Chapter 23

Atrial Septal Aneurysm

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Additional information is available at the end of the chapter

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1. Introduction

ASA is a rare entity incidentally diagnosed during conventional transthoracic echocardiography (TTE). It is defined as the presence of redundant and mobile interatrial septal tissue extending to at least 15 mm during the cardiorespiratory cycle. The incidence of ASA has been reported at about 2% in patients undergoing TTE [1]. Patent foramen ovale (PFO) and ASA have been cited as potential risk factors for cryptogenic stroke. For example, ASA was observed in 7.9% of patients with a history of possible embolic stroke. Most patients with previous cerebral ischemic events and ASA also have an interatrial shunt, usually via PFO. Interatrial shunt has been reported in 56-78% of patients with ASA [2]. To our knowledge, there have been few reports of surgical intervention in ASA, for which the surgical indications are not yet defined. We describe herein two cases of surgical repair of giant ASA.

2. Case report 1

A 59-year-old Asian female referred to our surgical team was admitted to our hospital for investigation of ASA after complaining of frequent palpitations starting eight years previously. ASA had been confirmed two years earlier in an examination for palpitation, to which the patient was very sensitive, making frequent visits to the emergency department. The arrhythmia consisted of paroxysmal atrial fibrillation (AF), which was refractory to antiarrhythmic medication. The medication did not include any anticoagulant or antiplatelet agents. Physical examination was normal. Auscultation detected no murmurs, rubs, or gallops, but a split S1 was noted. Laboratory data on admission were within normal limits except for slightly elevated liver enzyme, possibly due to chronic hepatitis C. Initial EKG showed no abnormality. Chest radiograph demonstrated a cardiothoracic ratio of 0.50 and no remarkable findings. TTE revealed a giant ASA with mobility into the right atrium and nearly prolapsing into the tricuspid orifice (Figure 1). It also showed a mildly dilated right

ventricle with no valvular dysfunction. Right ventricular systolic pressure was calculated to be 43 mm Hg. Chest computed tomography (CT) with contrast dye showed 47×22 mm of protruding tissue at the site of the atrial septum. Transesophageal echocardiogram demonstrated PFO at a site close to the superior vena cava and ascending aorta. In view of the enlarged right ventricle and paroxysmal AF, in addition to the high risk of stroke, surgical repair was recommended and performed.

The surgical approach was through medial sternotomy. Cardiopulmonary bypass was established and bilateral pulmonary vein isolation was performed with a bipolar radiofrequency device. Right atriotomy was then carried out. The aneurysm lay next to the fossa ovalis, enabling detection of PFO (Figure 2). The aneurysm in the interatrial septum was removed, a right atrium maze procedure was performed, and the defect was closed with a 4-0 polypropylene running suture.

The patient tolerated surgery very well and had an uneventful postoperative recovery without occasional paroxysmal AF. A postoperative MRI was performed, but no shunt flow was detected. TTE showed the same result. The patient was discharged uneventfully after surgery and remains symptom-free and in good health at two years postoperatively.

Macroscopically, the mass consisted of a thin protrusion of the atrial septum. The histological results from the septum showed a degenerative cardiac muscle with fibrosis. There was no evidence of atherosclerosis, specific inflammation, or tumorous lesion.

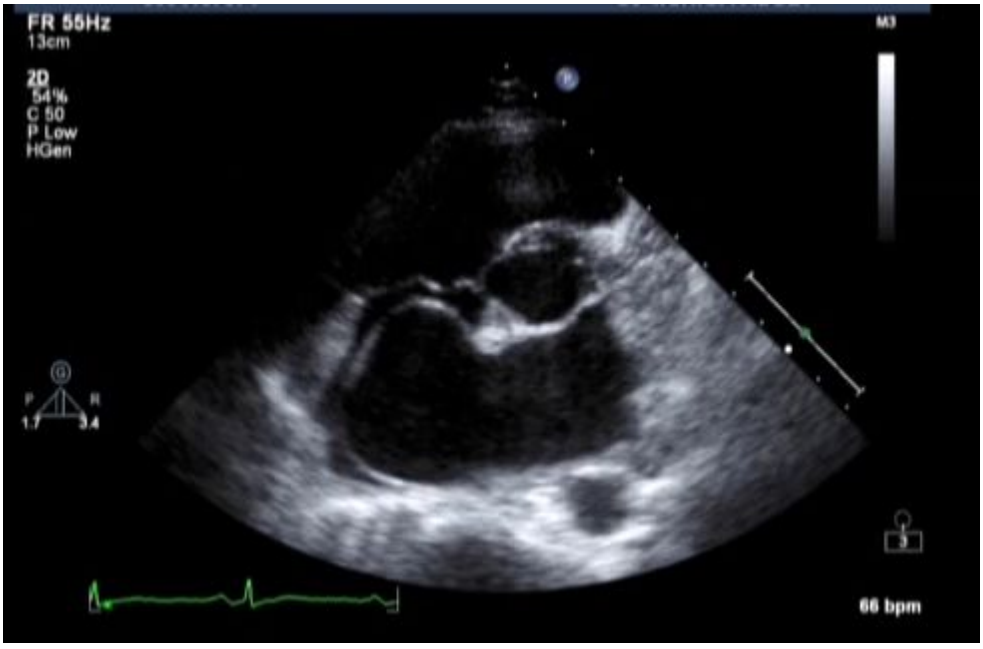

Figure 1. Giant atrial septal aneurysm (47×23 mm) with mobility into the right atrium nearly prolapsing into the tricuspid orifice

