Iranian Heart Journal; 2016; 17 (2)

Case Report

Patent Foramen Ovale: A Source of Emboli or an Innocent Bystander?

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ABSTRACT

Although a patent foramen ovale (PFO) has been suggested as a risk factor for recurrent strokes, many stroke patients with PFOs have another source of emboli that remains unidentified if the PFO is considered the only embolic source. We report a case of stroke due to emboli from an aortic aneurysm with a concomitant PFO. ***(Iranian Heart Journal 2016; 17(2):48-52)***

Keywords: Patent foramen ovale ■ Stroke

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Received: April 11, 2016 **Accepted:** June 18, 2016

A patent foramen ovale (PFO) is a congenital cardiac anomaly that often persists until adulthood. Although the majority of patients with PFOs are asymptomatic, some may show a variety of clinical manifestations—above all, cryptogenic stroke.[1-3] A PFO enables paradoxical emboli via right-to-left shunting when the pressure in the right atrium exceeds that in the left atrium. PFOs were found in 27% of the general population in an autopsy study.[4,5] The prevalence of PFOs is higher in patients with cryptogenic stroke, particularly in those < 55 years of age: in these patients, PFOs are more likely to be an incidental finding.[6,7] Approximately 40% of ischemic strokes in adults <55 years old are cryptogenic.[8,9]

For all the studies available in the existing literature, the role of PFOs as a risk factor for stroke is still widely debated**.** Therefore, although PFOs may be associated with stroke, some may present by chance in patients with stroke and do not benefit from closure.[6,7,10] In other words, the closure of innocent PFOs not only causes procedural risk[11] with no benefits for the patient, but also means that the cause of the stroke has remained unidentified.[12]

Studies have shown that a considerable proportion of stroke patients (52**.**9%) with PFOs had other sources of emboli such as cardiac sources and atherosclerotic lesions in the aortic arch.[12] We report a case of stroke due to emboli originating from an aneurysm of the ascending aorta that had a PFO coincidentally.

CASE REPORT

A 62-year-old man, who was a known case of coronary artery disease and had undergone coronary artery bypass grafting (CABG) 6 months previously, presented with a transient and sudden onset of left-sided hemiparesis. In the neurological examination, the cranial nerves were intact, muscle strength was 2/5 in the upper left extremity and 4/5 in the lower left extremity, deep tendon reflex was normal, and plantar reflex was negative. Contrast computed tomography (CT) scan of the brain showed a hypodense area in the right frontal lobe. Embolic stroke was diagnosed for the patient. The levels of antiphospholipid antibodies were within the normal range, and Doppler sonography of the neck arteries was normal. The patient was referred to our center for an evaluation of the source of the emboli.

Transthoracic echocardiography was normal; however, in the contrast study, a large number of contrast bubbles passed from the right atrium into the left atrium. We, therefore, performed transesophageal echocardiography (TEE) for the patient: it revealed a large PFO and a right-to-left shunt identified by color Doppler study and agitated saline injection (Fig. 1 and 2).

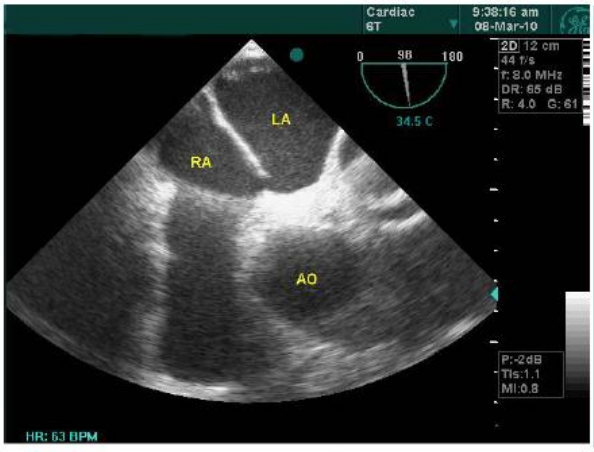


Fig. 1: Transesophageal echocardiography (TEE) in four chamber view revealed a large patent foramen ovale.LA, left atrium; RA, right atrium; AO, aorta

