INTRODUCTION

Until the 1990s, myectomy was the treatment of choice for reducing obstruction in patients with hypertrophic obstructive cardiomyopathy and symptoms refractory to drug therapy.1 The therapeutic options increased with the introduction of DDD ventricular pacing2 and more recently, percutaneous septal ablation.3-7 Because of its effectiveness and safety, the use of this last option has become increasingly more widespread.8,9 Nevertheless, complications or undesirable effects can occur on occasion.10-14 We describe for the first time a complication associated with this technique consisting of rupture of the occlusion balloon and extravasation of alcohol toward the left anterior descending artery.

CASE STUDY

A 57-year-old man diagnosed with hypertrophic obstructive cardiomyopathy and currently on beta-blockers, verapamil, and furosemide with aggravation of his symptoms over the past year, presenting with dyspnea on slight exertion and acute pulmonary edema that required hospitalization. The echocardiogram showed severe left ventricle hypertrophy affecting the septum (27 mm), systolic anterior motion of the mitral leaflet, and a maximum gradient of 116 mm Hg in the left ventricular outflow tract. A decision was made to perform percutaneous septal ablation.

Balloon Rupture and Alcohol Leakage Into the Left Anterior Descending Coronary Artery During Percutaneous Septal Ablation for Hypertrophic Obstructive Cardiomyopathy

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We present a case of rupture of the balloon during percutaneous transluminal septal myocardial ablation with alcohol in a patient with hypertrophic obstructive cardiomyopathy. Rupture of the balloon caused reflux of alcohol into the left anterior descending artery. Angina, mild global hypokinesia of the left ventricle and advanced atrioventricular block were observed. Cardiac function recovered in a few minutes and peak creatinine kinase was 526 U. Despite the restoration of sinus rhythm, there were episodes of complete atrioventricular block that made permanent pacemaker implantation necessary.

Key words: Ablation. Hypertrophic obstructive cardiomyopathy. Complications.
The procedure was performed using the technique described by Seggewiss et al. Following prophylactic placement of an intracardiac pacemaker, the ostium of the left coronary was canalized with a 7 French Judkins left 4.0 guiding catheter. Selective catheterization of the first septal artery was done using an ACS BMW intracoronary guidewire (0.014 × 190). Septal artery adequacy was studied by echocardiography with 1 mL of contrast material (Levovist® 350 mg/mL) (Figure 1). The diameter of the target vessel was 2.3 mm; a 2.5 × 9-mm Maverick® balloon was advanced and inflated to 7 atmospheres. After confirming the absence of reflux toward the distal territory of the left anterior descending artery, 2 mL of 96% alcohol were injected. The balloon remained inflated for 10 min, after which time a persistent gradient of 100 mm Hg was observed; therefore, a second 2-mL dose of alcohol was injected. The balloon ruptured immediately after the injection was started and part of the alcohol passed toward the left coronary vasculature, producing severe anginal pain. The echocardiogram showed “illumination” of the entire left ventricular endocardium, including the lateral wall (Figure 1C). Left ventricular contractility deteriorated and the ejection fraction dropped from 65% to 50%. This was accompanied by atrioventricular block (AV) with accelerated idioventricular rhythm that required ventricular pacing, and a maximum creatine kinase peak of 526 U/L. On the third day, some advanced AV block episodes persisted, left ventricle contractility was normal, and the gradient was >100 mm Hg. On the fifth day of hospitalization, a permanent DDDR pacemaker was implanted and the gradient was reduced to 35 mm Hg (Figure 2).
been reported. Several cases of alcohol reflux towards the left anterior artery due to poor balloon positioning (too proximal to the vessel outlet) or after a second injection of alcohol have been described. ST segment elevation, ventricular arrhythmia, and high enzyme elevation (1496 U) are observed in these cases. In our patient, the control echocardiogram showed that alcohol had passed into the myocardium (increased endocardial echogenicity), and the procedure was stopped prematurely, possibly limiting the resulting damage. Alcohol reinjection must be done with great care, since necrosis caused by the first reinjection can lead to increased coronary resistance and facilitate alcohol reflux.

REFERENCES