

Nephroquiz
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Acute renal failure and multiple sites of ischaemia: what is the unifying diagnosis?

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A 67-year-old man presented to the Accident and Emergency department in September 2004 with cough and sudden shortness of breath. In 2002, he had suffered a left occipital infarct that presented as a transient ischaemic attack. In 2003, he sustained bilateral cerebellar infarcts that left him with residual tremor and ataxia (Figure 1). He took regular aspirin, clopidogrel and simvastatin for secondary prevention of cerebrovascular events.

On presentation his oxygen saturation was 79%, heart rate 120/min, respiratory rate 40/min and blood pressure 155/112 mm Hg. Chest auscultation revealed crackles and decreased air entry at the left base. Examination of the heart and abdomen was normal. He had type 1 respiratory failure (pO_2 6.1 kPa, pCO_2 4.5 kPa). Full blood count, creatinine (75 $\mu\text{mol/l}$), electrolytes and clotting were normal. A chest radiograph showed left basal shadowing and prominent hila. He was treated with heparin and aspirin for suspected pulmonary embolism; CT pulmonary angiography was performed (Figure 2).

Two days later he developed nausea, vomiting and a sudden sharp constant pain in the left flank passing into the iliac fossa, not relieved by morphine. Abdominally, he was tender in the left hypochondrium and iliac fossa. There were no urinary symptoms. Concurrently, there was an abrupt rise of urea to 16.6 mmol/l and creatinine to 450 $\mu\text{mol/l}$. He underwent abdominal CT scan and echocardiography (Figures 3, 4).

Question

What is the unifying diagnosis?

Answers to the quiz

- Figure 1: Old right cerebellar infarct shown as a region of hypointensity and new left cerebellar infarct shown as a region of hyperintensity.
- Figure 2: Multiple large pulmonary emboli.
- Figure 3: Complete infarct of left kidney and wedge infarct of right kidney.
- Figure 4: Transthoracic contrast echocardiogram demonstrating atrial septal aneurysm and patent foramen ovale.

Clinical diagnosis

Paradoxical embolism, i.e. systemic embolism via patent foramen ovale (PFO).

Comment

The foramen ovale is patent in 27–35% of adults [1]. Such patency presents an opportunity for systemic venous thrombi to find their way into the systemic arterial circulation, and cause embolic phenomena including stroke, myocardial infarction and peripheral arterial occlusion. Therefore, in any patient presenting with an unexplained thromboembolic event, it is important to consider PFO.

PFO can be managed in one of two ways: medical treatment (antiplatelet or anticoagulation) or percutaneous catheter closure. Open surgical closure is now rarely, if ever, necessary.

A number of studies, in patients with thromboembolic events complicating PFO, have shown the benefits of anticoagulation. By using aspirin and/or warfarin, the stroke recurrence rate can be as low as 1.9% (95% CI; 1.48–2.60) [2]; 5-year survival rates are >90% [3].

In practice, percutaneous closure by catheter is now the standard management in adults whose PFO has produced symptoms [4]. The procedure, however, is not without complications—air embolism, perforation and pericardial

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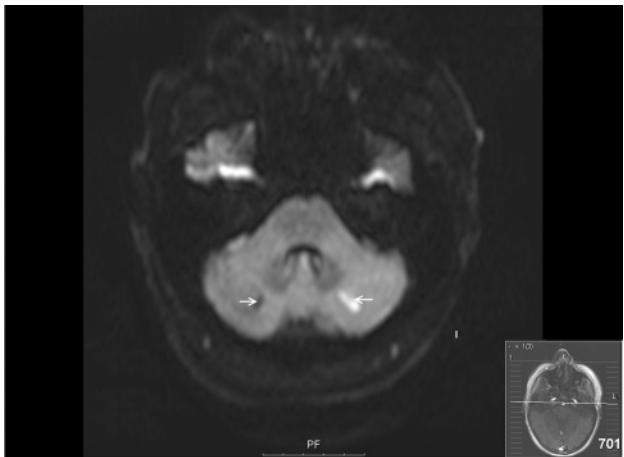


Fig. 1. Diffusion weighted MRI of head, 2003.



Fig. 2. CT pulmonary angiogram.



Fig. 3. CT of abdomen.

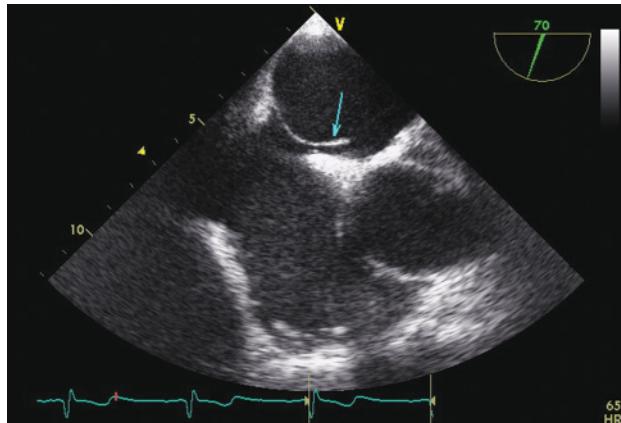


Fig. 4. Transthoracic contrast echocardiogram.

effusion, residual shunt and failure/embolisation of the device itself. There is evidence that in experienced hands complications are now very few [5].

In the patient described above, closure [deployment of a 25 mm right atrial disc sized Amplatz (GA Medical Institute, GA, USA) PFO occluder] was carried out in November 2005 under general anaesthesia using transoesophageal echocardiographic guidance. Heparin and antibiotics were given. After the procedure, echocardiography was most satisfactory and showed no residual shunt. Alternatively, the procedure could have been carried out under local anaesthetic using intracardiac echocardiography or even fluoroscopy alone.

When last seen in February 2008, the patient was well with stable creatinine ($143 \mu\text{mol/l}$).

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Conflict of interest statement. None declared.

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