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CASE REPORT

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Surgical management of a giant atrial septal aneurysm

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Abstract

We experienced a very rare case of surgical management of a giant atrial septal aneurysm. It is an interesting case and is supported by preoperative, intraoperative, and pathology images.

KEYWORDS

atrial septal aneurysm, atrial tachycardia, patent foramen ovale, thrombus

1 | INTRODUCTION

A 63-year-old woman presented with chest discomfort. Coronary angiography revealed vasospastic angina. Cardiac multidetector computed tomography and cardiac magnetic resonance imaging showed a 30 × 30 mm atrial septal aneurysm (ASA) protruding into the right atrium and a thrombus attached to the left side of the ASA pouch. We surgically resected the ASA because the patient was at risk for systemic thrombosis. The resection site was closed with a pericardial patch through a median sternotomy under a cardiopulmonary bypass. The postoperative course was uneventful, and the patient was discharged on postoperative day 26.

Technological improvements in transthoracic echocardiography and the widespread application of transesophageal echocardiography have simplified the identification of atrial septal aneurysms (ASAs) and the reported prevalence is now 2%-10%.[1] Although ASAs have recently become more common, they rarely require surgical resection. Herein, we describe the surgical management of a giant atrial aneurysm in a patient with a patent foramen ovale (PFO).

2 | CASE REPORT

A 63-year-old woman in good health with no past medical history presented with chest discomfort. Coronary angiography revealed vasospastic angina and transthoracic echocardiography (TTE) showed a giant ASA without a PFO, interatrial shunt, or mitral valve prolapse. Classification of this patient's ASA was Type 1R.[1] Transesophageal echocardiography (TEE) revealed trivial tricuspid regurgitation, and the pressure gradient of the tricuspid valve was 25 mm Hg. Cardiac multidetector computed tomography and cardiac magnetic resonance imaging showed that a giant ASA protruded approximately 30 mm into the right atrium and a thrombus was attached to the left side of the ASA pouch (Figure 1A,B). The patient had no heart murmur, regular rhythm, and leg swelling. She also had no symptoms except for chest discomfort, and her New York Heart Association Classification was I. The patient was started on nicorandil, aspirin, and anticoagulation therapy with warfarin. The prothrombin time/international normalized ratio (PT/INR) was controlled at 2.0-2.5. Electrocardiographic monitoring detected

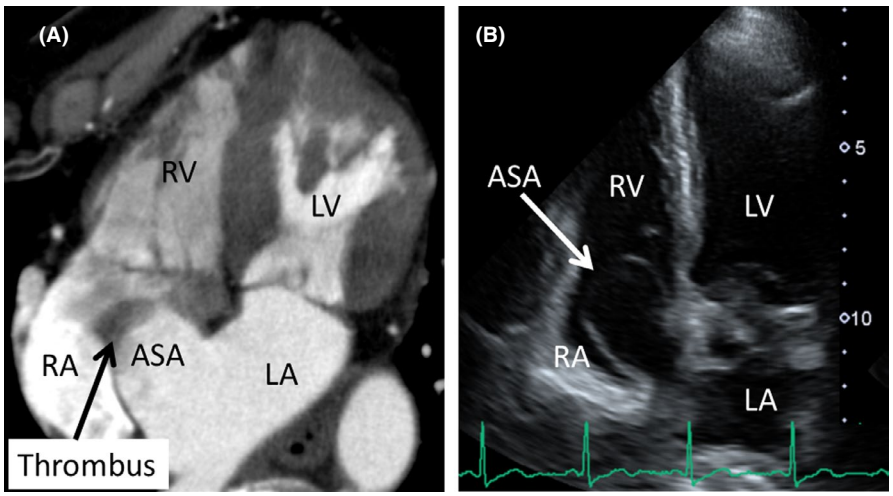

FIGURE 1 Preoperative imaging of an arterial septal aneurysm. Multi-slice computed tomography shows a giant ASA protruding into the right atrium and a thrombus attached to the left side of the ASA pouch (A). Transthoracic echocardiography shows an ASA attached to the right atrial free wall and protruding into the right atrium (B). ASA, atrial septal aneurysm; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle

