Paradoxical embolism with a patent foramen ovale and atrial septal aneurysm

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Abstract
Patent foramen ovale (PFO) and atrial septal aneurysm have been cited as potential risk factors for cryptogenic stroke. We present two cases which we propose to directly illustrate paradoxical embolisation as a mechanism of cerebrovascular accident. The diagnosis of PFO is discussed and the literature reviewed.

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Patent foramen ovale and atrial septal aneurysm have been cited as potential risk factors for cryptogenic stroke. We present two cases which we propose to directly illustrate paradoxical embolisation as a mechanism of cerebrovascular accident.

Case 1
A 55-year-old gentleman presented with sudden onset of expressive aphasia. The only other abnormality was atrial fibrillation. A transthoracic echocardiogram demonstrated echogenic masses within the right atrial and left ventricular cavities. Urgent transoesophageal echocardiography demonstrated mobile echogenic serpiginous masses within the right and left atria. These masses were connected to each other via a patent foramen ovale (Fig. 1). On the left side, this was seen to extend through the mitral valve into the left ventricle (Fig. 2). The appearance was consistent with thrombus. Studies of the inter-atrial septum revealed a large aneurysm with a marked excursion of the redundant atrial tissue (Figs. 3 and 4 which are sequential frames from the same cardiac cycle). The left atrial appendage was clear of thrombus.

Emergency cardiac surgery was performed whereupon a 30 cm thrombus was removed which had formed a true knot around itself within the left atrium. The atrial septal aneurysm was excised and the foramen ovale closed. Immediately after

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surgery, he developed a dense left hemiparesis. Further rehabilitation and recovery has been slow.

Case 2

A 47-year-old woman presented to a local district general hospital with sudden onset of weakness and loss of sensation in her left arm. This followed a 6-month history of progressive breathlessness on exertion. There were no other associated cardiovascular or neurological symptoms. Her brother had recently been diagnosed with myotonic dystrophy.

On examination she was hypoxic with oxygen saturations of 88% on air. Auscultation of the chest revealed signs consistent with a left pleural effusion. Left brachial and radial pulses were reduced. She was noted to have bilateral ptosis, with weakness and paraesthesia affecting the left upper limb. Her ECG showed sinus rhythm, left axis deviation and poor R wave progression, intraventricular conduction delay with repolarisation abnormalities. CXR confirmed a left sided pleural effusion. Shortly after admission she deteriorated, requiring non-invasive ventilatory support. The thrombus within the left atrium prolapsed via the mitral valve (Figure 2).
support on the high dependency unit. A trans-thoracic echocardiogram revealed a thin mobile echogenic mass in the right atrium. A provisional diagnosis of atrial myoxoma was made and she was transferred to our centre for further evaluation.

Transoesophageal echocardiography showed a long serpiginous mass in both the left and right atria consistent with thrombus (Fig. 5). This appeared to be fixed to the atrial septum in the region of the foramen ovale. Within the left side, the distal end was freely mobile and prolapsed through the mitral valve (Fig. 6). The left atrial appendage was normal with no thrombus seen. Left ventricular function was moderately impaired. Within the right heart, no continuation into the right ventricular cavity was seen. The right ventricle, however, was slightly dilated with an estimated pulmonary artery pressure of 40 mmHg.

A diagnosis of saddle embolus through a patent foramen ovale was made. The left arm neurological findings were thought to be secondary to paradoxical systemic embolisation from extension of the thrombus into the left atrium. The general appearance was confirmed as secondary to dystrophia myotonica. We postulated that the aetiology of her condition stemmed initially from a venous thrombosis which resulted in right atrial and pulmonary thromboembolism. This resulted in a secondary elevation in right heart pressures, reversal of pressure differences between the atria and thus opening of the PFO. Systemic neurological sequelae could then occur.

The patient underwent a successful emergency removal of atrial saddle embolus with closure of the PFO and insertion of an IVC Greenfield filter. A post-operative thoracic CT confirmed appearances consistent with multiple pulmonary emboli. She has been anticoagulated with warfarin.

**Discussion**

A patent foramen ovale is a persistent valvular-like connection between the left and right atrium. Normally, this closes by fibrous adhesions between the septum primum and secundum during the first months of life. However, if this connection persists into adult life, intermittent right to left shunting may occur. Autopsy studies have demonstrated a prevalence of patent foramen ovale in the population in the range of 17–35%.1

An atrial septal aneurysm consists of redundant atrial septal tissue bulging into the right or left atrium. There is variation in the anatomical definition, but in general, the basal width of an atrial septal aneurysm should be more than 15 mm and the excursion of the aneurysm beyond the plane of the residual atrial septum should be at least 10 or 15 mm.2

Patent foramen ovale and atrial septal aneurysm have been identified as potential risk factors for stroke. In subjects less than 55 years of age, a lower prevalence of large vessel atherosclerosis or small artery disease makes a diagnosis of cryptogenic stroke more frequent than older subjects. Association of cryptogenic stroke and patent foramen ovale has been reported in this age range.
More recently atrial septal aneurysm has also been described as the only potential source of embolism in a significant proportion of subjects who had sustained a transient ischaemic attack.\textsuperscript{4}

Potential mechanisms of stroke in patients with atrial septal abnormalities include paradoxical embolus from a venous source, direct embolisation from thrombi formed within an atrial septal aneurysm and the formation of thrombus as a result of atrial arrhythmias.

Recent studies have demonstrated that patients who have sustained cryptogenic stroke, and have a patent foramen ovale and atrial septal aneurysm constitute a subgroup who are at high risk of recurrent stroke.\textsuperscript{5} It remains unclear as to whether such patients are best managed with combination antiplatelet therapies, anticoagulation or closure of foramen ovale.

The current literature supports an association between cryptogenic stroke and patent foramen ovale or abnormal atrial anatomy. Although in the presented cases the initial precipitant of the thrombus remains unknown, we propose that the images presented illustrate a clear relationship of these cardiac abnormalities in the aetiology of cerebrovascular accident.

References