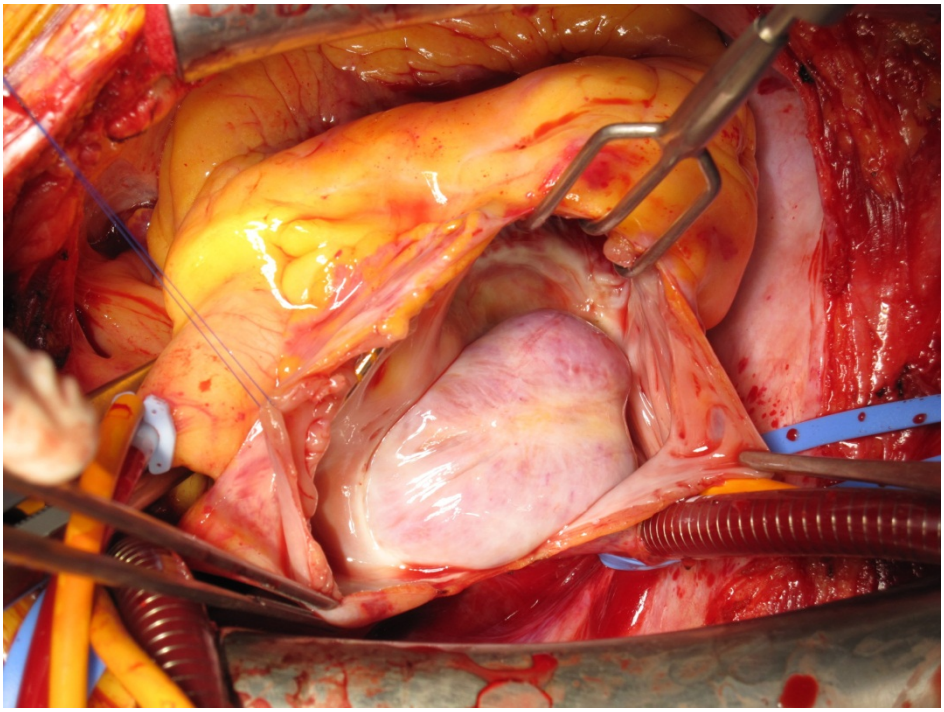

Figure 2. Intraoperative picture showing 50×25 mm of protruding tissue at the site of the atrial septum

3. Case report 2

A 37-year-old Asian woman with a 10-month history of general malaise and dyspnea was referred to our division. The patient had been well until a month earlier, when she began to have episodes of chest oppression. Transthoracic echocardiography showed almost normal wall motion without valvular dysfunction apart from the unusual feature of atrial septal defect (ASD) (Figure 3). It showed the atrial septum extending into right atrium and multidirectional right to left shunt flow using the color Doppler image. The ejection fraction was 64% and the shunt ratio was 50% (Qp/Qs=2.0). The patient was referred to our surgical team as a case of ASD.

The patient underwent an ASD closure. Following medial sternotomy, cardiopulmonary bypass was established. Right atriotomy was then carried out. The defect appeared to resemble ASD secundum, but protruded as seen in ASA had two large cribriform holes and numerous small pinholes (Figure 4). The aneurysm in the interatrial septum was removed and the defect was closed with a 4-0 polypropylene running suture.

The patient tolerated surgery very well and had an uneventful postoperative recovery without symptoms. The patient was discharged uneventfully after surgery and remains symptom-free and in good health at 12 months postoperatively.