occasional paroxysmal atrial tachycardia. We recommended surgical resection of the ASA because the patient was at risk for systemic thrombosis. The ASA was surgically repaired via a median sternotomy under a cardiopulmonary bypass. Visualized through a right atriotomy, the ASA protruded into the right atrium (Figure 2A). A PFO was also evident next to the ASA (Figure 2B). The aneurysm was excised, and the defect was closed with a 30 × 35 mm pericardial patch using 4-0 polypropylene sutures. No thrombus remained attached to the surface of the resected ASA. The postoperative course was uneventful, and postoperative TEE did not detect a shunt, so the patient was discharged on postoperative day 26 with normal sinus rhythm. Macroscopic assessment of the ASA showed a mixture of normal and very thin tissues that seemed vulnerable to imminent tearing (Figure 3A). Pathological assessment of the resected ASA showed that the thick tissues comprised a mixture of infiltrative fatty cells and fibrosis (Figure 3B,C). The patient is under follow-up as an outpatient and remains free of complications at 10 postoperative months.

3 | COMMENT

Atrial septal aneurysms are rare, and their prevalence varies due to variations in ASA diagnostic criteria, materials, methods, diagnostic equipment, study populations, and recognition by echographers.[1] These aneurysms are often associated with other cardiac anomalies, such as intra-arterial shunt, atrial septal defect type II, PFO, valvular prolapse, etc[2] Complications of ASA include cerebrovascular events, arrhythmia, and pulmonary hypertension. Candidates for the surgical repair of ASA and reports on ASA surgery are very rare.

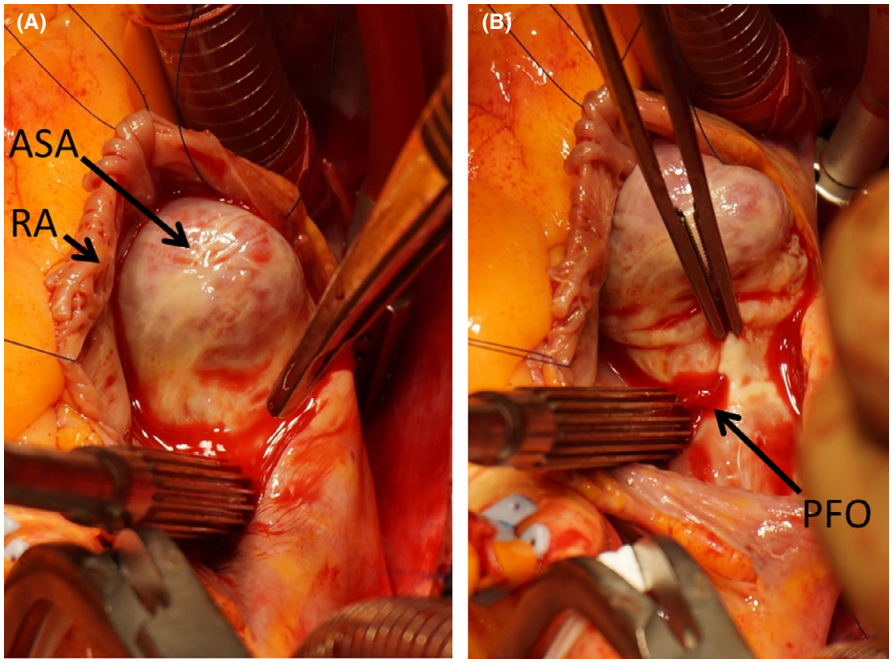

FIGURE 2 Intraoperative imaging findings. A giant atrial septal aneurysm protrudes into the right atrium (A). Patent foramen ovale (B). ASA, atrial septal aneurysm; RA, right atrium

