Pulmonary embolism and patent foramen ovale causing an ischaemic stroke

Introduction

This article reports a case of a patient presenting with an ischaemic stroke, secondary to pulmonary embolism and paradoxical embolization via a patent foramen ovale. This case highlights the need to screen such patients for a patent foramen ovale in the presence of a large pulmonary embolism.

Figure 1. Computed tomography of the head showing early signs of left middle cerebral artery infarction with loss of grey white matter differentiation, loss of lentiform nucleus and insula ribbon (arrow).

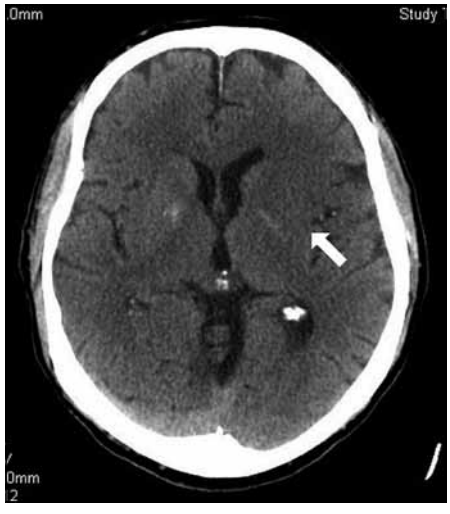
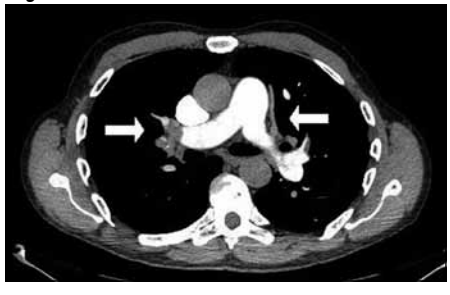


Figure 2. Computed tomography pulmonary angiogram showing bilateral pulmonary emboli in the right main pulmonary artery and the left lingular branches (arrow).



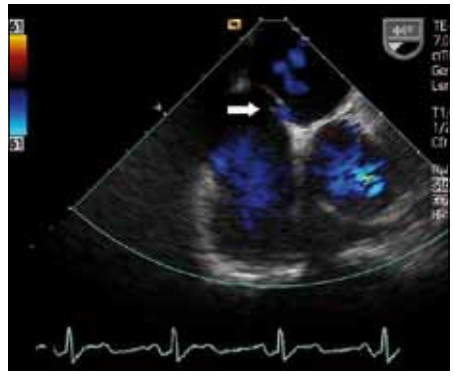
Dr Gavin Lewis *is Core Medical Trainee Year 1 and* **Dr Patrick McDonald** *is Consultant Physician, Southport and Ormskirk Hospital NHS Trust, Kew PR8 6PN*

Correspondence to: Dr G Lewis (gavinlewis85@hotmail.com)

Discussion

Patent foramen ovale occurs in 25–30% of the population and its role in the pathogenesis of ischaemic stroke by paradoxical embolism is controversial (Homma et al, 2002). In the presence of pulmonary embolism and secondary pulmonary artery hypertension, the presence of a patent foramen ovale increases the risk of ischaemic stroke from 2.2% to 13% (Konstantinides et al, 1998). This case highlights the need to screen for patent foramen ovale in the

Figure 3. Transoesophageal echocardiogram – colour Doppler showing right to left shunt across the atrial septum (arrow).



Case Report

A 66-year-old male non-smoker with no past medical history presented to hospital after waking up with speech loss and right-sided weakness. He was in sinus rhythm 75/min, blood pressure 133/86 mmHg, respiratory rate 18/min and oxygen saturation was 92% on air. Examination showed no abnormal cardiovascular or respiratory signs. Neurological examination revealed right-sided upper and lower limb weakness, global aphasia and right-sided inattention. He was diagnosed with an acute stroke and treated with high-dose aspirin. Thrombolysis could not be given, as there was no specific time of symptom onset.

