



Case Report

A PROMISING TREATMENT OF HOCM: PERCUTANEOUS TRANSLUMINAL SEPTAL MYOCARDIAL ABLATION (PTSMA)

Tariq Abbas, Aftab Ahmad Tarique, Saqib Shafi Sheikh, Muhammad Azhar.

INTRODUCTION

Treatment of symptomatic patients with Hypertrophic Obstructive Cardiomyopathy (HOCM) aims to reduce symptoms, improve function capacity and provide better quality of life. Medical therapy with administration of negative inotropes like betablockers, Verapamil or Disopyramide is just the treatment. Unfortunately, 10% of patients with marked outflow tract obstruction have severe symptoms that are unresponsive to medical therapy. In this group surgical treatment with Myectomy/Myotomy has been the main stay for decades with a reported mortality of 1.5-10%

Dual chamber pacing was subsequently introduced as an alternate to surgery but it has not proven to be efficacious in randomized trials. Percutaneous transluminal septal myocardial ablation (PTSMA), through alcohol induced occlusion of septal branch, aims directly to reduce hypertrophied interventricular septum with expansion of Left ventricular out flow tract (LVOT) and reduction in LVOT gradient.

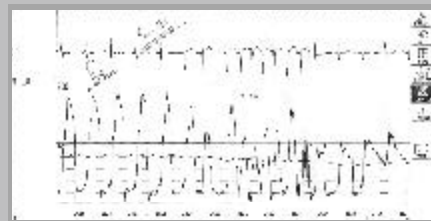
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CASE HISTORY

A 35 years old female came in the out patient department with a history of palpitation and shortness of breath of NYHA II/III for the last one and a half years. On clinical examination her pulse was 80/min with a BP of 100/80 mm of Hg. On precordial examination 1st and 2nd heart sounds were audible with grade III systolic murmur at apex; not radiating to the axilla. Her ECG showed normal sinus rhythm with mild ST-T changes in the precordial leads. Echocardiography revealed hypertrophy of left ventricle with asymmetric involvement of the septum. Systolic anterior displacement of septum (SAM) with mid systolic closure of aortic valve was present. Resting PG across LVOT was 100 mm of Hg. She was diagnosed to be suffering from hypertrophic obstructive cardiomyopathy with LVOT obstruction. She was prescribed Betablocker (Atenolol) 50 mg X OD and Verapamil 240 mg X TID. She could not tolerate the initial dose and her blood pressure dropped so PTSMA was planned.

Procedure was performed through the right and left femoral arteries and the right femoral vein was used for the insertion of a temporary pacemaker lead, which was

positioned in RV. A multipurpose catheter was positioned on the LV through the left femoral artery and 6F JL4 guiding catheter was used for cannulating the left coronary system. Coronary angiography was performed initially which showed large caliber arteries with no obstructive lesion (Figure A) and a dominant right coronary artery which was normal. LV angiogram was done in LAO cranial view, which revealed good LV function with EF=80% and a hypertrophied septum. The gradient across LV to LVOT was measured to be 83 mm of Hg (Picture 1).



Picture 1: The gradient across LV to LVOT



Fig A. Large first septal perforator

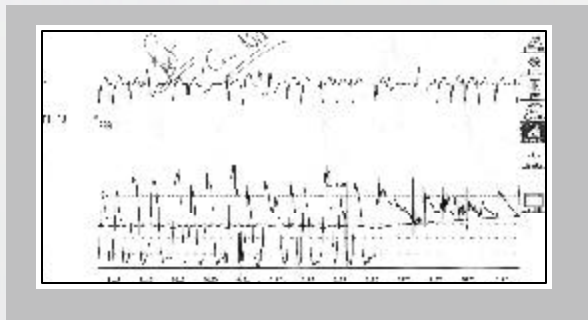
Address for correspondence

Tariq Abbas
Senior Registrar Cardiology
Punjab Institute of Cardiology, Lahore, Pakistan.
Tel No: +92-42-92003051-60 (Ext:336)
E mail: bilal200@yahoo.com



The first septal artery was a medium sized vessel and 0.014" BMW guide wire was positioned in this branch (Figure B). A 2.5x16mm D1 14S OTW balloon was positioned in the proximal third of the first septal and was inflated to a pressure of 12atm after ensuring that it was well within S1 and at a safe distance from LAD (Figure C). Check injection showed a patent LAD with a fully occluded septal perforator. At this stage, the PTCA guide wire was removed and 2cc of alcohol was injected through the inflated balloon catheter lumen (Figure D), then we waited for 5 minutes. Gradient across the LV outflow was measured by Echocardiography. As the residual gradient was more than 50mmHg, another 2cc of alcohol was injected and a repeat measurement of the gradient was done after five minutes. The residual gradient was <30mmHg by direct measurement as well as with Doppler. The balloon was deflated slowly and a final check injection showed occluded septal perforator with no forward flow and a patent LAD (Figure E). There was no withdrawal gradient from LV to LVOT at the end of procedure (Picture 2).

