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CASE REPORT

Alcohol Septal Ablation in Hypertrophic Obstructive Cardiomyopathy: A Patient with a High LVOT Gradient

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Abstract:

Background: Hypertrophic obstructive cardiomyopathy is a type of cardiomyopathy. The presence of a pressure gradient between the mid ventricle and LVOT on the hemodynamic evaluation revealed by echocardiography serves as the basis for the diagnosis.

Objective: To Perform Alcohol Septal Ablation in patient with Hypertrophic Cardiomyopathy

Method: Under both Echocardiography and Fluoroscopy first septal branch of LAD is identified and alcohol is injected to cause necrosis of the selected myocardial tissue to reduce the outflow gradient,

Result: A reduction of the outflow gradient measured after the procedure indicates that it was successful.

Key Words: Hypertrophic Obstructive Cardiomyopathy, Echocardiography, Alcohol Ablation

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A hereditary condition of the cardiac muscle known as hypertrophic cardiomyopathy (HCM) causes left ventricular hypertrophy, myofibrillar disarray, and myocardial stiffness. The most common cause of sudden death in young athletes, this condition affects around 1 in 500 people[1]According to estimates, HCM affects one in 500 people globally in the general population. Still, when all gene-positive people are considered, the prevalence rises to one instance in every 200 people.[2]

Chest pain, dyspnea, palpitations, exhaustion, or syncope are among the clinical signs, particularly after exercising. The majority of hypertrophic obstructive cardiomyopathy (HOCM) patients are detected incidentally; dyspnea is the frequent symptom in 90% of the cases, followed by chest pain in 70-80% and less frequently syncope in 20%[3]

The severity and extent of the hypertrophy will determine the symptoms.HCM patients may experience myocardial ischemia, mitral regurgitation, diastolic dysfunction, or obstruction of the left ventricular outflow tract (LVOT)[4]Exercise testing, cardiac magnetic resonance imaging (MRI), and echocardiography are among the diagnostic tools. LVOT pressure gradient, septal, anterior motion of the mitral leaflet with eccentric MR, and LV wall thickness can all be assessed by echocardiography.[5]

Currently, septal reduction therapy for patients with HCM can be done either by surgical myectomy or by alcohol septal ablation (ASA). Surgical myectomy is

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the most commonly performed surgical procedure in patients with a resting or maximum provoked left ventricular outflow tract (LVOT) gradient of ≥50 mm Hg, who are in NYHA functional Class III-IV, despite maximum tolerated medical therapy.[5]

Alcohol septal ablation, initially documented by Sigwart in 1995, is a catheter-based minimally invasive approach utilized to attain septal reduction.[6]By using angiographic imaging technique and echocardiographic imaging, contrast media is injected into septal perforating arteries, it is possible to determine the arterial supply to the SAM-septal contact area of the basal septum. A small quantity of alcohol injected via an inflated balloon catheter induces an iatrogenic myocardial infarction, induces paradoxical septal motion, promptly eliminates LVOT obstruction, and induces an immediate cessation of SAM. Subsequently, as septal thinning progresses, the obstruction further diminishes. Reductions in mitral regurgitation and end-diastolic pressure of the left ventricle, size and pressure of the left atrium, regression of left ventricular hypertrophy, and alleviation of pulmonary hypertension are among the long-term advantages. Septal ablation improves exercise capacity and symptoms of heart failure, as measured by increased peak oxygen consumption.[7] Similar to the long-term survival of individuals in the general population of the same age and gender, patients who have septal ablation exhibit comparable prognoses over time as those who get surgical myectomy. In contrast to surgical myectomy, alcohol septal ablation is a minimally invasive procedure that necessitates a shorter recovery period and hospital stay.

Few individuals necessitated in-hospital permanent pacemaker placement, and adverse cardiovascular changes happen infrequently throughout the hospitalization process.

Case Presentation

The present case represents successful percutaneous treatment with septal ablation for a patient with hypertrophic obstructive cardiomyopathy associated with systolic anterior motion of the mitral valve and obstruction.

A 24-year-old man with Hypertrophic Cardiomyopathy who has been treated with 100 mg of metoprolol daily was sent to our clinic with complains of chest pain and dyspnea when he exerted himself. His primary diagnosis of Hypertrophic Cardiomyopathy was three years prior. He had a class III New York Heart Association (NYHA) functional capacity. A respiratory system evaluation was performed, and respiratory causes were excluded.

