterized by symptoms of progressive heart failure, secondary to left ventricular outflow tract (LVOT) obstruction. Implantation of a prosthesis in the mitral position to address the valvular pathology will not improve the patient's clinical condition, who will continue to be symptomatic due to persistent intracardiac obstruction. Diagnosing LVOTO in patients with an implanted mitral prosthesis is often challenging with standard transthoracic echocardiography, but transesophageal echocardiography and cardiac CT imaging provide valuable diagnostic accuracy. Surgical intervention using a transverse aortotomy proved effective in this case, allowing direct visualization and resolution of obstruction without replacing the mitral prosthesis, consequently reducing operative time and avoiding unnecessary procedural risks.

CASE PRESENTATION

A 50 - year - old woman diagnosed with hypertrophic obstructive cardiomyopathy, history of mechanical prosthesis implantation in mitral position for severe regurgitation (2017), with recurrent episodes of atrial fibrillation, is admitted to our cardio-vascular unit in order to undergo septal myectomy . The patient was symptomatic by dyspnea on small exertion and constrictive chest pain, associating phenomena of congestive heart failure (NYHA class III heart failure). Our patient had also a history of persistent atrial fibrillation with radiofrequency ablation in 2023.

Physical examination on admission revealed a patient with good general condition , BMI 29 kg/ m2 , hemodynamically and respiratory stable. Cardiac auscultation noted well audible prosthetic clicks in the mitral area, third degree systolic murmur in the aortic area, mild ankle edema and no pulmonary crackles.

Current outpatient treatment included: Bisoprolol 5 mg/ day, Acenocumarol, Atorvastatin 20 mg/ day, Euthyrox 50 mcg/ day, Furosemide 40 mg/ day .

INITIAL WORK-UP

The electrocardiogram on admission revealed sinus rhythm, ventricular pacing rhythm , QRS duration 168 msec, PR interval 180 msec , QT duration 510 msec , major left bundle branch block (LBBB) .

Transthoracic preoperative X-ray revealed : transverse heart diameter at the upper normal limit, suture sternal cerclage wires, mechanic prosthesis in mitral position . No acute pleuro-pulmonary injuries were found.

Transthoracic preoperative echocardiography revealed non-dilated left ventricle (LV), with asymmetric severe hypertrophy, predominantly involving the interventricular septum [Fig 1], which had a maximum thickness of 30 mm, no wall motion abnormalities, but with apical rocking secondary to LBBB, normal LV systolic function . Right ventricle (RV) had normal function based on surrogate parameters. The mitral prosthesis had a normal function, with normal transprothetic gradients and an excess of pannus extending towards the LVOT, with subvalvular flow aortic acceleration[Fig 2]. Significant reduction of the LVOT diameter of only 11 mm was noted[Fig 1, 3]. Maximum resting LVOT velocity was 5.91 m/s, peak gradient of 146 mmHg and mean gradient 56 mmHg (at rest, no provocative manoeuvres) [Fig3] . Aortic valve was tricuspid and competent. Possible pulmonary hypertension was suspected and there was no pericardial effusion.