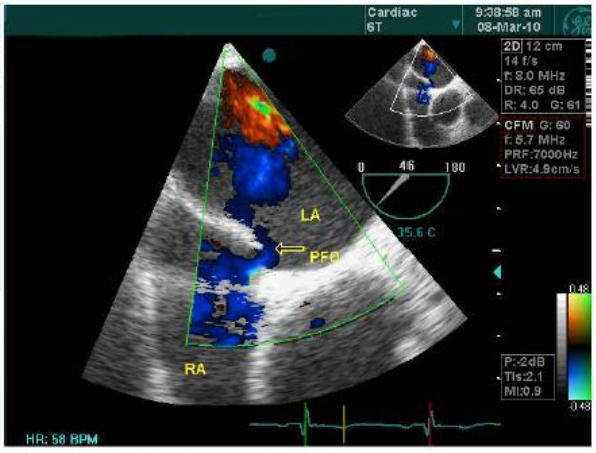
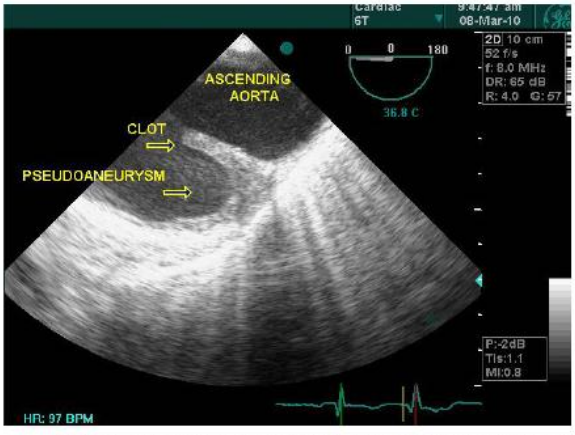
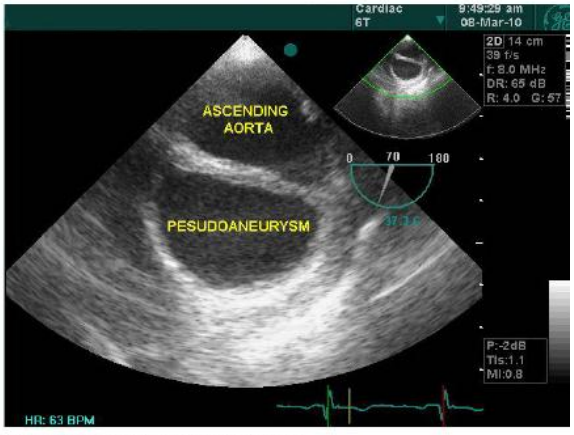


Fig. 2: Color Doppler imaging revealed an interatrial left to right shunt across a patent foramen ovale (PFO). LA, left atrium; RA, right atrium

Nonetheless, upon further examination, a saccular aneurysm was detected in the distal part of the ascending aorta (Fig. 3 and 4).

Multiple fixed and mobile hypoechoic small masses filled the aneurysm, suggesting thrombosis. No other source of emboli was detected by TEE. CT angiography established the diagnosis (Fig. 5).





.Fig. 3, 4: TEE image of ascending aorta. Two dimensional echo in four chamber view detect a saccular aneurysm with a large thrombus

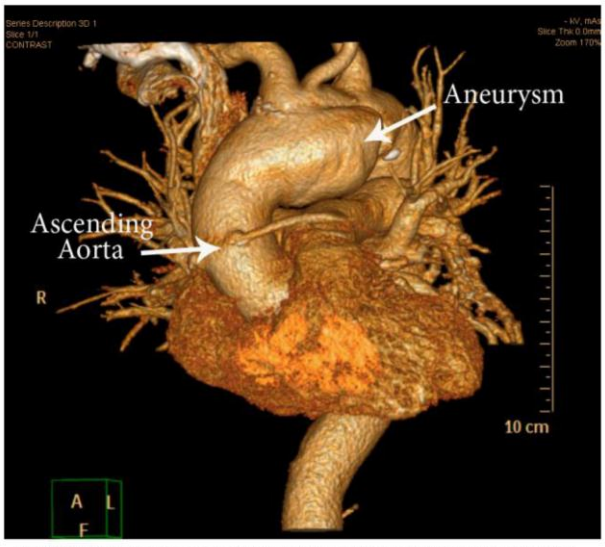


Fig.5 : CT angiography of ascending aorta showed the aneurysm in the site of cannulation of the ascending aorta during coronary artery bypass graft.

DISCUSSION

The association between PFOs and strokes remains unconfirmed, especially in patients > 55 years old.[13] Approximately one-third of PFOs are found in the general population.[4] Moreover, studies have shown that only 3.2% of patients with cerebrovascular accidents with PFOs fit the criteria for a definite paradoxical brain embolism.[12] PFOs are, therefore, more likely to be associated with stroke in younger patients.[6,7,14-17] In addition, given the high rate of PFOs in the general population, it is possible that the atrial communication is an "innocent bystander".

This is particularly applicable to patients > 55 years of age and those with known risk factors for stroke (i.e., hypertension, hypercholesterolemia, and smoking).[18,19] Additionally, PFO morphology (size and degree of shunting), hypercoagulopathies, and coexisting arterial sepal aneurysms may also affect this association.[20-22] Emboli from the heart are distributed in nearly 20% of ischemic strokes.[23,24] In old patients, nonvascular arterial fibrillation is the most important embolic source of stroke.[25,26] Emboli originating from aortic atherosclerotic lesions may also play an important role in the development of stroke.[23]

In the present case, in order to find the cardioembolic source of the brain emboli, we performed TEE for the patient. Initially, the large PFO seemed to be of a cardioembolic source. Since PFOs are detected in 10%–15% of the population by TEE, we continued looking for another source of emboli. Finally, the detection of an aortic aneurysm and the clot within it by TEE led us to the actual source of the emboli. CT angiography also established the diagnosis. Due to the past history of CABG (6 months earlier), it seemed that this aneurysm was formed after cardiac surgery**.** Furthermore, the site of this aneurysm was in accordance with the site of the cannulation of the ascending aorta when the patient was connected to the cardiopulmonary pump during the surgery. In addition, if this aneurysm had presented before surgery, angiography and aortogram would have revealed this pathology and the cardiac surgeon would have been able to detect this during the operation.

In conclusion, it should be kept in mind that a PFO in a patient with stroke may be an incidental finding and hence further examination seems to be the logical course of action in order to find any other sources of emboli, especially in elderly patients**.**

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