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.

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 exercising 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 10°, 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.

**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. 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 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.

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clinically or enzymatically significant infarction was induced, even though the vessel we treated was an angiographically important (1.8 mm) septal artery, total occlusion was achieved, and there were no other angiographically visible septal or collateral arteries nearby. This may be because the special organization of the myocardium in these patients requires the local abrasive effect of ethanol if permanent necrosis is to be achieved, and the ischemia produced through occlusion of the vessel may not be sufficient to trigger an infarction.

When Sigwart\(^2\) described the first 3 cases of nonsurgical myocardial reduction in HOCM, he noted that hemodynamic effects on the LVOT gradient immediately reverted when the balloon occluding the septal branch was deflated, even after inflations lasting 30 min. In their patient, de la Torre et al\(^8\) observed no reduction in the gradient until alcohol infusion, even though the artery had been occluded for 20 min. Faber et al\(^5\) who measured the variation in the LVOT gradient in 91 patients during balloon occlusion to confirm the target branch before alcohol treatment, reported that simple occlusion led to a decrease in LVOT gradient from 73.8±35.4 to 36.4±29.3 mm Hg—less than that produced by the alcohol infusion (16.6±18.1 mm Hg) but still statistically significant. However, it is not known how the gradient might have changed afterwards if ethanol infusion had not been performed. There are no previous studies of the results that might be achieved through permanent mechanical occlusion of the first septal branch. We used a permanent coronary occlusion method with microcoils, but the procedure did not provide a satisfactory outcome. Though a significant reduction 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