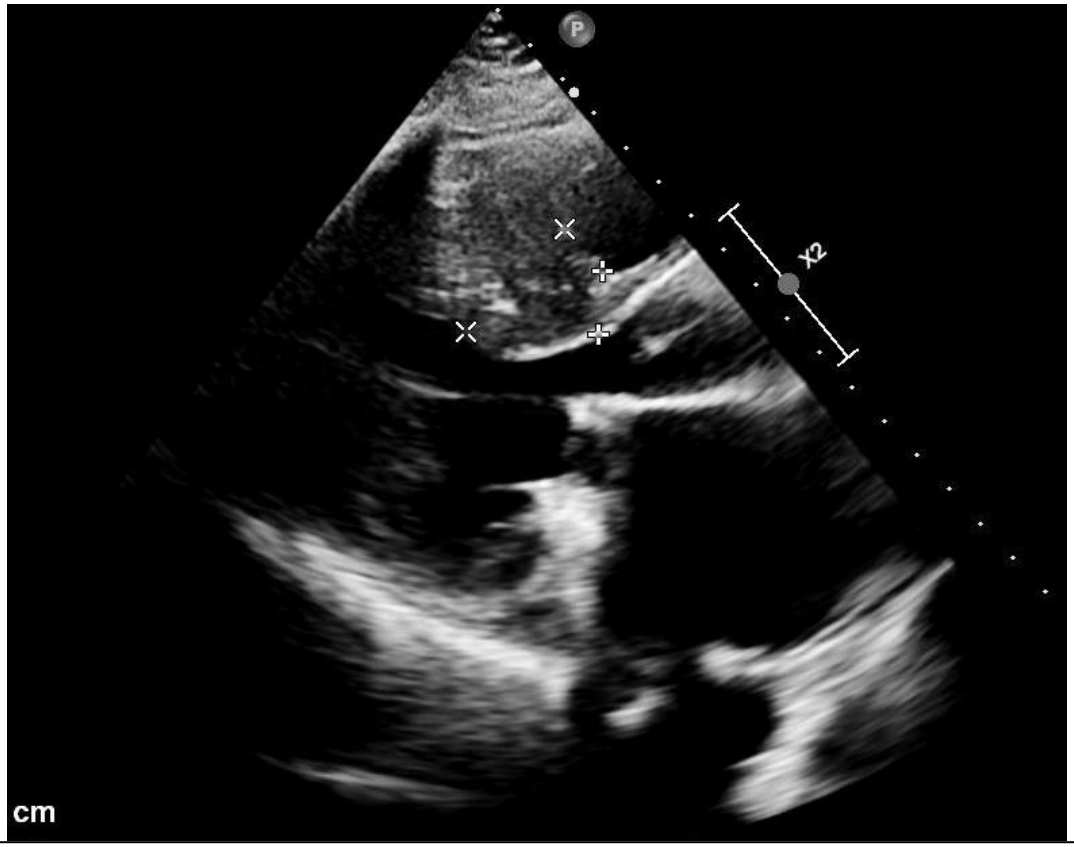

Fig 1. Severe interventricular septum hypertrophy with narrowing of the LVOT diameter

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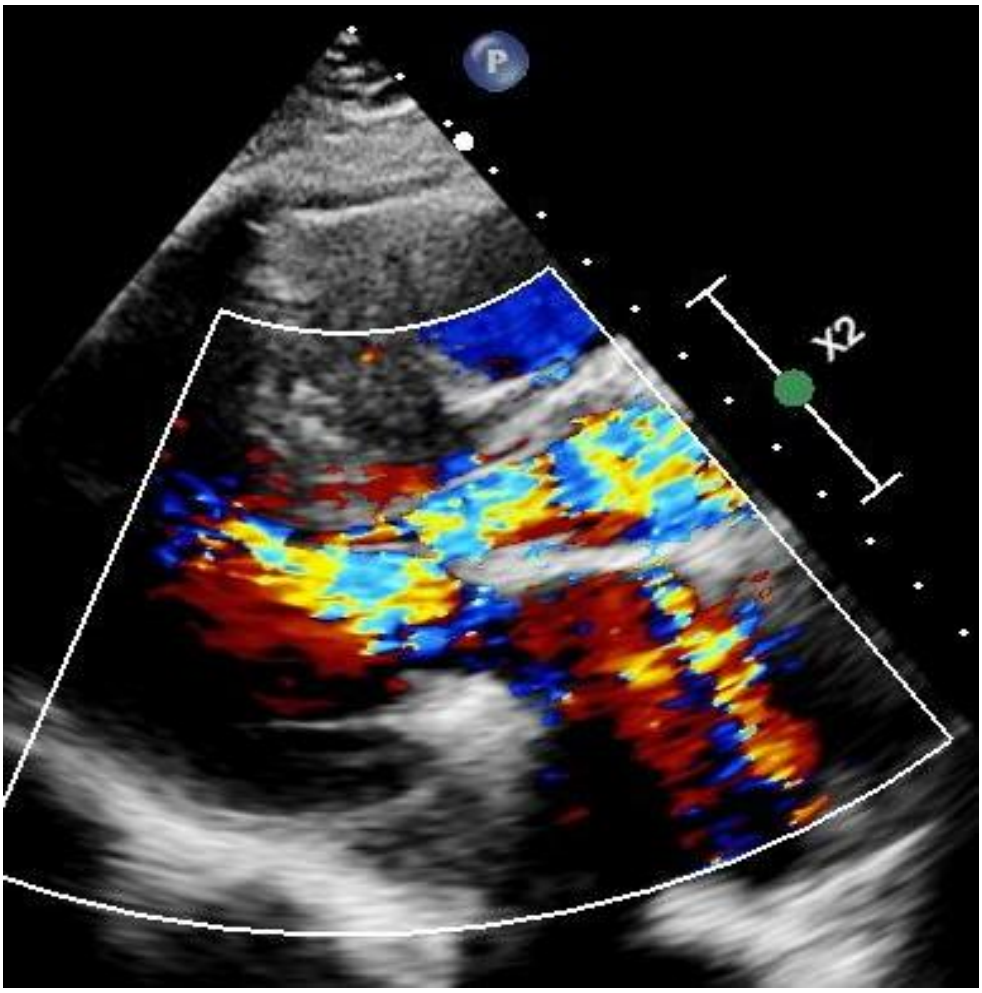

