Long road to success. Endocardial radiofrequency ablation of septal hypertrophy using three-dimensional electroanatomic mapping guided by echocardiography in a patient with hypertrophic obstructive cardiomyopathy and severe aortic stenosis

Dorota Żmudzińska[1]*, Krzysztof Ściborski[1]*, Paweł Szymkiewicz[1], Marek Mak[2], Artur Telichowski[1], Dorota Kustrzycka-Kratochwil[1], Adrian Doroszko[1, 3], Waldemar Banasiak[1, 3], Krystian Josiak[1]

[1]Department of Cardiology, Centre for Heart Diseases, 4th Military Hospital, Wrocław, Poland
[2]Department of Cardiac Surgery, Centre for Heart Diseases, 4th Military Hospital, Wrocław, Poland
[3]Clinical Department of Cardiology, Faculty of Medicine, Wroclaw University of Science and Technology, Wrocław, Poland
*Both authors equally contributed to the study.

Correspondence to:
Dorota Żmudzińska, MD, Department of Cardiology, Centre for Heart Diseases, 4th Military Hospital, Weigla 5, 50–981 Wrocław, Poland, phone: +48 794 118 077, e-mail: dr.zmudzinska@gmail.com

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As far as the literature is concerned, approximately 75% of patients with hypertrophic cardiomyopathy have resting or latent obstruction (hypertrophic obstructive cardiomyopathy [HOCM]) of the left ventricle outflow tract (LVOT). Standard procedures for septal reduction include alcohol septal ablation and septal myectomy [1, 2]. However, the scenario in which HOCM coexists with severe aortic stenosis (AS) remains challenging, and septal myectomy with valve replacement seems to be the most favorable option. However, if the patient is ineligible for cardiac surgery, which strategy might be beneficial remains an open question.

A 77-year-old woman who had previously undergone coronary artery bypass grafting (CABG) 7 years earlier, with well-controlled arterial hypertension, hypercholesterolemia, and body mass index of 24 kg/m^2, was admitted to the cardiology department due to angina pectoris.

Transthoracic echocardiography (TTE) was conducted before the CABG procedure. However, the results did not indicate the presence of HOCM. The examination showed the absence of valvular defects and normal contractility. Based on these, the patient was promptly transferred from the regional cardiology department to the cardiac surgery department. Her imaging findings were not confirmed, and the cardiosurgical procedure included only CABG.

Her subsequent TTE exam revealed significant hypertrophy and LVOT obstruction (resting peak gradient over 100 mm Hg) (Figure 1A), AS, and mitral regurgitation (MR) caused by systolic-anterior motion (SAM). The aortic valve area (0.81 cm^2) on transesophageal echocardiography and calcium score (>2000 AgU) on computed tomography confirmed severe AS diagnosis. Coronary angiography showed patent bypass grafts, including the left internal mammary artery-left anterior descending coronary artery, which was the cause of her disqualification from surgical treatment.

Despite favorable conditions (occlusion of the septal branch perfusing the basal segment of the interventricular septum), there were no significant effects of septal ablation (Figure 1B). Before the implantation of a cardioverter-defibrillator, magnetic resonance imaging revealed the presence of fibrotic foci, which may have been associated with hypertrophic cardiomyopathy. The gradient reduction after implantation of the cardioverter-defibrillator was not achieved using optimal atrio-ventricular delay.

The subsequent intervention was transcatheter aortic valve implantation. Hemodynamic measurements performed immediately after the procedure demonstrated persistent

