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Percutaneous closure of a ventricular septal defect after surgical treatment of hypertrophic cardiomyopathy

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Surgery or ethanol ablation is indicated in patients with hypertrophic cardiomyopathy who have symptomatic New York Heart Association (NYHA) class III disease with severe left ventricular outflow tract (LVOT) gradients despite medical treatment.¹ A ventricular septal defect (VSD) after surgical treatment is rare.² Alternatives for surgical closure are available in patients with congenital VSDs or post-myocardial infarction VSDs. Percutaneous closure provides a reasonable alternative.^{3,4}

We describe the technique of percutaneous closure of a VSD after surgery for hypertrophic cardiomyopathy.⁵

Clinical Summary

A 50-year-old man was referred for surgery with symptomatic hypertrophic cardiomyopathy despite medical therapy. He was in NYHA class III with effort-related angina. Echocardiography showed asymmetrical left ventricular hypertrophy with a septal end-diastolic thickness of 19 mm, systolic anterior motion, an ejection fraction of 66%, and an LVOT gradient of 64 mm Hg. During catheterization the pressure gradient measured 100 mm Hg. Ethanol ablation failed. A surgical procedure was performed via a transaortic approach. By a Morrow septal myomectomy the LVOT was increased from 16 to 23 mm. However, on control transesophageal echocardiography the distal part of the LVOT was still

obstructive. This part being out of reach, a second attempt was made via the left atrium during the same procedure. The antero-external commissure and the A1 and A2 portions of the anterior leaflet of the mitral valve were detached from the annulus, and the myomectomy was completed distally. The anterior leaflet of the mitral valve was reattached to the annulus. Control echocardiography showed total relief of the LVOT obstruction, absence of mitral regurgitation, but a small muscular VSD. The initial post-operative course was uneventful. During the following weeks, the patient's clinical condition deteriorated, with left and right ventricular failure and mild renal and hepatic failure. On control echocardiography a muscular VSD 12 mm in diameter with a left-to-right shunt of 2:1 and half systemic right ventricular pressure was seen. At the right side of the ventricular septum, the VSD was located near the attachments of the septal leaflet of the tricuspid valve; at the left side, the distance from the aortic valve was 18 mm. The thickness of the septum surrounding the VSD varied between 10 and 14 mm.

On the 20th postoperative day, percutaneous closure of the VSD was attempted. Under fluoroscopy and transesophageal echocardiographic guidance, the VSD could be crossed via the right jugular vein and a Mullins transseptal sheath was positioned in the left ventricle. An Amplatzer 14-mm VSD occluder (AGA Medical Corporation, Golden Valley, Minn) was delivered in the VSD and released (Figure 1). The patient had a second-degree atrioventricular block necessitating temporary pacing. Hemodynamic recovery was immediate, but the postoperative course was complicated by transient hemolysis and *Candida* septicemia. One month after closure of the VSD, the patient could be dismissed. An echocardiogram showed a correct position of the VSD device, without any gradient or residual shunt across the LVOT (Figure 2).

Discussion

A VSD complicating surgery for LVOT obstruction in hypertrophic cardiomyopathy is rare.² These VSDs can be hemodynamically significant. Surgical closure of a VSD can be cumbersome in a patient who has had recent surgery. Percutaneous or percuticu-

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Figure 1. Fluoroscopic image before release of the VSD device. The device is seen on the image to the right of the transesophageal echocardiography probe in front of the vertebral column. It is fully deployed in the VSD but still attached to the delivery wire (arrow) in the sheath.

lar closure of muscular VSDs has been reported,^{3,4} with reasonable results and limited complications. Percutaneous closure has been advocated for the closure of postinfarction VSDs in adults also, but with lower success rates.⁴ Only a few reports address percutaneous closure of a VSD after surgery for hypertrophic cardiomyopathy.⁵

In this case, the percutaneous technique to close the VSD proved successful, but several complications have been encountered. Unlike this case, the procedure can be technically challenging and lead to rhythm disturbances or damage to the tricuspid valve.

Moreover, conduction tissue can run near the edges of the VSD and be damaged by manipulation of the device. The risk is more important if the VSD is located near the aortic or tricuspid valves and can result in temporary or permanent atrioventricular block.^{3,4}

Hemolysis caused by residual flow through the fabric of the device is widely known and generally self-limiting.^{3,4}

The devices do not always present with a nice flat profile after release. We hesitated before implanting such a device in a narrow LVOT, fearing obstruction. This did not occur.

Figure 2. Echocardiographic image of the device during follow-up (long axis). The left part of the device is seen as a dense crescent-like structure at the left side of the interventricular septum. The LVOT is unobstructed. LV, Left ventricle; LA, left atrium.

Most devices become completely endothelialized within 6 months after implantation. In the meanwhile, the device is foreign material in the circulation and can become infected. Any infection during this period should be aggressively treated with careful echocardiographic inspection for possible vegetations.

Conclusion

A VSD is rare after surgery or ethanol septal ablation for severe LVOT obstruction in hypertrophic cardiomyopathy. Percutaneous closure offers a reasonable therapeutic option in symptomatic patients.

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