Fig 2: Color Flow Doppler shows LVOT flow turbulence due to LVOT obstruction

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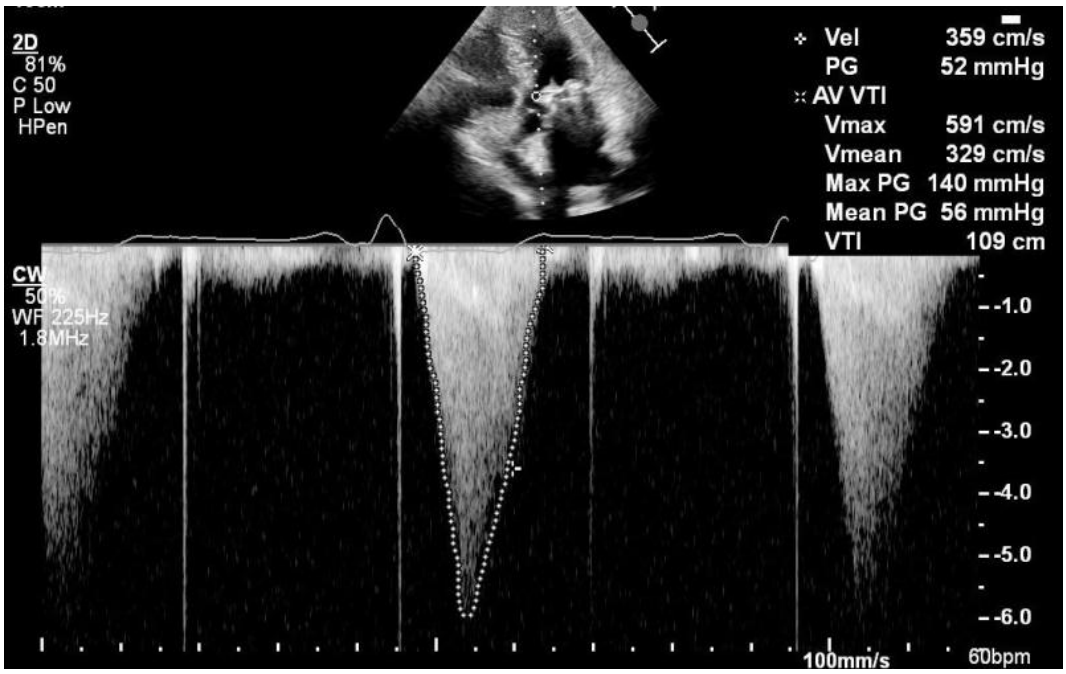

Fig 3. Continous flow Doppler: Peak and mean gradients at the LVOT level

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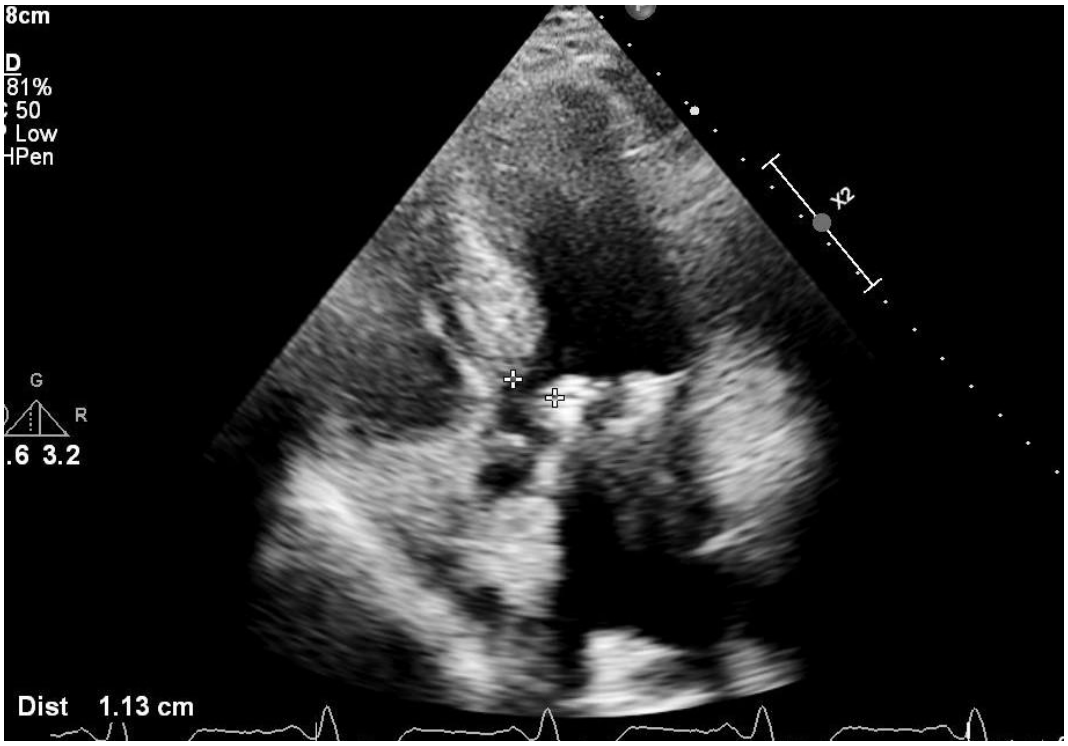