Picture 2. Gradient from LV to LVOT at the end of procedure.



The patient had mild chest pain during the procedure for which 3mg of IV Morphine was given twice. The patient was transferred to CCU with out any complications.

During the procedure the patient developed CHB; TPM took up the heart rate. However at the end of procedure she was having junctional rhythm with RBBB pattern.

TPM lead was kept in for next 3 days. On the 2nd day she had alternating RBBB and LBBB, however in sinus rhythm. Her CPK, CPK-MB rose up 1381 and 184 respectively post procedure.

She was monitored in CCU for 3 days and after restoration of sinus rhythm TPM lead was removed and she was shifted in to the ward where she remained for 3 further days and was discharged on day 7 after the procedure.

Fig B. 0.014" BMW guide wire being placed in first septal perforator



Fig C. 2.5x16mm D1 14S OTW balloon was positioned in proximal third of first septal perforator

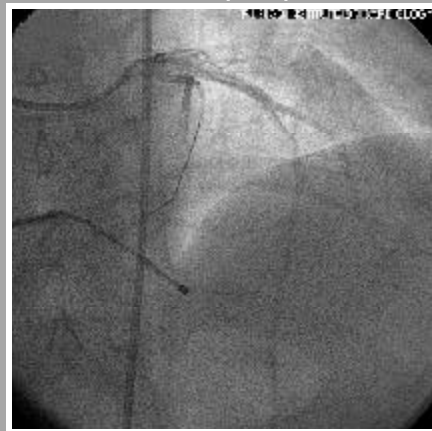


Fig D. Alcohol was injected in first septal perforator territory

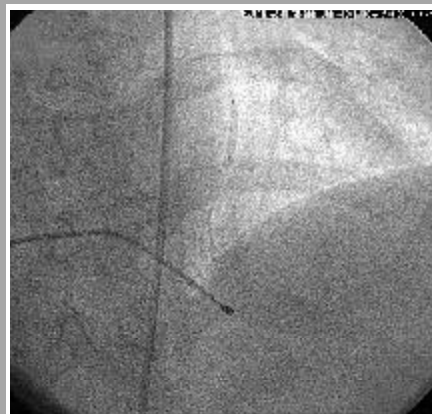




Fig E. The totally occluded first septal perforator with no forward flow



DISCUSSION

Nearly one decade after its introduction, percutaneous septal occlusion is a promising treatment option for symptomatic patients of hypertrophic obstructive cardiomyopathy refractory to medical treatment. Seggewiss H et al⁷ reported the first successful case of PTSMA after the occlusion of the 1st septal branch using 96% alcohol. PTSMA has gained significant popularity in the treatment of HOCM refractory to medical treatment.

PTSMA is indicated in symptomatic patients with NYHA III/ CCS III despite optimal drug therapy or with substantial side effects of medication in patients with high out flow gradients (>30 mm Hg at rest or 100 mm Hg under stress). Patients with less severe symptoms should only be treated if they have high gradient and documented high risk factors for sudden cardiac death, such as recurrent exercise induced syncope. Moreover patients with previous but hemodynamically unsuccessful surgical myectomy or DDD pacemaker implantation can also be treated with alcohol ablation. Symptomatic patients with HOCM and extensive coronary artery disease requiring revascularization are normally candidates for surgical treatment (myectomy+CABG). However in single vessel disease PCI+PTSMA can be performed.

More than 2000 patients are known to have been treated world wide. LVOT gradient reduction can be achieved accurately in about 90% of cases. Younger patients less than 40 years of age had a lower gradient reduction than the elderly, probably due to larger septal thickness. However continuous improvement during fol-

low-up due to post infarction remodeling and shrinkage of the ablated septum has been observed.

