

## IMAGING

### IMAGING VIGNETTE: CLINICAL VIGNETTE

# Giant Aneurysm of the Interatrial Septum

## A Multidisciplinary Approach



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### ABSTRACT

A 46-year-old woman presented for a routine echocardiogram, which revealed a giant atrial septal aneurysm (ASA) with an atrial septal defect (ASD). We surgically excised the ASA and closed the ASD with a pericardial patch. ASA, although rare, poses embolic risks, thus necessitating closure when associated with shunts. Optimal stroke prevention remains uncertain. (J Am Coll Cardiol Case Rep 2024;29:102206) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

A 46-year-old woman with a history of rheumatic fever was referred to our hospital (Puerta de Hierro University Hospital, Majadahonda) for routine transthoracic echocardiography (TTE). She was asymptomatic. On physical examination, we found a pulmonary flow murmur. The electrocardiogram showed sinus rhythm and an incomplete right bundle branch block with right axis. Blood values were normal. A Holter monitor ruled out arrhythmic events. TTE revealed a giant, multiperforated atrial septal aneurysm (ASA) (approximately 8 cm in diameter) that protruded into the right atrium and prolapsed through the tricuspid valve, causing mild regurgitation (**Figure 1A**, **Video 1**). A 3-cm atrial septal defect (ASD) with left-to-right shunting was identified with color flow Doppler imaging (**Figure 1B**, **Video 1**). The right ventricle was mildly enlarged, but systolic function was preserved. No pulmonary hypertension was detected. Transesophageal echocardiography (TEE) confirmed the results (**Figure 1C**, **Videos 2 and 3**), and cardiac magnetic resonance estimated a pulmonary-to-systemic flow ratio (Qp/Qs) of 1.5 (**Figure 1D**, **Video 4**). Aspirin, 100 mg daily, was prescribed for stroke prevention, and the patient underwent surgery to excise the aneurysm and close the ASD with a pericardial patch. The tissue measured 8 cm and contained multiple fenestrations (**Figure 1E**). The postoperative period was uneventful, and there was no residual shunt noted on follow-up TTE. Antiplatelet therapy was discontinued postoperatively. Currently, the patient remains asymptomatic and without arrhythmic events.

ASA is a congenital abnormality characterized by mobile and redundant interatrial septal tissue located near the fossa ovalis, exhibiting phasic excursion of at least 10 to 15 mm beyond the plane of the atrial septum. Prevalence ranges from 1% to 4.9% depending on the studied population and diagnostic method<sup>1</sup>; however, giant aneurysms such as that seen in our patient are rare. Diagnosis can be attained by TTE, but TEE offers greater sensitivity because it provides a clearer view of the interatrial septum. ASAs are

