Atrial septal aneurysm and spontaneous echo contrast: An association with higher embolic risk?

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Abstract We report the transesophageal echocardiogram of a patient who was admitted to the intensive care unit with a diagnosis of acute ischemic stroke, in whom a large atrial septal aneurysm (ASA) was seen bulging toward the right atrium with marked spontaneous echo contrast (SEC) inside but without thrombi, suggesting that ASA and SEC could represent a new association with higher embolic risk.

INTRODUCTION

In the population older than age 55, ischemic stroke is most often caused by atherosclerotic disease of the large vessels and/or hyaline disease of the small intra-cerebral vessels related to hypertension and diabetes.

Cryptogenic stroke occurs more frequently in young adults. Pathological studies have emphasized the association between an aneurysm of the interatrial septum and stroke in up to 40% of cases. Among such abnormalities, patent foramen ovale (PFO) (a potential cause of right-to-left shunt) and atrial septal aneurysm (ASA) should be underscored. Several autopsy series have reported a prevalence of 17–27% for PFO, and 2–4% for ASA; studies performed with transesophageal echocardiography (TEE) revealed similar findings.

The mechanisms proposed as responsible for systemic embolism are: (1) paradoxical embolism, with passage of thrombi from the peripheral venous system to the left cardiac cavities through a septal defect. (2) Thrombi formation in the atria as a consequence of possible atrial arrhythmias,
often associated to ASA and PFO. (3) Thrombi formation within the aneurysm.

In the third setting, an ASA could be responsible for blood stasis inside the aneurysm, since it involves a low-velocity chamber. Its presence is confirmed with an echocardiogram by documenting "smoke" or spontaneous echo contrast (SEC). Hence, there might be a relation between the larger size and/or reduced motion of the ASA and the magnitude of SEC detected.

In this report, we describe a 53-year-old patient who presented with an ischemic stroke, and in whom the transesophageal echo revealed a large ASA with SEC inside.

Case report

The patient is a 53-year-old male with a history of hypertension and smoking, who was admitted to the intensive care unit with a diagnosis of right motor faciobrachiocrural deficit and dysarthria. The brain CT showed an ischemic image which measured 10 mm in diameter in the left thalamus. There were no significant lesions in the neck vessels. The transthoracic echocardiogram (TTE), with suboptimal acoustic windows, revealed an ASA (Fig. 1A), which was confirmed by TEE (Fig. 1B); it measured 24 mm, bulged into the right atrium and was associated to a PFO, with mild right-to-left shunt, which was only visualized following a Valsalva maneuver. The TEE in the 85° bicaval view showed a large ASA involving the entire atrial septum (Fig. 2), and exhibited only mild respiratory motion but never crossed the septal midline; there was spontaneous echo contrast (SEC) inside without thrombi. No SEC was observed outside the ASA after increasing the gain setting. Left ventricular function, left atrial dimensions and flow velocity in the left atrial appendage were within normal ranges. Mitral inflow and pulmonary venous flow velocities were normal.

Venous Doppler-echo of the lower limbs and pelvis was normal, and allowed to rule out a potential paradoxical embolism through the PFO.

All the tests performed to rule out a hypercoagulability state were normal: antiphospholipid antibodies (anticardiolipin and lupus anticoagulant), Protein S, Protein C, Antithrombin III, Plasminogen and Fibrinogen. Anticoagulation was started.

After hospital discharge, the patient presented with a new episode of right faciobrachial paresis in spite of anticoagulation; therefore, an angio-MR of the neck vessels and brain was ordered.

No vascular malformations, areas of stenosis in the proximal vessels or significant carotid lesions were detected. There was vertebral asymmetry, secondary to a decreased diameter of the left vertebral artery and thus, a dominant right vertebral artery. The MR reported a hyperintense image in the left thalamogeniculate region, compatible with an ischemic sequela in the area of the perforator artery of the vertebrobasilar system.

Figure 1  Transthoracic echocardiogram (A) and transesophageal echocardiogram (B): four-chamber view showing a large atrial septal aneurysm (ASA) bulging toward the right atrium (RA). LA: left atrium, RV: right ventricle, LV: left ventricle.
Discussion

The causes of ischemic stroke are many, and the etiology varies according to the age group considered. Various structural cardiac diseases, arrhythmias and hematological factors may be involved in its origin.

In recent years, several publications have recognized new cardiac malformations that are responsible for ischemic neurological events. Findings such as ASA and PFO may be found alone or in association and may potentially be responsible for embolic episodes in young adults, aged less than 55 years.

The study by Mas et al. \(^5\) assessed the risk of new embolic events in patients with a history of prior cryptogenic stroke and abnormalities of the interatrial septum, and the association of ASA with PFO was a clear indicator of risk for recurrence, in spite of aspirin treatment (the risk of recurrent ischemic stroke at 4 years in patients who only had PFO was 2.3% versus 15.2% in patients with an association of PFO and ASA).

In contrast, Homma et al. \(^6\) did not document a higher risk for new stroke in patients who had both PFO and ASA (14.5% recurrence of stroke in patients with PFO versus 15.9% in patients with the association of PFO and ASA).

Of note, there were important differences between the populations of both studies. Homma studied an older population and included all types of stroke; hence, such variables may have had an impact on the results.

In a recent review of the most relevant papers on this subject, Messe et al. \(^7\) state that PFO as a single finding does not convey a risk for a worse outcome. There was no conclusive information regarding isolated ASA, and results about the prognosis of the association were inconsistent.

Since SEC is an independent predictor of high embolic risk in other disorders such as atrial fibrillation, mitral stenosis, etc., it would be reasonable to suspect that its association with a large, non-mobile ASA would provide an anatomic and rheological substrate for in situ thrombus formation.

Normal mitral inflow and pulmonary venous flow velocities indicated normal left atrial pressure, but continuous bulging of the ASA into the right atrium suggested that left atrial pressure was higher than the right atrial pressure.

Left ventricular function, left atrial dimensions and flow velocity in the left atrial appendage were within normal ranges, so the presence of paroxysmal atrial arrhythmias as a concomitant cause of thrombus formation is less likely.

A new event in the same vascular territory favors the presence of disease of the small intracerebral vessels, but a cardioembolic source of emboli should not be ruled out. Hence, we believe that according to the mechanisms proposed for the origin of cardiac embolism with abnormalities of the interatrial septum, SEC could be considered as a new risk marker to be taken into account when making treatment decisions.

Although until present there is insufficient evidence to decide whether aspirin treatment is better than anticoagulation for secondary prevention of ischemic stroke, \(^8\) the finding of SEC inside an ASA could favor our choice of the second strategy. Therefore, in our patient, we decided
to prescribe anticoagulation after the initial evaluation and continued treatment in spite of the symptomatic recurrence.

When faced with a young patient who survives an ischemic stroke and repeats new episodes of neurological deficits during follow-up in spite of anticoagulation and with no other apparent cause, several authors have proposed the surgical or percutaneous closure of the septal defect.8

In the present case, our patient was borderline with regard to age, had a small PFO and other possible associated causes of neurological disease, all of which kept us from adopting a more invasive attitude. Today, 6 years after the second ischemic episode, the patient is alive and without recurrence of symptoms.

References


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