Fig 4. LVOT reduced diameter of only 11mm

Coronary angiography did not highlighted significant epicardial coronary stenoses. Respiratory tests (spirometry) were within normal limits. Abdominal ultrasound revealed mild hepatic steatosis and uncomplicated left renal cyst .

DIAGNOSIS AND MANAGEMENT

Based on clinical and paraclinical data, the working diagnoses were:

Severe obstructive hypertrophic cardiomyopathy with significant dynamic obstruction of the left ventricular ejection tract. NYHA class III heart failure. Bidisk mitral valve prosthesis implanted in 2017 with normal function .Radiofrequency ablation for persistent atrial fibrillation (2023). Dyslipidemia. Mild hepatic steatosis. Hypothyroidism on replacement therapy. Overweight .

After case review, the Heart Team recommended surgical septal myectomy , considering persistent symptoms and severe obstruction at the LVOT level, despite maximal medical treatment (class IB indication according to the ESC Guidelines).

Preoperative transesophageal echocardiography evaluated the mechanism of obstruction in the LVOT and also revealed flow acceleration and significant high gradients in the LVOT. Mitral valve disks had normal mobility and no intra or paraprosthetic leaks were recorded[Fig 5].

3D transesophageal echocardiography provided additional diagnosis, clearly demonstrating severe LVOT obstruction secondary to septal hypertrophy [Fig 6, 7] .

Fig 5:3D TEE: mitral prosthesis (atrial perspective): normal mobility of the disks which are full opened in diastole

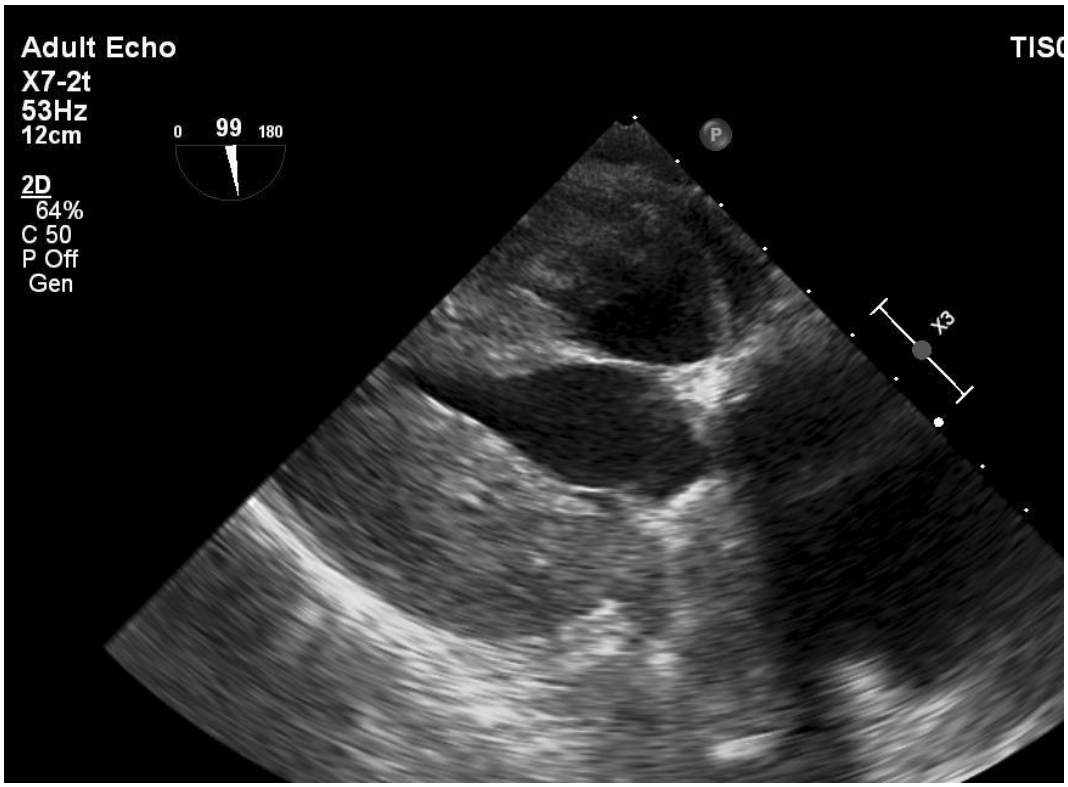

Fig 6: TEE Transgastric long axis view of the left ventricle demonstrates severe hypertrophy of the walls , small LVcavity