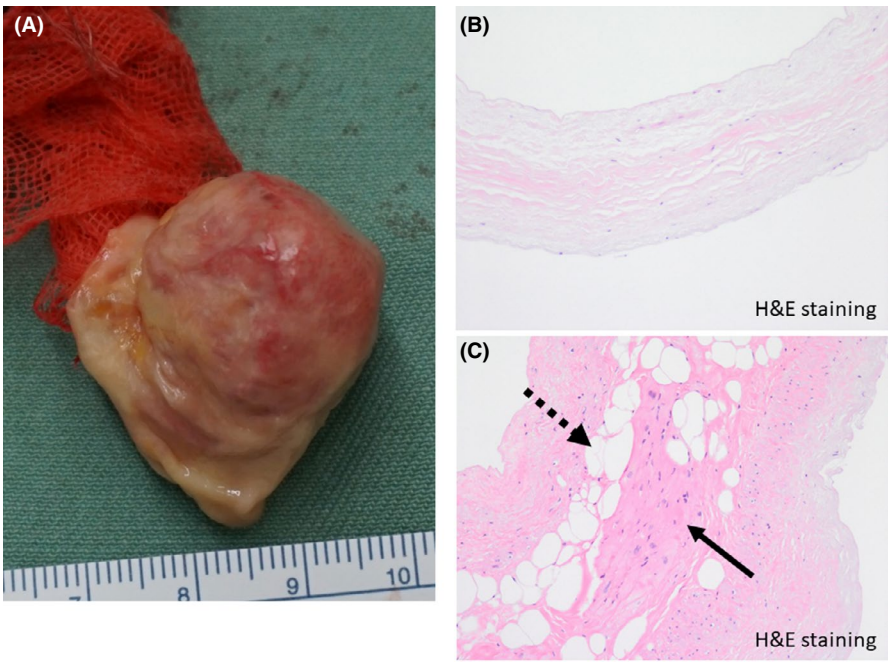

FIGURE 3 Pathological findings of an atrial septal aneurysm. Blood-stained gauze was placed inside the giant ASA to understand the nature of the thin tissue. A mixture of thickened and very thin tissues appeared vulnerable to imminent tears (A). Hematoxylin and eosin staining. (B, C) Histological section of very thin ASA tissues (B). Histological section of normal ASA tissues mixed with fatty cell infiltration (dotted arrow) and fibrosis (black arrow) (C). H&E staining, Hematoxylin and eosin staining, Magnification, ×100. ASA, atrial septal aneurysm

Stroke is one of the worst complications of ASA. Mattioli et al[3] reported that ASA is the only potential cardiac source of embolism detected by TEE in patients aged <45 years. Cabanes et al[4] reported that PFO and ASA are significantly associated with stroke in adults aged <55 years. In contrast, Shinohara et al described a thrombus attached to the left side of an atrial septal aneurysm that disappeared with anticoagulation therapy, but the aneurysm was eventually excised and repaired with an atrial patch because of the risk of recurrent thrombus and the need for lifelong anticoagulation therapy.[5] The thrombus that was attached to the left side of the ASA pouch in our patient disappeared with anticoagulation therapy. We identified a PFO next to the ASA during surgery; however. Because our patient was at risk for cardiogenic embolism with an interatrial thrombus and PFO, we decided on surgical management of the ASA. We consider a large ASA that has a risk of thrombus with a PFO to be an indication for surgical correction.

Interatrial shunts are complications of ASA, and their prevalence is 54.4%-77%. One risk of cardiogenic embolism with ASA is an interatrial shunt.[2,6] Pathological and macroscopic findings showed that some parts of the wall were so thin that rupture appeared imminent. The cause of the interatrial shunt in our patient remains uncertain, but it might have been due to rupture of the weakened atrial septal wall due to long-term bulging and stretching.

Atrial tachycardia is another complication of ASA, with a prevalence of 18%-25%.[1,2,7] The cause of arrhythmias in patients with ASA is not clear, but cardiac abnormalities, such as hypertension, atrial enlargement, systolic dysfunction, or valvular prolapse, might be responsible.[1] Although atrial tachycardia in patients with ASA is not a risk for cardiac embolism per se, ASA does confer thromboembolic potential, and long-term anticoagulant therapy is indicated for patients with ASA and history of embolic events.[2,6]

The incidence of mitral valve prolapse associated with ASA is 12%-20.5%,[1,2,7,8] and the cause is also unclear. Mitral valve prolapse and ASA might have a similar pathological basis, namely, a connective disorder involving fibrous cardiac tissue.[1] The connective tissue of an ostium primum atrial septum can become defective, especially when patients have myxomatous degeneration of the mitral valve. As a result, a weakened atria1 septum might lead to outpouching of the atrial septal wall.[4] Taking these factors into consideration, echocardiography did not reveal mitral valve prolapse or regurgitation in our patient, but our patient has a potential risk of mitral valve prolapse.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

MK: treated the patient, captured the images, co-conducted the literature review, and co-wrote the paper. JN: treated the patient and edited the paper. MT: treated the patient. ES: analyzed the case and co-wrote the paper. KM: analyzed the case and co-wrote the paper.

ETHICAL APPROVAL

An informed consent was obtained from the patient for publication.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author.

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