Echo-guidance with myocardial contrast echocardiography had a crucial impact on selection of ablated area; it helped to identify an atypically originating septal branch as a target, or to avoid alcohol misplacement.

As far as complications are concerned in-hospital mortality which was initially observed up to 4% has now come down to less than 1% due to increase in experience. The most frequent complication (60%) pre and post procedure is trifascicular block as was observed in our patient. But in the majority of cases these blocks are transitory. After the introduction of MCE the number of permanent pacemaker implantations has been reduced to <5%. A reported dreadful complication is iatrogenic reflux of alcohol into the LAD with transitory vessel occlusion and Antero-Lateral ischemia.

Clinical and hemodynamic follow-up studies up to 6 years have shown no increased risk of sudden death or arrhythmic complication. Furthermore no septal perforation has been reported during follow-up. The most important finding is an impressive symptomatic improvement during short and long term follow-up. Improvement in the mean functional class from NYHA 2.8±0.6 to 1.6±0.7 was seen after a mean follow-up period of >3 years (p<0.0001). Objective finding showed an increase of exercise capacity from 88±57 to 110±40 watts after 3 months (p<0.0001). Moreover, a continuous decrease in the ventricular out flow gradient has also been observed, probably remodeling after PTSMA.

Up to now no randomized trials comparing surgical treatment and PTSMA have been published. Non-randomized trials have shown significant reduction in LVOT gradient and symptomatic improvement in both the treatment options. The advantages of PTSMA comprise the avoidance of cardiopulmonary bypass, especially in elderly with concomitant diseases, shorter hospital stay and recovery time as well as the fact that the Percutaneous approach is less expensive.

Potential drawbacks of PTSMA are the risk of damage to LAD requiring urgent CABG or the technical impossibility to identify and to reach the target septal branch.

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REFERENCES

1. Seggewiss H, Rigopoulos A, Faber L, Ziemssen P. Percutaneous septal ablation in hypertrophic obstructive cardiomyopathy: indication, technique and results. In: The Paris Course on Revascularization, Marco J, Serruys P, Biaino G, et al. (eds.). Paris 2003:283-293.
2. Wigle ED, Rakowski H, Kimball BP, William WG. Hypertrophic cardiomyopathy: clinical spectrum and management. *Circulation* 1995; 92:1680-92.
3. Spirito P, Seidman CE, Mc Kenna WJ, Maron BJ. The management of hypertrophic cardiomyopathy. *N Engl J Med* 1997; 775-85.
4. Maron BJ, Nishimura RA, Mc Kenna WJ, et al. Assessment of permanent dual-chamber pacing as a treatment for drug refractory symptomatic patients with obstructive hypertrophic cardiomyopathy. A randomized, double blind, crossover study (M-PATHY). *Circulation* 1999; 99:2927-33.
5. Faber L, Seggewiss H, Gleichmann U. Percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy: Acute and 3-months follow-up results with respect to myocardial contrast echocardiography. *Circulation* 1998; 98:2415-21.
6. Faber L, Seggewiss H, Fassbender D, et al. Guiding of Percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy by myocardial contrast echocardiography. *J Interv Cardiol* 1998; 11:443-48.
7. Seggewiss H, Faber L, Meyers W, et al. Simultaneous Percutaneous treatment in hypertrophic obstructive cardiomyopathy and coronary artery disease: a case report. *Cathet Cardiovasc Diagn* 1998; 44:65-69.
8. Knight C, Kurbaan AS, Seggewiss H, et al. Non-surgical septal reduction for hypertrophic obstructive cardiomyopathy: outcome in the first series of patients. *Circulation* 1997; 95:2075-81.
9. Faber L, Meissner A, Ziemssen P, Seggewiss H. Percutaneous transluminal septal myocardial ablation for hypertrophic obstructive cardiomyopathy: long term follow up of the first series of 25 patients. *Heart* 2000; 83:326-31.
10. Seggewiss H, Faber L, Ziemssen P, Meyners W. Non-surgical septal ablation (PTSMA) in patients with NYHA class IV and hypertrophic obstructive cardiomyopathy (HOCM). *Circulation* 1999; 100(Suppl I):515 (abstract)
11. Singh M, Edwards WD, Holmes DR Jr, Tajik AJ, Nishimura RA. Anatomy of the first septal perforating artery: a study with implications for ablation therapy for hypertrophic cardiomyopathy. *Mayo Clin Proc* 2001; 76:799-802.