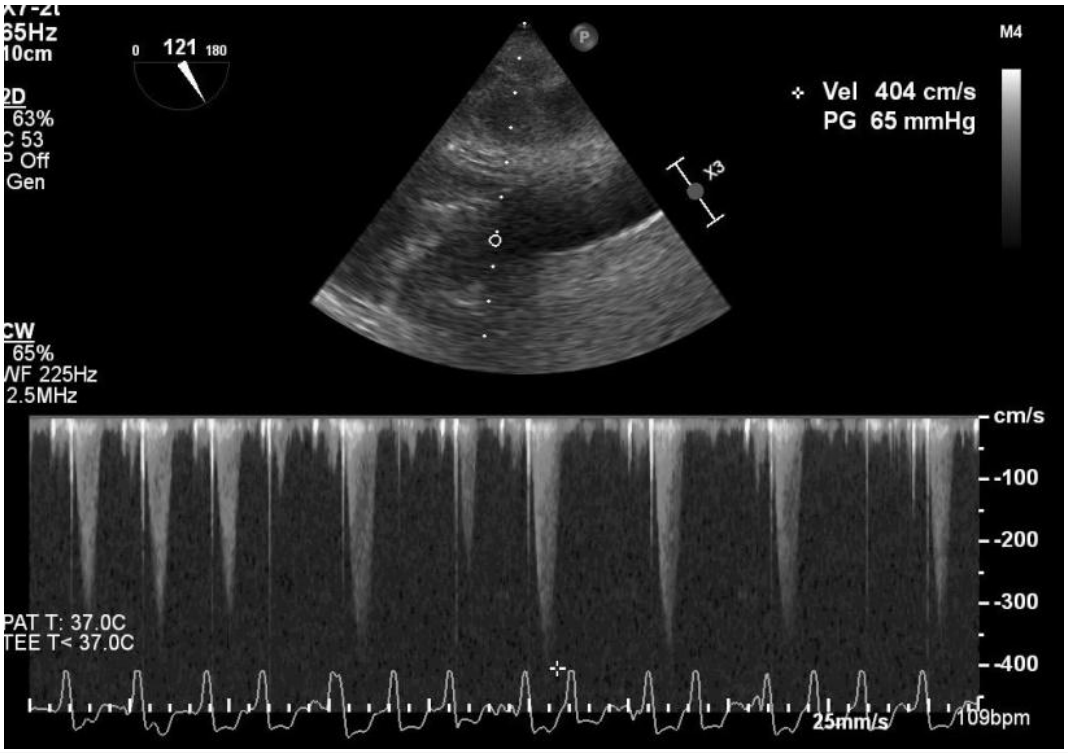

Fig 7. TEE deep transgastric view:continuous flow Doppler shows peak velocity of the flow in the LVOT of 4 m/s

Cardiac CT scan confirmed LVOT narrowing in diastole and turbulent flow at this level in systole [Fig 8,9].

The surgical intervention was performed via median sternotomy under extracorporeal circulation support. Following transverse aortotomy , intraoperative inspection revealed extensive endocardial circumferential fibrosis at the level of the LVOT, involving the anterior base of the mitral prosthesis , which has been anteriorly retracted by fibrotic pannus, also contributing to obstruction at this level. Resection of the fibrotic areas was performed, together with anterior septal myectomy, with LVOT enlargement .

Postoperatively transesophageal echocardiography demonstrated reduction of dynamic gradients in the LVOT, normal functioning mitral valve prosthesis, normal biventricular contractility and laminar flow in the LVOT[Fig 10, 11]