He had no known relatives who have Hypertrophic Cardiomyopathy. During cardiac auscultation, the fourth heart sound and a systolic murmur in the mitral area that radiates to the mid-clavicular line were audible. The patient had repolarization anomalies (negative T waves), left axis deviation, and sinus rhythm on the ECG. Neither cardiomegaly nor pulmonary congestion could be seen on a chest X-ray. The LV anatomy was evaluated by echocardiography, and the measurements of the LV at end-diastole (45 mm) and end-systole were found to be normal (29 mm), SAM of the mitral valve with mild mitral regurgitation, left atrial enlargement (43-mm), and hypertrophy interventricular septum (basal:14 mm, mid:18 mm, distal:13mm). LV ejection fraction was 65%. Continuous-wave Doppler measurements (Figure 1) showed a peak systolic dynamic LVOT obstruction of 96 mmHg at the LV outflow

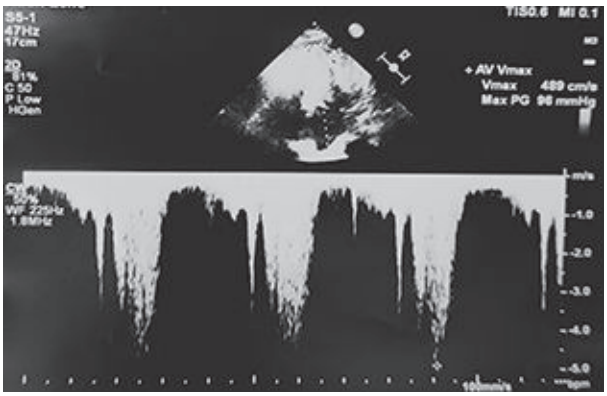

Figure 1: *Preprocedural Echo*

A temporary pacemaker (Figure 2) was inserted as a precautionary measure, and Coronary angiography and an invasive hemodynamic evaluation were carried out. Coronary angiograms revealed that the main epicardial coronary arteries were non-critical. The left ventriculography showed a hyperkinetic contraction pattern at the mid-ventricle with a narrow muscular tunnel between the LV apical and basal cavities

Upon hemodynamic evaluation, a significant intraventricular pressure gradient of 75 mmHg (LV apex

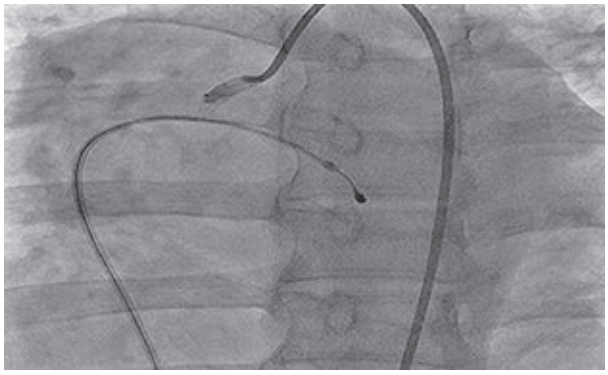

Figure 2: *TPM lead placed*

178/10 mmHg, LV outflow 122/12 mmHg) with mild LV outflow tract pressure gradient of 40 mmHg (aorta 92/64)

We finally diagnosed Hypertrophic Obstructive Cardiomyopathy associated with SAM of the mitral valve.

After being informed of his treatment choices, the patient provided written approval for percutaneous septal myocardial ablation (Figure 3). The first septal branch was dominant (diameter: 2.1 mm) and showed prominent septal myocardial distribution.

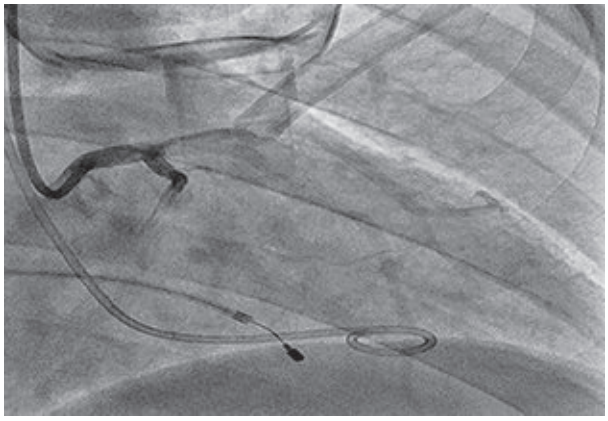

Figure 3: *Angiogram of the left system done and first septal branch identified.*

The floppy wire (Choice scimed guide wire, 0.014", 300 cm, Boston Scientific, Miami, USA) was passed to the first septal branch. A 2.5 × 20 mm over the wire balloon (Maverick OTW PTCA dilatation catheter, Boston Scientific, Maple Grove, USA) was placed in the proximal part of the vessel. Before injecting alcohol, the balloon location was checked using a dye injection. Hemodynamic measurements (Figure 4) were repeated after the balloon had inflated and a transient occlusion of the separate branch. 2 ccs absolute alcohol (Figure 5) in portions of 1 cc/min was injected slowly.

