**Brief Report**

Occlusion of the First Septal Branch With Microcoils in a Patient With Hypertrophic Obstructive Cardiomyopathy

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Nonsurgical septal reduction by induced septal infarction is one of the management options in the treatment of patients with hypertrophic obstructive cardiomyopathy. Good immediate and long-term clinical and hemodynamic results have been reported with this technique for occlusion of the first septal branch of the anterior descending coronary artery followed by ethanol infusion. This is the first report of a case in which nonsurgical septal reduction with microcoils has been attempted.

**Key words:** Ablation. Cardiomyopathy. Infarction. Coils.

Full English text available at: www.revespcardiol.org

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**INTRODUCTION**

In patients with hypertrophic obstructive cardiomyopathy (HOCM) for whom medical treatment has proved ineffective, the non-surgical reduction of the interventricular septum is a therapeutic option that can provide sustained improvement in exercise capacity as well as persistent reduction in the resting and exercise gradients of the left ventricular outflow tract (LVOT). Since Sigwart et al. described the technique of septal reduction via ethanol ablation of the first septal branch of the anterior descending coronary artery, numerous reports on the success and viability of this therapy have been published.

Microcoils are devices designed to occlude blood vessels or undesired conduits. They are deployed by catheters (several systems are available), and are effective in occluding both veins and arteries. These devices have been used successfully to occlude coronary arteries in patients with fistulas at this site. The literature contains no previous reports of attempts to reduce the LVOT gradient in patients with HOCM by ablation of the first septal branch with microcoils.

**CLINICAL CASE**

A 64-year-old woman presented with a five-year history of exercise-associated dyspnea but with no cardiovascular risk factors or other medical antecedents of note. The patient was studied in the outpatient clinic, where an electrocardiogram showed sinus rhythm with a frontal plane QRS axis of 10º, signs of left ventricular hypertrophy by voltage criteria (Sokolov 63 mm), and secondary changes in repolarization. No pathological Q waves were found, nor were any changes in conduction rhythm observed.

An echocardiogram showed a non-dilated left ventricle with preserved systolic function (ejection fraction 69%), but severe hypertrophy (septum 20 mm, posterior wall 17 mm). Systolic anterior motion of the mitral valve septal cusp was appreciable. Although it did not make contact with the septum, it
but declined. Non-surgical reduction of the septum by inducing a septal infarction was therefore proposed as an alternative, making it very clear that this was an experimental technique. Because our department has no experience in septal ablation with alcohol, but has ample experience in the use of coils in different situations, the decision was made to close the first septal branch with this technique. During catheterization, a first main septal branch of 1.8 mm was identified with a wide perfusion field in the interventricular septum (Figure 1). A 3-m Anibal guidewire was passed into the septal branch, and via this a 2.3 Fr Microrapid Transit Cordis catheter was introduced, which was placed distally in the septal branch. Via this catheter a thermal system was used to deploy 3 thermo-expandable microcoils (Boston GDF 2 mm×1 cm, 2.5 mm×3 cm, and 2.5 mm×3 cm), achieving complete closure of the vessel (Figure 2). It is noteworthy that the patient showed only a small increase in creatin phosphokinase MB (CPK-MB) (687 IU total CPK [maximum reference value 170 IU] with an MB fraction of 19%). The increase in troponin I was more significant, reaching 14.8 ng/mL (maximum reference value 2.3 ng/mL). Clinically, the patient complained only of slight chest discomfort. No clear changes with respect to the baseline ECG could be observed. However, the LVOT gradient decreased to 113 mm Hg (i.e., by about 50%) after treatment, although no appreciable morphological changes in the septum or its thickness could be seen. The patient was released on beta-blocker treatment but continued to show class III dyspnea. Two months later her LVOT gradient was 186 mm Hg. Eventually, the patient agreed to surgery and a myectomy was performed. One year later, the patient showed class II dyspnea and a subaortic gradient of 32 mm Hg.

DISCUSSION

The non-surgical reduction of the interventricular septum is a potential alternative to surgery for the treatment of HOCM. It provides substantial improvement in symptoms, a notable improvement in exercise capacity, and a reduction in the LVOT gradient. These improvements have been maintained in patients followed over the long-term.17 Although controlled, randomized studies comparing this still-experimental technique with surgery are required, it would seem to offer an alternative for certain patients with HOCM, especially for those who are unwilling to undergo surgery or for whom comorbidity renders surgery inadvisable.

Despite treatment, the patient showed class III-IV functional status and was admitted to hospital, where dual-chamber stimulation was considered. In tests performed in the electrophysiology laboratory, different atrioventricular delays failed to produce any significant variation in the gradient; this therapy was therefore ruled out. The patient was offered surgery caused a subvalvular gradient of 232 mm Hg, and moderate mitral insufficiency was apparent. The Doppler velocity curve was typical of dynamic obstruction. Considering the high speed of the blood flow, special attention was paid to make sure the LVOT velocity spectrum was not distorted by the signal originating from the patient’s mitral insufficiency. The pressure in the pulmonary artery was 50 mm Hg. Cardiac nuclear magnetic resonance imaging showed morphological changes consistent with the echocardiographic results. The patient was treated with beta-blockers and with beta-blockers plus calcium antagonists, but no clinical results or significant variation in the subaortic gradient were achieved.
in the gradient was initially seen, within two months it had returned to the baseline value. These findings suggest that simple mechanical occlusion of the first septal branch is not effective in the treatment of HOCM, and that ethanol is needed to produce the degree of necrosis required for effective hemodynamic results to be achieved.

REFERENCES