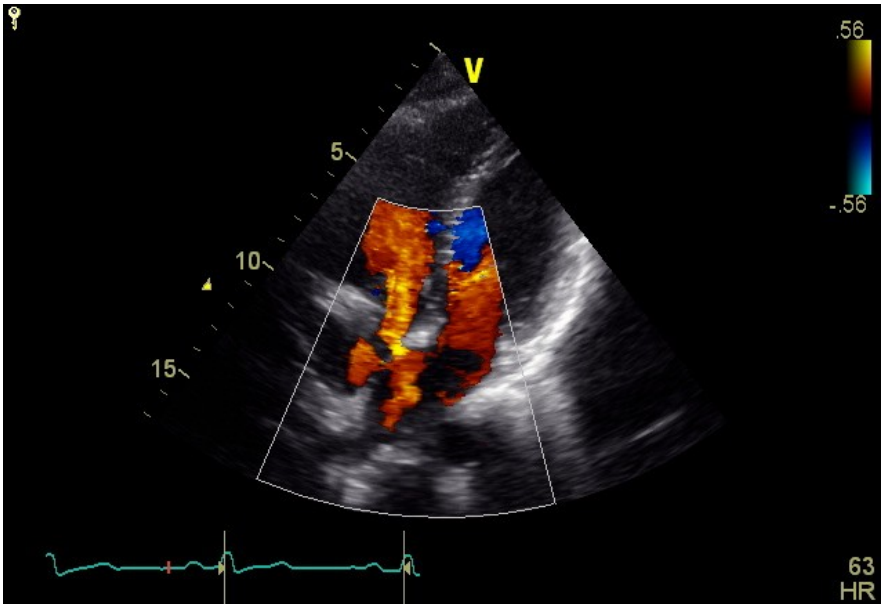

Figure 3. Echocardiography showing shunt flow through atrial septal defect. The multiple direction of the flow suggested the presence of a number of holes in the atrial septum.

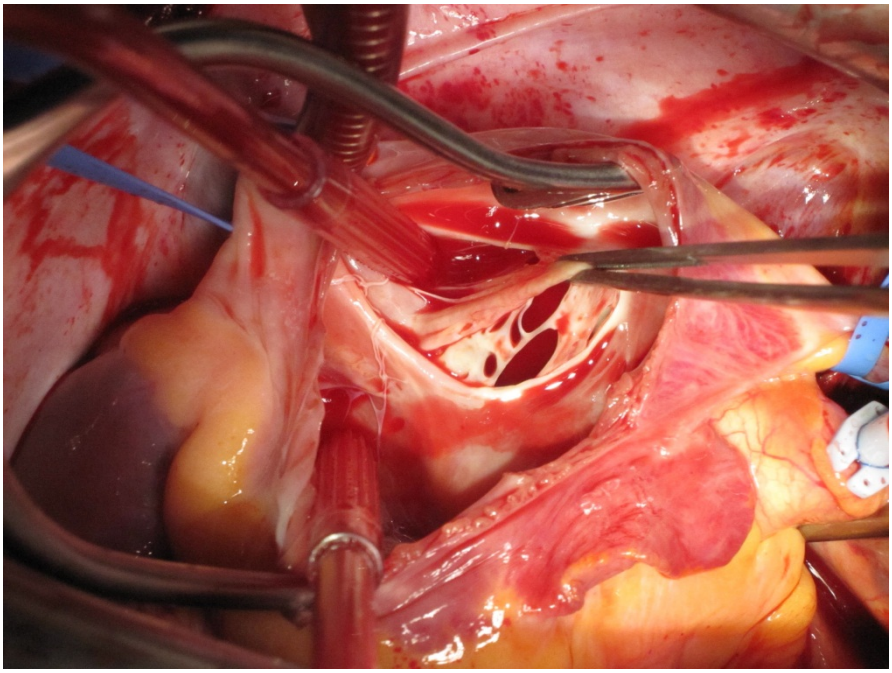

Figure 4. Figure 4. Atrial septum with numerous small pinholes and cribriform atrial septal defect

4. Discussion

The incidence of ASA has been found to be higher after a cerebral ischemic event in patients evaluated with transesophageal echocardiogram. A meta-analysis of case-control studies found that the presence of a PFO, ASA, or both was significantly associated with ischemic stroke in subjects less than 55 years of age [2, 3]. It is reported from PFO-ASA study that the presence of PFO together with ASA is a significant predictor of recurrent stroke [4]. Aggressive therapy such as warfarin or surgical repair may be the best option in such patients, but this question needs to be assessed in randomized clinical trials. The 2004 American Academy of Neurology practice parameter concluded that the combination of PFO and ASA increases the risk of subsequent stroke in medically treated patients below age 55 compared with other cryptogenic stroke patients without atrial abnormalities. It also concluded that there is insufficient evidence to evaluate the efficacy of surgical or endovascular closure [5].

The pathological mechanisms that lead to the development of ASA have not yet been clarified. To explain the association between ASA and cryptogenic stroke, two mechanisms have been proposed. Because of the frequency of intraatrial shunt, paradoxical embolism may occur. In patients with ASA without intracardiac shunt, it has been hypothesized that direct thrombi form within the aneurysm or as a result of atrial fibrillation, causing embolism [6].

Surgery is seldom performed for ASA patients. Shinohara and colleagues reported on a three-year follow-up of ASA [7], while Aoyagi and colleagues reported on a case of ASA and stenotic mitral valve [8]. In these two cases ASA was successfully removed and the atrial septum repaired with a pericardial patch. The reports concluded that surgery may be considered as an alternative therapy for patients with atrial arrhythmia and ASA.

The present cases occurred in patients without history of stroke, but who had numerous strong predictors of cryptogenic stroke, including ASA, PFO, ASD, and AF. The right ventricle was mildly dilated and right ventricular pressure mildly elevated in one case. Although the indications for surgical treatment of ASA and PFO remain undetermined, we considered that the symptoms were unlikely to resolve and that surgical intervention was the only curative treatment available. We reported in the above on cases of ASA. We believe surgical repair should be considered for giant ASA to reduce the future risk of cerebral embolism or heart failure.

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5. References

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