

Update on alcohol septal ablation for hypertrophic obstructive cardiomyopathy

Josef Veselka, Eva Polaková, Jiří Bonaventura

Department of Cardiology, University Hospital Motol and 2nd Medical School, Charles University, Prague, Czech Republic

Article Dąbrowski et al., see p. 181

The first alcohol septal ablation (ASA) for hypertrophic obstructive cardiomyopathy (HOCM) was performed by Ulrich Sigwart, and the description of the procedure was published in *The Lancet* in 1995 [1]. Currently, based on the increasing body of evidence, it is known that severe and symptomatic left ventricular obstruction usually needs effective mechanical relief in the form of septal reduction therapy (ASA or myectomy) [2].

The technique of ASA involves an injection of a small amount (1–3 mL) of desiccated alcohol into an appropriate septal branch [3–6]. The rapid postprocedural pressure gradient decrease is caused mainly by stunning and myocardial necrosis. However, the main mechanism of the continuous pressure gradient relief is the widening of the left ventricular outflow tract developing secondarily to infarction and fibrosis of the basal septum. Subsequently, the basal septal shrinking is followed by a gradual reduction in the left ventricular mass, improvement of the diastolic function, and reduction in the degree of mitral regurgitation [3–7]. Also, successful procedures lead to an improvement in symptoms of angina pectoris and dyspnoea [3–7].

The first European multinational study focused on early outcomes of ASA undoubtedly demonstrated its clinical efficacy, but the safety of this interventional procedure was limited as complete heart blocks occurred in one-third of the procedures and one-tenth of the patients needed permanent pacing after ASA [7, 8]. It was also found that in the younger HOCM patients, characterised by a thicker basal septum, post-ASA haemodynamic improvement was slower, but the procedure was effective irrespective of the age of the treated patients [9].

Although encouraging results of single-centre or national ASA registries were published in the first two decades after the first procedure performed by Sigwart, the long-term safety and efficacy of ASA have been a matter of ongoing debate.

Therefore, we reported the long-term outcomes from the largest multinational ASA registry (the Euro-ASA registry, $n = 1275$) [10]. The principal findings of this study were as follows: (i) the left ventricular outflow gradient was lowered by 76%, (ii) 86% of patients experienced improvement of one class or more in New York Heart Association (NYHA) functional classification; (iii) a more pronounced reduction of the left ventricular outflow gradient was associated with a lower resultant NYHA class; (iv) the 30-day postprocedural mortality was 1%, and 12% of treated patients required an early postprocedural pacemaker implantation; (v) the annual post-ASA mortality rate was 2.4%, and the risk of a sudden mortality event was 1% per year. It has also been reported that residual post-ASA outflow gradient ≥ 30 mmHg is associated with significantly higher risk for subsequent cardiovascular mortality events [11].

In this issue, Dąbrowski et al. [12] presented a single-centre observational analysis of ASA patients compared with patients receiving optimal pharmacotherapy. The size of the ASA cohort was rather limited ($n = 30$) and long-term outcomes were not clearly mentioned. Nevertheless, the authors presented new findings regarding N-terminal pro-B-type natriuretic peptide levels and concluded that this parameter may be useful in assessing the efficacy of ASA which is interesting.

Current evidence in the ASA field is encouraging and suggests that this interventional procedure can positively affect lives of HOCM patients, especially if they are treated in centres dedicated to hypertrophic cardiomyopathy [13].

Conflict of interest: none declared

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Address for correspondence:

Josef Veselka, MD, PhD, Department of Cardiology, University Hospital Motol, V uvalu 84, Prague 5, 15000, Czech Republic, e-mail: veselka.josef@seznam.cz

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