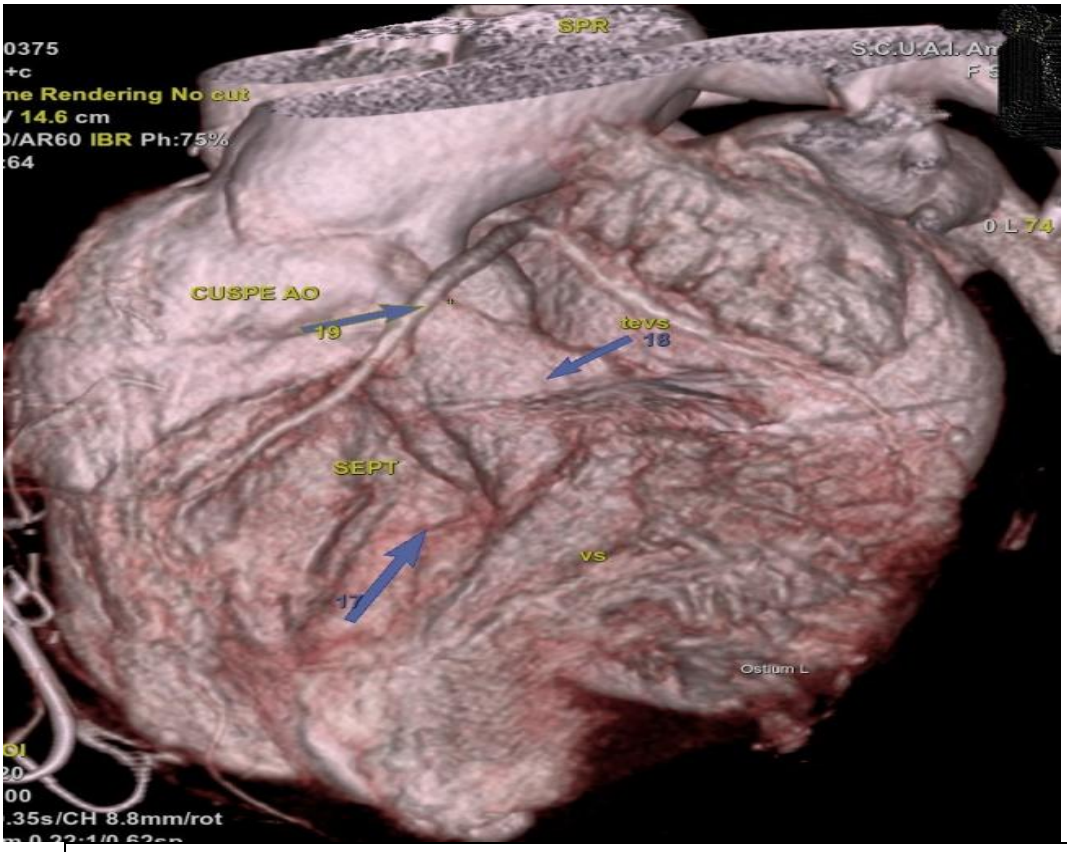

Fig 8: Cardiac CT confirms severe LVOT narrowing due to septal hypertrophy

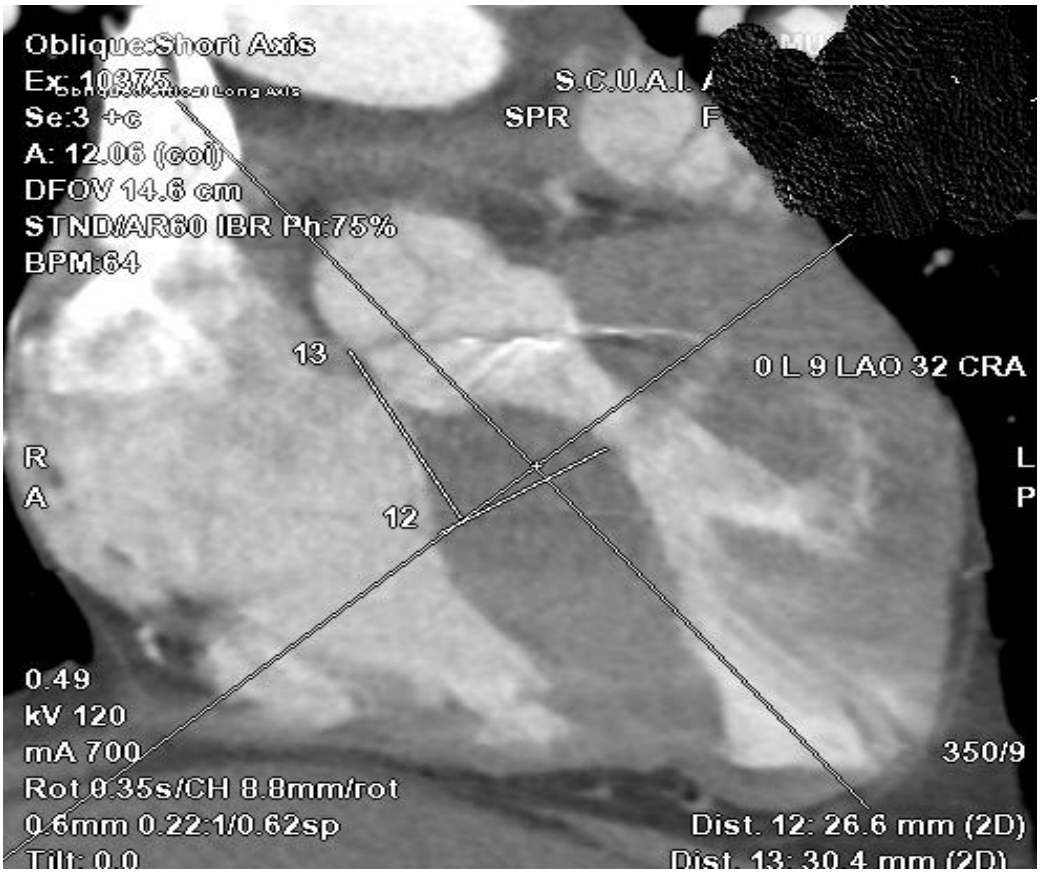

Fig. 9 .Cardiac CT showing severe septal hypertrophy, narrowed LVOT and small LV cavity