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

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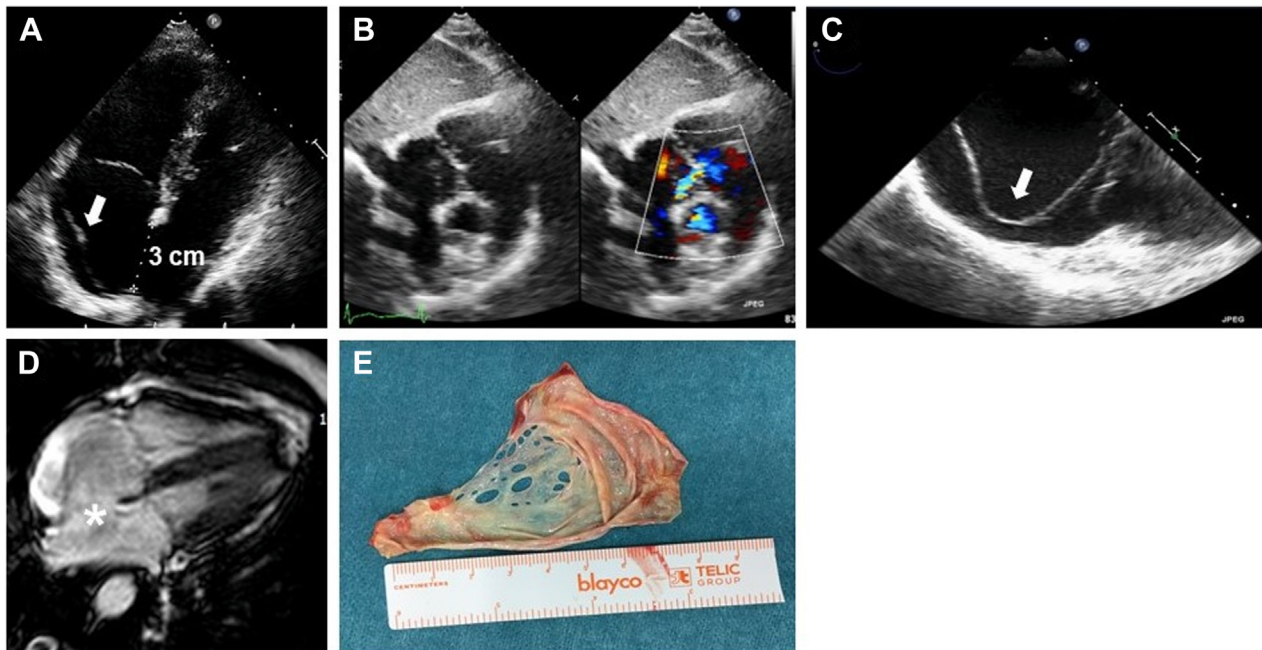
**ABBREVIATIONS  
AND ACRONYMS****ASA** = atrial septal aneurysm**ASD** = atrial septal defect**TEE** = transesophageal  
echocardiography**TTE** = transthoracic  
echocardiography

commonly discovered incidentally in asymptomatic patients. However, evidence suggests that ASA may be a source of embolisms causing ischemic cerebral and/or peripheral events,<sup>1,2</sup> and it has been associated with atrial tachyarrhythmias and other congenital defects (ASD, patent foramen ovale, or mitral valve prolapse) that increase the embolic risk.<sup>2</sup> The association of ASA with cryptogenic stroke is explained by 2 mechanisms. First, in patients with ASA and patent foramen ovale or ASD, there is a risk of paradoxical embolism through the septal defect. Second, in patients with ASA but without intracardiac shunts, it is theorized that fibrin-platelet particles may adhere to the left atrial side of the aneurysm and become dislodged in response to aneurysm oscillations, thereby leading to systemic embolism. Regarding treatment, uncomplicated, isolated ASA requires no specific therapy other than follow-up. In the presence of a shunt, however, closure is recommended to prevent recurrent embolisms.<sup>2</sup> The optimal medical treatment for stroke prevention is unknown. In the present case, a multidisciplinary team decided that surgical closure of the ASD was the best approach. According to the latest guidelines for the management of congenital heart disease, ASD closure is recommended, regardless of symptoms, when the patient presents with right ventricular volume overload without pulmonary arterial hypertension or left ventricular disease (Class I, Level of Evidence: B),<sup>3</sup> as observed in our patient.

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**FIGURE 1** Giant Aneurysm of the Interatrial Septum

(A) Transthoracic echocardiography 4-chamber view showing the atrial septal defect (arrow), measuring 3 cm in diameter. (B) Transthoracic echocardiography subcostal view (left) with color Doppler (right) showing left-to-right shunting through the atrial septal defect and mild tricuspid regurgitation. (C) Transesophageal echocardiography 90° bicaval view, displaying the atrial septal aneurysm (arrow). (D) Cardiac magnetic resonance imaging, 4-chamber view, confirming the presence of the atrial septal defect (asterisk). (E) Intraoperative image displaying the resected multiperforated interatrial septal aneurysm, measuring 8 cm.

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**KEY WORDS** atrial septal aneurysm, atrial septal defect, cardiogenic embolism, echocardiography

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 **APPENDIX** For supplemental videos, please see the online version of this paper.