Atrial septal aneurysm and stroke

Alok Tyagi, *MRCP, DM,*
Hani T. Benamer, *MRCP, PhD.*

Cardioembolic events probably account for 20% of all ischemic strokes. Cardiovascular embolism has also been recognized as a cause of otherwise unexplained stroke or transient ischemic attack in a significant number of patients. An association between atrial septal aneurysm (ASA) and focal cerebro-vascular disease has been suggested. However, the role of ASA as a risk factor for cerebral ischemia is not clearly defined. We report a young woman who had 2 episodes of neurological deficit in the presence of an ASA.

A 30-year-old woman presented to casualty with a 5-day history of sudden onset weakness over the right side of her face associated with difficulty speaking for the same duration. She had noted right fronto-parietal aching headaches for a few days before the onset of her neurological symptoms. She had a baby more than a year ago and had restarted the contraceptive pill 6 weeks previously. She had no history of any similar episodes and had no suggestion of a thrombotic tendency in herself or her family members. She did not smoke and had no history of illicit substance usage. On examination, she had a right-sided hemiparesis with the upper limb affected to a greater degree. She felt her speech had improved to a great degree but on examination she did have literal paraphasias and was not able to name parts of objects. She had no carotid bruit and her cardiovascular system examination was normal. A provisional diagnosis of a left middle cerebral artery infarct was made. Her routine blood investigations were all normal including a coagulation profile and autoantibody screen. Her prothrombotic screen was normal as were the serum homocysteine levels. Her chest x-ray and routine electrocardiogram were normal as well. A magnetic resonance image of brain revealed a left frontal infarct. She also had a cerebrospinal fluid study carried out, which was normal, and no oligoclonal bends were detected. She proceeded to have a trans-thoracic echocardiogram (echo) and a carotid ultrasound which were both normal. At this stage, she was discharged home on Aspirin with a diagnosis of a left middle cerebral artery infarct the cause of which was not clear. She was readmitted 3 months later with similar symptoms of speech difficulties and a headache, which resolved spontaneously within a couple of hours. A repeat computed tomography scan of the brain did not reveal any new abnormality. A 4 vessel cerebral angiogram was carried out which was entirely normal. A trans-esophageal echo was also requested as an outpatient. This showed a clearly defined ASA but with no evidence of a patent foramen ovale (PFO) (**Figure 1**). She was commenced on warfarin and has been asymptomatic since then.

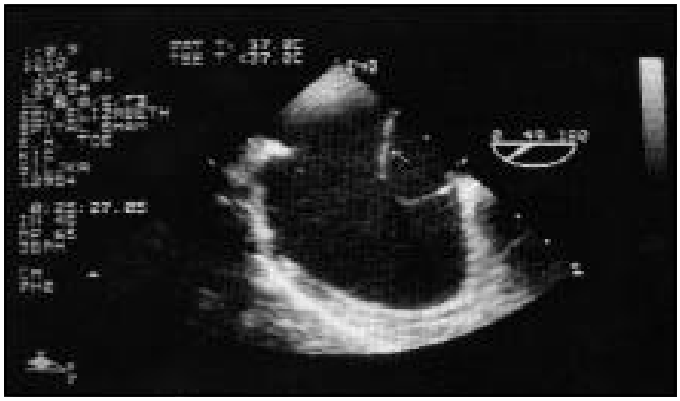

Figure 1 - Trans-esophageal echocardiogram demonstrating the atrial septal aneurysm (arrow) bulging into the right atrium.

The cerebral embolism task force defines cardio-embolic stroke as the presence of a potential source of embolism in the absence of other diseases of the intra- and extra-cranial vessels in patients with a non-lacunar infarct.[1] The normal cerebral angiogram in this case renders the ASA as the likely cause for the stroke. Cardiac sources of embolism can be divided into certain sources, namely, atrial or ventricular thrombosis, thrombosis on heart valve prosthesis, cardiac tumors or endocarditis, and potential sources namely, PFO, valve strands, ASA, dystrophy and calcification of the mitral valve, atheromatous plaque of the aorta or spontaneous echocontrast. These potential sources of embolism can be easily missed by trans-thoracic echo and therefore patients who do not have an obvious cause for a stroke should be investigated further with a trans-esophageal echo (as demonstrated in our case). In a study of 73 patients with unexplained stroke who underwent both trans-thoracic and trans-esophageal echo, the latter technique detected 70 sources of embolism as compared to only 14 identified by trans-thoracic echo.[2]

An ASA consists of redundant atrial septal tissue bulging into the right or the left atrium, or both. The accepted definition of ASA includes a basal width of more than 15 mm and an excursion of at least 15 mm of the aneurysm beyond the plane of the residual atrial septum.[3] Atrial septal aneurysm has been found in approximately 1% of consecutive autopsies and in up to 4.9% of patients undergoing trans-esophageal echo for reasons other than a search for sources of emboli. Between 4% and 27.7% of pre-selected patient populations with otherwise unexplained cerebral ischemic events have been found to have ASAs.[3] Despite the significant difference in the frequency of ASA detection, all studies demonstrated the higher prevalence of ASA in patients with a cerebral event than in controls. Results from a meta-analysis of all case-control studies of inter-atrial abnormalities associated with stroke suggested that ASA are strongly associated

with ischemic and cryptogenic stroke in patients less than 55 years of age.[4]

The cause of stroke in our patient was an isolated ASA, in the absence of a PFO. There is a strong association between PFO and ASA and 97% of ASA patients under the age of 45 also have a PFO.[5] Right to left shunting through a PFO, thrombus formation in the ASA, associated mitral valve prolapse and supraventricular arrhythmias are the potential sources of cardioembolism associated with ASA.[2] The optimal therapeutic regimen for secondary and possible primary prevention of stroke in subjects with ASA remains to be determined. It has been suggested that larger PFO's and ASA are more strongly associated with cryptogenic stroke than smaller abnormalities and are more likely to lead to recurrences.[4] Therapy with antiplatelet agents or anticoagulants for patients with ASA is controversial. Anticoagulation may be a reasonable choice for patients with both a PFO and an ASA where no other probable cause of the stroke can be identified. We elected to anticoagulate our patient due to the recurrence of neurological symptoms despite anti-platelet therapy. Surgical treatment has been recommended in patients, with both PFO and ASA, in whom maximal medical therapy has proven ineffective.

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From the Department of Neurology, New Cross Hospital, Wolverhampton, United Kingdom. Address correspondence and reprint request to: Dr. Alok Tyagi, Department of Clinical Neurophysiology, Royal Infirmary, North Staffordshire Hospital, Stoke-On-Trent, United Kingdom, ST4 7LN. Tel. +44 (1782) 554155. Fax. +44 (1782) 555315. E-mail: alokrajan@yahoo.co.uk

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