Thrombus in transit in a patient with ischaemic stroke

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ABSTRACT
Stroke or cerebrovascular accident is common and is most commonly embolic or haemorrhagic. A common source of embolism is the heart and as such echocardiogram is an essential investigation. However, clinicians need to be aware of rare sources of embolism. We report a rare and interesting case of a 61-year-old Caucasian man who presented with stroke and on evaluation was found to have a large thrombus coiled striding over a patent foramen ovale into the right and left atrium consistent with a ‘thrombus in transit’. The origin of the thrombus was later confirmed to be from the left saphenous vein. This case highlights an interesting case of ‘thrombus in transit’.

Keywords: Cerebrovascular accident, myxoma, patent foramen ovale, paradoxical embolism

INTRODUCTION
Stroke or cerebrovascular accident is common and an important cause of morbidity. A proportion of stroke is due to ischaemia as a result of embolism or thrombosis. The risk for embolic stroke is classified as either high or low. The risk is categorised as high in the presence of atrial fibrillation, rheumatic heart disease, artificial heart valves, known intra-cardiac thrombus, atrial flutter, recent myocardial infarction, congestive heart failure with reduced left ventricular function, non-infective and infective endocarditis, cardiac tumours such as papillary fibroelastoma and left atrial myxoma. The low risk category includes mitral annular calcification, patent foramen ovale (PFO), atrial septal aneurysm, left ventricular aneurysm without thrombus, isolated left atrial spontaneous ‘smoke’ on echocardiogram and complex atheroma in the ascending aorta or proximal arch. Many of these risk factors can be identified by echocardiogram. Hence echocardiogram is an essential investigation for ischaemic strokes. Infrequently, a rare pathology is detected. We report a rare and interesting case of stroke who on evaluation was later found to have a ‘thrombus in transit’ entangled in PFO.
CASE REPORT

A 61-year-old Caucasian man, otherwise healthy, presented in the early hours with confusion and aphasia. His wife had noticed him confused with unsteady movements after returning from toilet at 4.00 AM. He was unable to talk and had difficulty dressing. He did have some shortness of breath of one week duration. His past medical history was unremarkable, mother had stroke at the age of 80 and he was not taking any medications. He did not smoke.

On examination, he was conscious and was haemodynamically stable with a blood pressure of 130/89 mmHg, heart rate of 75/min regular. He had aphasia with facial asymmetry with reduced power on right side with brisk reflexes and up-going planter reflex. The rest of the clinical examination was unremarkable. Investigations revealed normal blood counts, erythrocyte sedimentation rate (ESR) and blood sugar. Clotting, liver and renal profiles were within normal limits. Other investigations including cardiac enzymes, syphilis serology (Venereal Disease Reference Laboratory VDRL, Tropanema Palladium Haema-Agglutinin TPHA), anti-nuclear antibody (ANA), and anti-double stranded DNA (Anti-dDNA), serum homocysteine, protein C and S, and anti-thrombin III levels were normal. Electrocardiography showed sinus rhythm and non-specific ST-T segment changes in the anterior leads. Computed tomography scan of brain showed ischaemic stroke in the left middle cerebral artery territory (Figure 1).

Over the next few days, the neurological deficits improved and he was able to mobilise independently and converse normally. A carotid Doppler study showed calcified atherosclerotic plaques in the left carotid bifurcation with minimal turbulence but no restriction of flow in the carotids or vertebral arteries. Interestingly, a routine transthoracic ec-hocardiogram (TTE) showed mobile masses in both left atrium (LA) and right atrium (RA) and appeared to be attached to the inter-atrial septum (IAS). A trans-oesophageal echocardiogram (TOE) showed the large mobile masses in both atria, more in the RA and attached to IAS (Figure 2). The RA mass also moved across the tricuspid valve (TV) into the right ventricle (RV). The echogenicity, lobular pattern and the mobility of the mass were not specific for either a myxoma or thrombus.