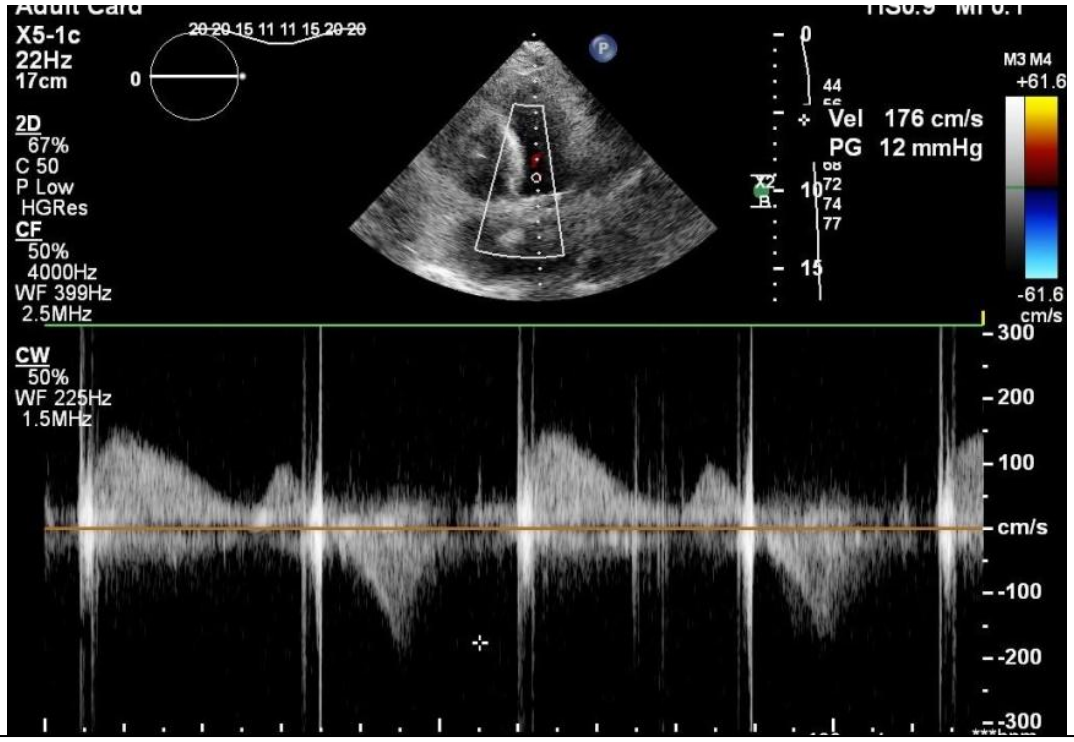

Fig 10: Transthoracic echocardiography 5 C view: postoperative peak flow velocity reduced to 1.7 m/s