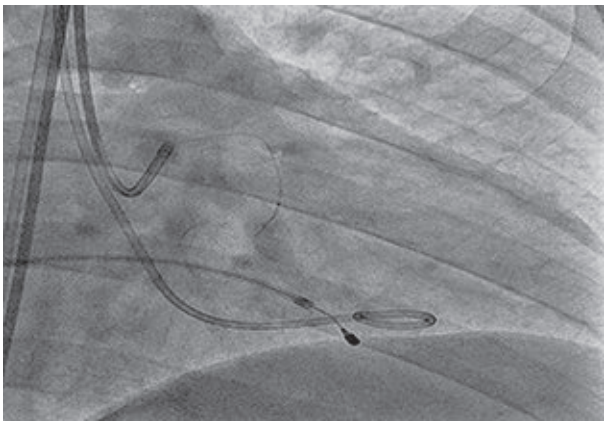

Figure 4: *Invasive hemodynamic measurement*

Repeated hemodynamic measurements revealed that the post-extrasystolic gradient at the midventricular level decreased to 30 mmHg and that there was no resting gradient at the LV outflow level.

The procedure caused the patient to have mild chest pain. There were no complications, such as pericardial effusion. A 1mm ST elevation in V1-2 and a 1mm ST segment depression in II, III, and aVF were visible on the post-procedure ECG as well as RBBB.

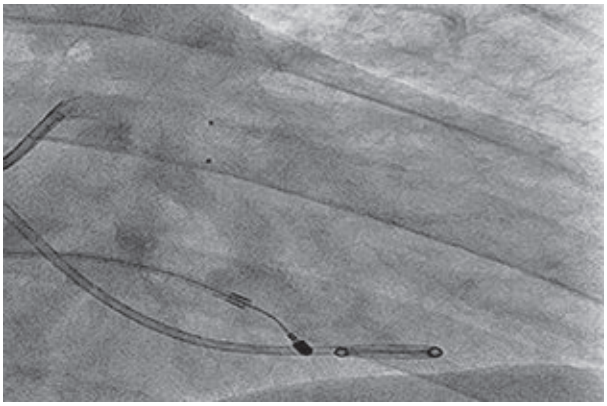

Figure 5: *During alcohol septal ablation*

After 48 hours, temporary pacemaker leads were removed as the patient maintained a sinus rhythm.

Before discharge, a post-procedural echocardiogram was performed, and the results showed that the left atrial dimension had decreased to 42 mm, the

interventricular septum thickness had decreased to 13 mm, there was septal hypokinesia, there was no mitral regurgitation without SAM of the mitral valve. The end-systolic and end-diastolic diameters of the LV were 46 mm and 28 mm, respectively. A peak systolic gradient of 44 mmHg was observed using continuous-wave Doppler measurements.

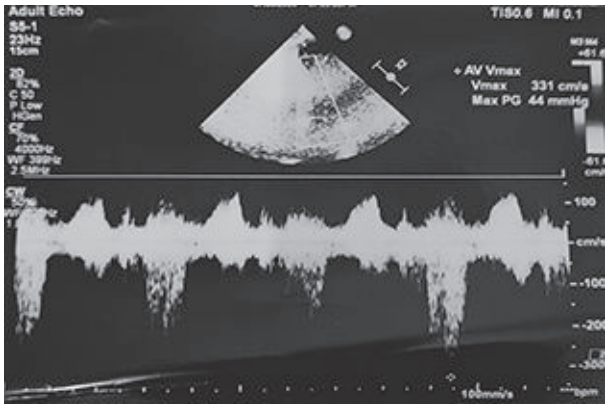

Figure 6: *Post procedural echo before discharge*

After a seven-day, unremarkable hospital stay, the patient was released. Medication with 100 mg metoprolol was continued at discharge. The patient was asymptomatic, and his functional capacity improved during the one-month follow-up to NYHA class I. Physical examination showed a mild systolic murmur in the mitral area. On the ECG, there was no Q wave in V1-2; there was RBBB and sinus rhythm.

Conclusion:

The purpose of septal ablation is to deliver alcohol to the area of the basal septum that contacts the anterior mitral leaflet during systole by using echocardiography guidance to locate the septal artery that supplies it (approximately 1 cc per 1 cm septal thickness). Alcohol septal ablation is a reliable therapy method for specific patients with symptoms associated with HOCM that are drug-refractory[8]. Morbidity is significantly decreased, procedure-related mortality is equal to or lower than with surgical myectomy, and long-term symptom alleviation and survival are comparable. Only a small percentage of individuals need permanent pacing and second ablation treatments[3].

Over the past five to ten years, the course of treatment for hypertrophic cardiomyopathy has seen a significant transformation, moving from surgical myectomy to percutaneous cardiac intervention. The results of alcohol ablation are comparable to those of surgical myectomy and are safe and effective[9].

However, research tends to show that septal myectomy offers patients better long-term symptom alleviation, a decreased risk of complications, and immediate results over ASA with a swift decline in LVOT gradient. Finally, individuals are considered poor candidates for ASA if they have extremely high LVOT gradients[10].

Competing interests

The author(s) declare that they have no competing interests.

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