The patient was referred for surgical removal of the mass. Pre-surgery diagnostic coronary angiogram showed anomalous left circumflex artery with 70% stenosis. The patient proceeded to an open heart surgery for removal of the mass and coronary artery bypass graft of the left circumflex artery. Harvesting of the left saphenous vein was performed and interestingly, it was found to be thrombosed. The right saphenous vein was
harvested then instead. When the RA was opened, a long clot-like structure was found coiled up in the right atrium (Figure 3a), crossing to left atrium through PFO that measured one centimetre in diameter (Figure 3b). This was removed and the PFO closed with a patch. The clot measured 18 centimetres in length. Histopathological examination of the atrial and saphenous vein specimens confirmed them to be clots.

The patient had an uneventful recovery and an inferior vena cava filter was inserted to prevent further migration of clots. He was discharged on medical treatment for coronary artery disease and anticoagulation. He was well when he was last seen one year after surgery.

**DISCUSSION**

Our case highlighted a rare and interesting case of ‘thrombus in transit’ in a patient who presented with an ischaemic stroke, probably from a paradoxical embolus. Another interesting aspect was that our patient did not have any risk factors or symptoms of deep vein thrombosis.

PFO has long been described as a cause of paradoxical embolism and a potential cause of significant pathology. ²⁻⁴ Paradoxical embolism occurs when an embolus originating from the systemic venous system such as a deep vein thrombosis crosses over to the systemic arterial circulation manifesting as embolic disease. The thrombus typically passes through a PFO but can also pass through septal defects and pulmonary arterio-venous

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**Fig. 2:** A trans-oesophageal echocardiogram showing a lobulated mass in the right (Large arrow) and left atria (Small arrow): RA (right atrium), LA (Left atrium) and LV (Left ventricle).

**Fig. 3:** Intra-operative pictures showing: a) a long clot being extracted from the right atrium and b) a patent foramen ovale (PFO) seen; c) Extracted clot with a large head and a long tail.
malformations. This usually occurs in the setting of increased pressure on the right side resulting in right to left shunting. However, a transient increase in pressure during coughing or valsalva maneuver can also lead to paradoxical embolism.

The diagnosis of paradoxical embolism is usually presumptive in the setting of embolic disease in the presence of a PFO and absence of source of thrombus in the left heart or arterial system. Venous thromboembolic disease usually manifests as pulmonary embolism. Even in the presence of a PFO, the initial manifestation may be with pulmonary embolism. In cases of ‘thrombus in transit’ reported in the literature, some of the patients initially presented with pulmonary embolism. In our case, our patient did not have any history of dyspnoea or chest pain and investigations did not show any evidence of pulmonary embolism.

Occasionally, a thrombus can get entangled in a PFO and as in our case it mimic bilateral atrial mass especially if the PFO is not clearly discernible. It may then be difficult to differentiate from bilateral myxoma. Previous studies have reported such cases but there are no specific clinical or echocardiographic features to differentiate between these two conditions. In our case, the initial suspicion included bilateral atrial myxomas and the diagnosis of ‘thrombus in transit’ was only made during surgery when the long clot was extracted and a PFO was found. In our case, one end of the clot was very big and probably had contributed to the difficulty in seeing the PFO even on TOE. On echocardiography, ‘thrombus in transit’ has often been described as ‘worm-like structures’ seen in both atria and often the PFO is visible.

In cases of thromboembolic stroke, echocardiography is an important investigation to assess for cardiac causes or sources. Although TTE remains the cornerstone of non-invasive imaging, TOE has been shown to be superior for identification of cardiac sources of emboli and is more cost effective. Proximity of the TOE probe to the posterior of the heart and lack of intervening lung and bone tissues results in better spatial resolution and improved detection rate of intracardiac thrombi and tumour, PFO, vegetations, atheromatous plaques in aorta and spontaneous contrast (‘smoke’) in left atrium, a marker of blood stasis. However, even TOE has limitations. In our case, although it clearly defined the mass and showed that it was attached to the septum, it was not possible to differentiate the exact nature of the mass, either a thrombus or a myxoma, in this case a bilateral myxomas. Furthermore, as the thrombus was quite large, the PFO was not visible on both echocardiography.

In conclusion, ‘thrombus in transit’ is rare and to our knowledge, this is the first case to be reported from Brunei Darussalam. Our case highlights to clinicians a rare and interesting case and the need to be aware of paradoxical embolism and ‘thrombus in transit’ in patient where echocardiography shows tumour mass in both the atria.

REFERENCES
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