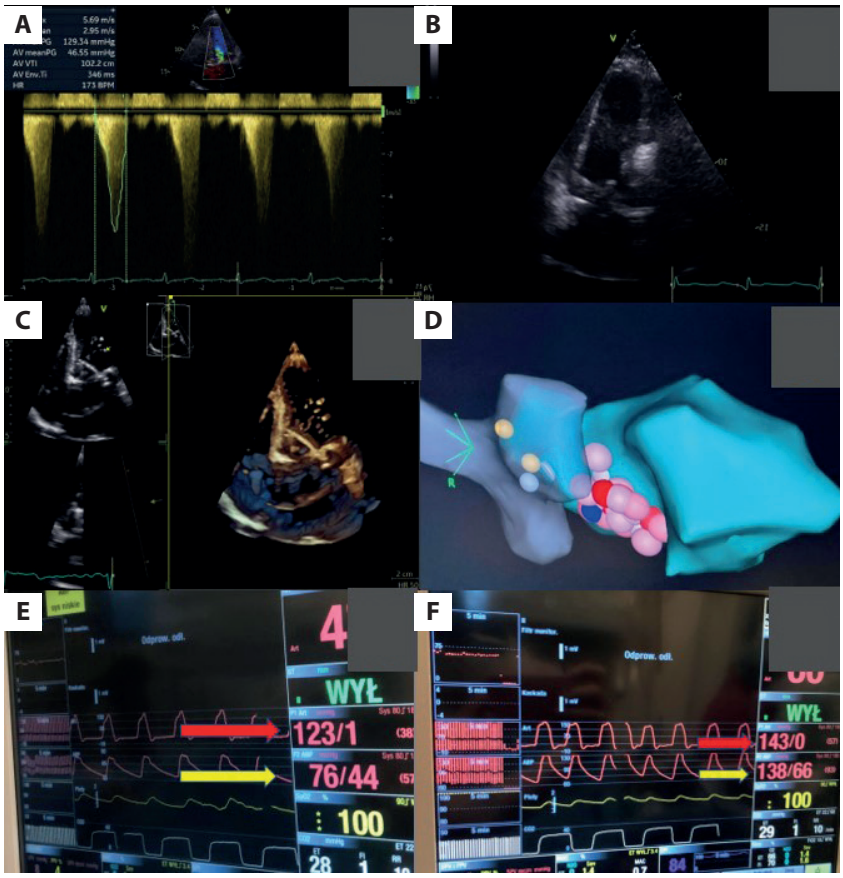

Figure 1. A. Severe obstruction of the left ventricular outflow tract (2D echocardiography, CW Doppler resting gradient). **B.** Alcohol septal ablation, optimal perfusion of the basal segment of the interventricular septum. **C.** 3D-echocardiography, position of the ablation catheter. **D.** 3D-mapping system, area of ablation. **E.** Hemodynamic measurements at the beginning of the procedure; the red arrow indicates the left ventricular pressure; the yellow arrow indicates the pressure in the ascending aorta. **F.** Hemodynamic measurements at the end of the procedure; the red arrow indicates the left-ventricular pressure; the yellow arrow indicates the pressure in the ascending aorta

high LVOT obstruction, and at the 3-month follow-up, the prosthetic valve exhibited good functionality with persistent LVOT (peak gradient of 100 mm Hg), and the patient remained symptomatic.

As the previous procedures failed, we decided to perform endocardial radiofrequency ablation of septal hypertrophy. Under general anesthesia using right-femoral-vein access *via* transseptal-puncture, left-atrium and mitral-annulus, an irrigated, contact-force ablation catheter (Thermocool-Smart-Touch-SF, Biosense-Webster) was introduced into the LV. Current hemodynamic measurements were conducted with two pig-tail catheters in the LV-apex and the ascending aorta (Figure 1E–F). Combining TTE, transesophageal echocardiography, and 3D-mapping system (CARTO-3, Biosense-Webster) determined the best placement of ablation over the SAM-septal contact area and electroanatomic-mapping allowed marking conduction system preventing thus its damage.

The target area was designated on the maximal contact zone between the basal septum and the mitral valve (Figure-1C). A total of 30 applications of radiofrequency energy (power 40–45 W, 60 sec duration) were delivered to the target area. The estimated lesion area was 2.5 cm^2 (Figure 1D), which resulted in a significant reduction in the LVOT gradient: to 5 mm Hg measured invasively and to 20 mm Hg on TTE. There were no complications, including atrio-ventricular block or arrhythmias.

At the 12-month follow-up, the TTE revealed persistent reduction in the LVOT gradient, regression of the SAM, with reduction of the mitral regurgitation, and the patient remained asymptomatic.

Although this method is not included in the guidelines, several cases have demonstrated that endocardial radiofrequency ablation of septal hypertrophy may lead to a significant and constant LVOT obstruction reduction [3–5]. This intervention may provide an option to treat HOCM in patients in whom septal reduction therapy has failed or in those who are not good candidates for such treatment. Combining echocardiography with 3D electro-anatomical mapping is crucial for choosing the optimal target.

Article information

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