Myectomy-Like Extended Alcohol Septal Ablation for Hypertrophic Obstructive Cardiomyopathy

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Int J Angiol 2016;25:e153–e155.

Abstract

Keywords

- hypertrophic cardiomyopathy
- coronary artery
- symptomatic

Currently, two therapeutic options are available for patients with drug-resistant obstructive hypertrophic cardiomyopathy. While there is a tendency in extended myectomy to perform a larger septal reduction to eliminate left ventricular outflow tract gradient more effectively, there has been a trend in recent years toward using lower dosages of alcohol during alcohol septal ablation (ASA) to reduce the risk of arrhythmic complications. We report a case of a symptomatic patient with severe septal hypertrophy who underwent an extended ASA of three septal branches. This led to a result similar to an extended myectomy with a favorable short-term follow-up.

Case Report

A 34-year-old man with a history of type 1 diabetes mellitus and obstructive hypertrophic cardiomyopathy (HCM) was referred to our department for exertional dyspnoea and chest pain, despite medication with 200 mg metoprolol daily. His functional capacity was class III according to the New York Heart Association (NYHA). Echocardiography revealed marked hypertrophy of, mainly, the basal anteroseptal segment, a significant systolic anterior motion (SAM) of the mitral valve, and a combination of midventricular and subaortic obstruction with a resting maximal left ventricular outflow tract (LVOT) gradient of 150 mm Hg (► Fig. 1A). Considering the symptomatic LVOT obstruction and symptoms of heart failure that were refractory to the medical therapy, the patient was indicated for septal reduction therapy. After a thorough discussion about his therapeutic options, the patient chose alcohol septal ablation (ASA). Hemodynamic measurement confirmed a severe LVOT gradient and coronary angiography revealed no abnormalities (► Fig. 2A, B). Because of the significant septal hypertrophy (28 mm) and the combination of midventricular and subaortic obstruction, a large septal reduction was indicated.

Three septal perforator coronary artery branches were chosen as the target septal arteries (TSA). The perfusion area of TSA was subsequently confirmed by myocardial contrast echocardiography (MCE). First, a temporary right ventricular pacemaker was placed and prophylactic analgesia was administered. Subsequently, ASA of TSA was performed using the total dose of 2.7 mL 96% ethanol. This procedure caused an immediate decrease of the peak-to-peak gradient to 5 to 10 mm Hg (► Fig. 2C, D), with an optimal MCE finding. The patient was observed in the coronary care unit after the procedure, and as no conduction abnormality developed, the temporary pacemaker was soon removed. The postprocedural echocardiography showed a reduction of maximal LVOT gradient to 19 mm Hg at rest. The patient was discharged on the sixth day after ASA. At 2-month follow-up, the patient was free of symptoms with NYHA class I functional capacity. The control echocardiography revealed a reduction of the inter-ventricular septum thickness with an akinesis of the basal segment and the disappearance of SAM with a favorable reduction of maximal LVOT gradient to 11 mm Hg (► Fig. 1B). Cardiovascular magnetic resonance imaging confirmed significant reduction of basal septal thickness at 3-month follow-up.

published online
September 30, 2015

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Tel: +1(212) 584-4662.

DOI <http://dx.doi.org/10.1055/s-0035-1564660>.
ISSN 1061-1711.

Fig. 1 (A) Preprocedural echocardiography showing a septal hypertrophy and systolic anterior motion (SAM). (B) Control echocardiography showing the reduction of septal hypertrophy and the disappearance of the SAM.

Fig. 2 (A) Left coronary angiography before alcohol septal ablation showing the target septal arteries. (B) Left ventricular and aortic pressures before and (C) immediately after alcohol septal ablation. (D) Postprocedural left ventricular and aortic pressures.

Discussion

In the treatment of patients with LVOT gradient ≥ 50 mm Hg and advanced symptoms of heart failure that are refractory to medical therapy, two different approaches are

available.¹ ASA reduces LVOT obstruction by creating a myocardial shrinking in the area of the basal septum after a selective injection of alcohol into a septal artery. Since the myocardial scar resulting from ASA might cause ventricular arrhythmias, there has been a trend toward using lower

dosages of ethanol (≤ 2 mL) during the procedure to decrease the risk of arrhythmic complications.² The low-dose ASA seems to be a suitable treatment for patients with a subaortic LVOT obstruction, an absence of midventricular obstruction, and a milder septal hypertrophy with marked hemodynamic and clinical improvement in the long-term follow-up. On the contrary, the current approach of surgical septal reduction therapy is based on extended myectomy, which helps to abolish obstruction more effectively by extending to the midventricular segment of interventricular septum with partial excision and mobilization of the papillary muscles.³ Although surgical myectomy is the preferred therapy for young, drug-resistant HCM patients,¹ ASA seems to be an equal alternative that may be considered even for patients younger than 50 years.⁴ In our report, we present a case of a patient with severe LVOT obstruction, in whom we attempted to perform an extended myectomy-like ASA. We used a total dose of 2.7 mL ethanol, which is higher than the low dose of 1.5 to 2 mL routinely used nowadays.² We achieved a reduction in the maximal resting LVOT gradient from 150 to 11 mm Hg. Both in-hospital and short-term complications have not been

observed and the patient remained asymptomatic during 3 months after ASA. However, we have to bear in mind that long-term follow-up is needed to determine whether this approach in the management of obstructive HCM is both effective and safe.

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