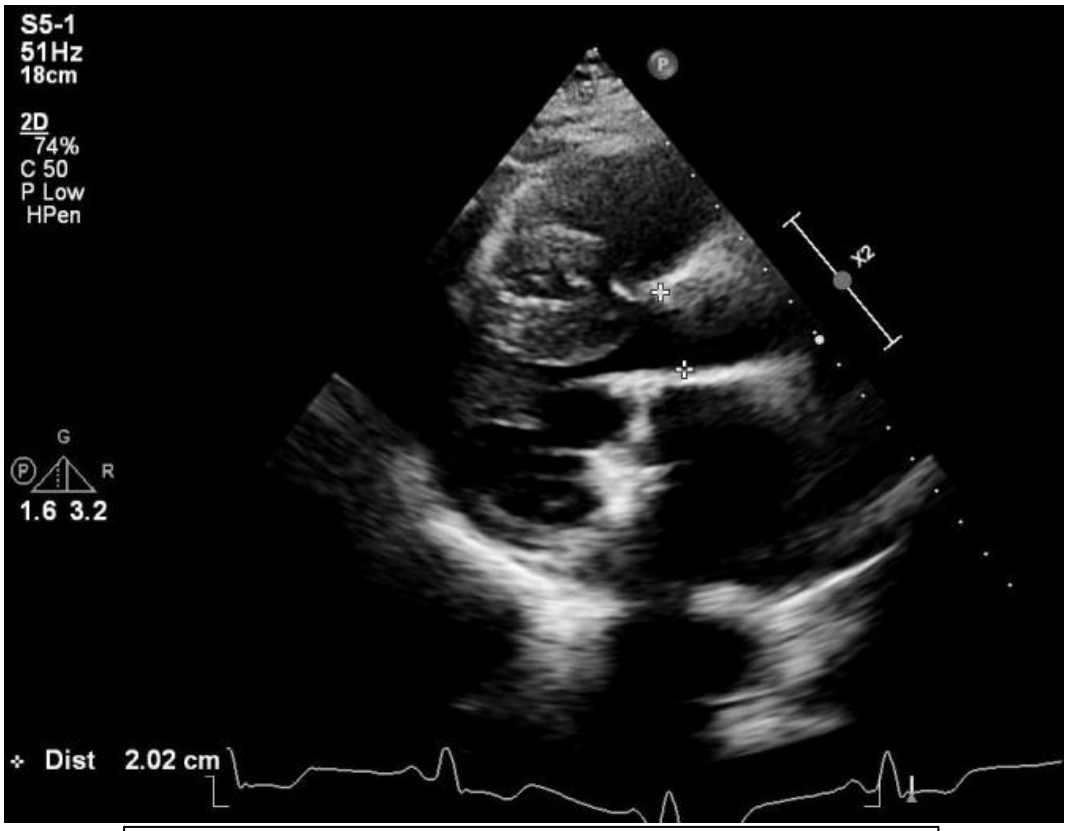

Fig 11. Postoperative enlargement of the LVOT (20 mm diameter)

FOLLOW-UP

In the ICU, our patient presented favorable postoperative evolution , afebrile, hemodynamically and respiratory stable after extubation, with no liver and renal dysfunction and she was transferred on Cardiovascular Surgery Department on postoperative day 4 . Clinical improvement continued; she was respiratory stable, with normal diuresis, normal liver and kidney function and with healing surgical wounds. During cardiac ward stay, the patient underwent respiratory and cardiac rehabilitation. Given the favorable paraclinical and clinical evolution , the patient was discharged on the postoperative day 6 with the following ambulatory medication : Acenocumarol , Bisoprolol 7.5 mg/ day , Amiodarone 100 mg/ day , Amlodipine 5 mg/ day , Torasemide 50 mg/ day , Atorvastatine 20 mg/ day , SGLT2 inhibitor, Euthyrox 50 mcg/ day and bronchodilatator therapy. She was recommended maintaining a Mediterranean diet, low-sodium, low-fat diet and advised to enroll in a cardiac rehabilitation program . In case of dental or surgical bleeding interventions it was recommended endocarditis prophylaxis.

At the 6- month reevaluation, our patient was in good general condition, without clinical signs of left or right heart failure, normal biventricular systolic function, normal valvular function, possible pulmonary hypertension and no pericardial fluid. Echocardiography revealed no subaortic obstruction and a mid -ventricular gradient of 25-39 mmHg at rest.

DISCUSSIONS

Left ventricular outflow tract obstruction is a well-recognized complication following solely replacement of the mitral valve in HOCM. Contributing factors include the profile of the prosthetic valve, a reduced aorto -mitral angle, left ventricular hypertrophy, small ventricular cavity and thickening of the interventricular septum, all of each can result in a narrowed LVOT. In patients with a native mitral valve, such hypertrophy may remain subclinical and asymptomatic; however the presence of a prosthetic valve may exacerbate flow disturbance and lead to symptomatic obstruction. In this case, intraoperative inspection revealed a fibrous banding encircling the LVOT, together with severe interventricular septal hypertrophy, contributing further to flow restriction. We hypothesize that this band likely developed as a result of turbulent flow generated by prosthetic valve protrusion into the outflow tract. The prosthesis valve itself protruded into the LVOT, further compromising flow. This was clearly visualized in transthoracic echocardiographic images (oblique parasternal long-axis views, which demonstrated a thickened interventricular septum and the protruding mitral prosthesis.

Following the surgical procedure, our patient demonstrated excellent clinical recovery, with improved functional capacity and no recurrence of obstruction during follow-up.

CONCLUSIONS

Left ventricular myectomy remains the procedure of choice for patients diagnosed with hypertrophic obstructive cardiomyopathy.

Concomitant mitral valve repair and septal myectomy in patients with HOCM has been associated with favorable results, scientific data suggesting unfavorable outcome in patients who undergo only mitral valve replacement.

Surgical intervention in patients with severe obstructive hypertrophic cardiomyopathy is recommended to be performed in dedicated cardiovascular surgery centers with substantial experience and high operator volumes for this genetic pathology.

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