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Surgical Repair of a Giant Atrial Septal Aneurysm with Patent Foramen Ovale

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ABSTRACT

Atrial septal aneurysm (ASA) is a rare occurrence. Many studies have established a correlation between ASA and both ischemic stroke of unidentified cause and cardiac embolism. We describe the case of a 59-year-old woman who had a giant ASA with patent foramen ovale. The aneurysm was successfully removed. Although the detailed mechanism involved in the degeneration of the atrial septum is unclear, we recommend that such damage be surgically repaired to reduce the risk of cerebral embolism or heart failure in symptomatic patients. Surgery is recommended for larger ASA.

INTRODUCTION

Atrial septal aneurysm (ASA) is a rare entity incidentally diagnosed during conventional transthoracic echocardiography (TTE). It is defined as redundant and mobile interatrial septal tissue extending to at least 15 mm during the cardiorespiratory cycle. The incidence of ASA has been reported to be approximately 2% of patients undergoing TTE [Hanley 1985]. 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 a PFO. An interatrial shunt has been reported in 56% to 78% of patients with ASA [Agmon 1999]. To our knowledge, there have been few reports of surgical intervention in ASA for which the surgical indications are not yet clear. We describe a case of surgical repair of a giant ASA with PFO.

CASE REPORT

A 59-year-old Asian woman referred to our surgical team was admitted to our hospital for investigation of an ASA after she began complaining of frequent palpitations starting 8 years previously. An ASA had been confirmed 2 years earlier in an examination for palpitations. The patient was very sensitive to palpitations and made 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. The results of a physical examination were normal. Auscultation detected no murmurs, rubs, or gallops, but a split S1 was noted. Laboratory data on her admission were within normal limits except for slightly elevated liver enzymes, possibly due to chronic hepatitis C. The initial electrocardiogram showed no abnormality. A chest radiograph demonstrated a cardiothoracic ratio of 0.50 and no remarkable findings. A TTE evaluation revealed a huge ASA with mobility into the right atrium and nearly prolapsing into the tricuspid orifice (Figure 1). The TTE also showed a mildly dilated right ventricle with no valvular dysfunction. The right ventricular systolic pressure was calculated to be 42.7 mm Hg. A chest computed tomography scan with contrast dye showed protruding tissue measuring 47 × 22 mm at the site of the atrial septum. A transesophageal echocardiogram demonstrated a PFO at a site close to the superior vena cava and the ascending aorta. In view of the enlarged right ventricle and the paroxysmal AF, in addition to the high risk of stroke, a surgical repair was recommended and performed.

The surgical approach was through a medial sternotomy. Cardiopulmonary bypass was established, and bilateral pulmonary vein isolation was performed with a bipolar

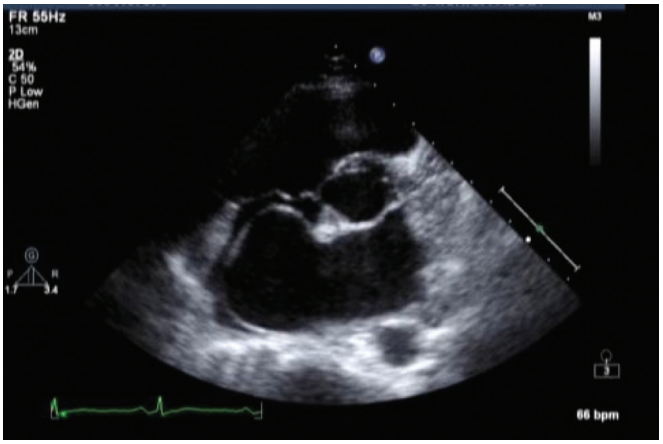

Figure 1. Huge atrial septal aneurysm (47 × 23 mm) with mobility into the right atrium and nearly prolapsing into the tricuspid orifice.

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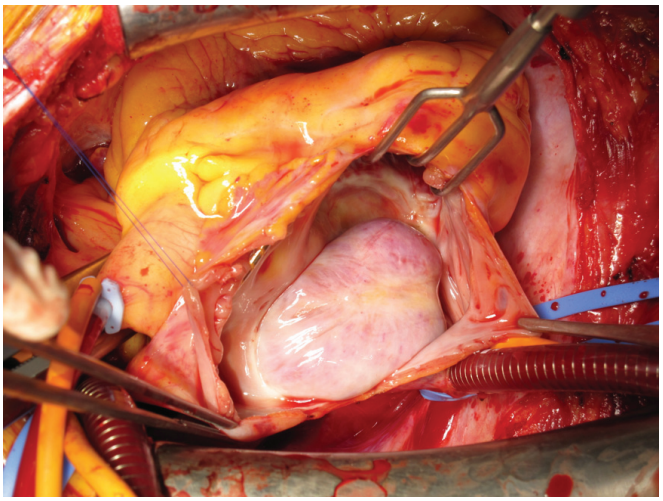

Figure 2. Intraoperative photograph showing protruding tissue (50 × 25 mm) at the site of the atrial septum.

radiofrequency device. A right atriotomy was then carried out (Figure 2). The aneurysm lay next to the fossa ovalis, enabling detection of the PFO. 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 the surgery very well and had an uneventful postoperative recovery without occasional paroxysmal AF. A postoperative magnetic resonance imaging examination was performed, but no shunt flow was detected. A TTE showed the same result. The patient was discharged uneventfully after surgery and remains symptom free and in good health at 7 months postoperatively.

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

DISCUSSION

The incidence of ASA is higher after a cerebral ischemic event in patients evaluated with a 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 patients younger than 55 years [Agmon 1999; Overell 2000]. Study of ASA with PFO has revealed that the presence of a PFO together with an ASA is a significant predictor of recurrent stroke. Aggressive therapy, such as warfarin therapy or surgical repair, may be the best option in such patients, but this question needs to be assessed in randomized clinical trials [Mas 2001]. 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 younger than 55 years of age, 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 [Messe 2004].

The pathologic mechanisms that lead to the development of ASA have not yet been clarified. Two mechanisms have been proposed to explain the association between ASA and cryptogenic stroke. Because of the frequency of intra-atrial shunt, paradoxical embolism may occur. In patients with an ASA without an intracardiac shunt, thrombi have been hypothesized to form directly within the aneurysm or as a result of AF, causing embolism [Schneider 1990].

Surgery is seldom performed for ASA patients. Shinohara and colleagues reported on a 3-year follow-up of an ASA patient [Shinohara 2001], and Aoyagi and colleagues reported on a case of ASA and a stenotic mitral valve [Aoyagi 2009]. In these 2 cases, the ASA was successfully removed, and the atrial septum was repaired with a pericardial patch. The reports concluded that surgery may be considered an alternative therapy for patients with atrial arrhythmia and ASA.

The present case occurred in a patient without a history of stroke but who had numerous strong predictors of cryptogenic stroke, including ASA, PFO, and AF. The right ventricle was mildly dilated, and the right ventricular pressure was mildly elevated. Even though 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 have reported on this case of a giant ASA with PFO. We believe surgical repair should be considered for giant ASA to reduce the risk of cerebral embolism or heart failure, even in asymptomatic patients.

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