

Isolated large atrial septal aneurysm and multiple cerebral infarcts: Is there any association?

G Gavrielatos, K Letsas, L Pappas, I Alexanian, M Efremidis, H Sioras, F Kardaras

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Abstract

An atrial septal aneurysm (ASA) is a thin, localized saccular deformity of the atrial septum that bulges into the right or left atrium. Diagnosis can be established using transthoracic (TTE) and transesophageal (TEE) echocardiography. Although these abnormalities are considered clinically benign entities, they have been independently associated with ischemic stroke. The case of a large atrial septal aneurysm in a patient with transient ischemic attack and previous undiagnosed cerebral infarcts is described in the present report.

CASE PRESENTATION

A 60 year old woman with no medical history presented in the emergency department for presyncope evaluation. She reported generalised weakness, dizziness, sudden occurrence of vision disturbances, and transient dysphasia. Neurological signs and symptoms gradually improved over a period of 20 minutes. Consciousness was never impaired and there were no other symptoms.

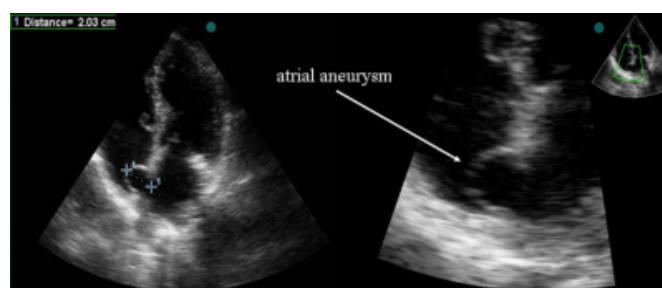
Physical examination following the episode revealed body temperature 36,6° C, a mid systolic murmur, best heard at the apex. No evidence of arterial hypertension or arrhythmias was found. She did not exhibit any postural changes of arterial blood pressure or pulse. Electrocardiogram showed sinus rhythm with LBBB pattern. Chest X-ray was normal. Laboratory findings including CK-MB and cardiac troponin T, as well as coagulopathy screening presented no abnormalities.

Carotid ultrasound Doppler examination, electroencephalography and routine 48-hour ambulatory (Holter) ECG were normal. Brain computer tomography (CT) scan showed old ischemic infarcts at the cerebral stem and basal ganglia, as well as microinfarcts of the cerebral cortex, located at the right temporal lobe. Doppler echocardiography of the lower extremities excluded deep vein thrombosis. A clinical diagnosis of transient ischemic attack was established.

Transthoracic echocardiography revealed a large atrial septal aneurysm (Fig. 1).

Figure 1

Figure 1: Four chamber view on TTE study revealed a large atrial septal aneurysm bulging into right atrium during cardiac systole.



According to Hanley's diagnostic criteria, atrial septum considered to be aneurysmal, when a dilated segment protruded at least 15 mm beyond the level surface of the atrial septum₁. An echo contrast study was also performed in order to exclude patent foramen ovale. The images recorded were not diagnostic of right to left shunting. A transesophageal echocardiography was recommended but the patient denied further evaluation, when she was informed with details the examination procedure.

The patient was started on anticoagulation treatment with warfarin. She improved and was discharged from hospital a week after admission with no neurological deficit.

DISCUSSION

Atrial septal aneurysm is a congenital malformation of the septum primum layer of the interatrial septum, but differences between interatrial pressure forces have also been reported as a cause of its development₂. The widespread availability of TTE and TEE has identified atrial septal aneurysm with increasing frequency. The rate of ASA recognition with echocardiography varies from 0.22% in repeated transthoracic studies to much higher rates by TEE, which allows almost ultimate imaging of the interatrial septum although a gold standard is absent in the detection of ASA_{1,3}. The frequency of this anomaly in the general adult population is low (2.2%) and similar to a 1% proportion described in a large autopsy series₄.

Several studies suggest a possible relationship between ASA and cerebral ischemia_{3, 4, 5}. The mechanism of stroke in patients with ASA remains poorly understood₇. Cerebral embolism might result from paradoxical embolism of venous thrombi across a right to left shunt, passage of a thrombus created on the left atrial side of the aneurysm or within the left atrium as a consequence of the association of atrial septal aneurysm with atrial fibrillation_{8,9}.

In our case brain CT-scan demonstrated multiple old ischemic infarcts that possibly reflect cryptogenic stroke in a patient with isolated ASA. Thrombus formation on the left atrial side of the aneurysm appears to be the most possible underlying mechanism. Bulging of the aneurysm that extends the portion between the primum and secundum atrial septum and thus, leading to a minor interatrial communication that allows paradoxical embolism seems to be a less plausible explanation. However, other causes of ischemic stroke at this age could not be excluded.

Aspirin and warfarin constitute the mainstay of therapy in cases of isolated ASA and ischemic cerebrovascular events. However, the treatment of choice still remains controversial. No significant superiority of warfarin over aspirin has been demonstrated in patients after first ischemic stroke. Oral

anticoagulation seems to be the preferred medical therapy in high-risk patients or those with multiple strokes on aspirin₉.

The presence of ASA may be a possible risk factor for ischemic stroke. Further prospective studies must be conducted in order to elucidate the mechanism between such cardiac abnormalities and ischemic cerebral disease.

CORRESPONDENCE TO

Gavrielatos Gerasimos, M.D. Evangelismos General Hospital of Athens, Second Department of Cardiology 45-47 Ipsilantou Street, 10676, Athens, Greece Phone: 00302102921140, Fax: 00302107217687 e-mail: makisgab@yahoo.gr

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Author Information

Gerasimos Gavrielatos

2nd Department of Cardiology, 'Evangelismos' General Hospital

Konstantinos P. Letsas

2nd Department of Cardiology, 'Evangelismos' General Hospital

Loukas K. Pappas

2nd Department of Cardiology, 'Evangelismos' General Hospital

Ioannis Alexanian

2nd Department of Cardiology, 'Evangelismos' General Hospital

Michalis Efremidis

2nd Department of Cardiology, 'Evangelismos' General Hospital

Hlias Sioras

2nd Department of Cardiology, 'Evangelismos' General Hospital

Fotios Kardaras

2nd Department of Cardiology, 'Evangelismos' General Hospital