Of his initial investigations, the electrocardiogram showed sinus rhythm, QRS axis 90°, ST depression in V2–V5 and non-specific T-wave inversion in V1–V6 (changes non-dynamic on repeated tracing). His haemoglobin was 16.5 g/dl, and haematocrit was 0.52 (52%). Arterial blood gases were pH 7.37, pCO_2 4.9 kPa, pO_2 8.1 kPa, HCO_3 28.6 mmol/litre. His troponin T was 24 ng/litre on admission and 18 ng/litre after 12 hours. A chest X-ray showed mild pulmonary venous congestion, computed tomography of the head showed acute left middle cerebral artery infarction (*Figure 1*), and there was no carotid stenosis on Doppler ultrasound scan of the carotid arteries.

The patient was transferred to the acute stroke unit. Review of his abnormal electrocardiogram prompted further cardiac investigation with an echocardiogram, which showed a dilated right atrium and ventricle with severely impaired systolic function, elevated pulmonary artery pressure estimated at 98–103 mmHg and mild left ventricular systolic impairment.

Because of the pulmonary artery hypertension and persistent hypoxia the patient had a computed tomography pulmonary angiogram that demonstrated bilateral pulmonary emboli (*Figure 2*). He had no clinical signs of deep vein thrombosis. Thromboembolism occurring in both the right and left circulations suggested paradoxical embolism or thrombophilia. Therefore the patient underwent bubble echocardiogram with bubbles in the left ventricle post Valsalva manoeuvre confirming right-to-left shunting and transoesophageal echocardiogram which showed a moderate-sized patent foramen ovale (*Figure 3*). His JAK2 gene, erythropoetin level and thrombophilia screen were negative.

He was anticoagulated and received rehabilitation, being discharged 6 weeks post event. His presenting history did not reveal any specific actions that may have caused a transient right-to-left shunt from a Valsalva manoeuvre such as sneezing, but the presence of a pulmonary embolism leading to pulmonary artery hypertension increased the likelihood of a paradoxical embolism. Following discharge he underwent surgical closure of the patent foramen ovale.

presence of large pulmonary emboli and may influence subsequent management of the patent foramen ovale. BJHM

Homma S, Sacco RL, DiTullio MR et al for the PFO in Cryptogenic Stroke Study (PICSS) Investigators (2002) Effect of medical treatment in stroke patients with patent foramen ovale: Patent Foramen Ovale in Cryptogenic Stroke Study. *Circulation* **105**: 2625–31

Konstantinides S, Geibel A, Kasper W et al (1998) Patent foramen ovale is an important predictor of adverse outcome in patients with major pulmonary embolism. *Circulation* **97**: 1946–51

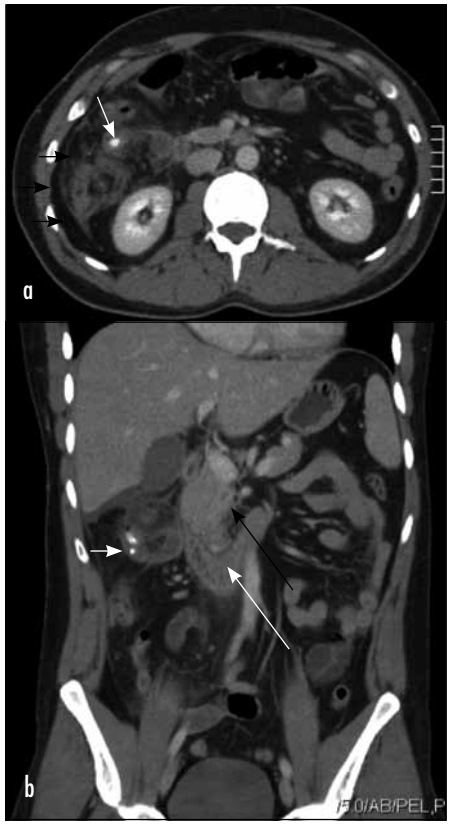
LEARNING POINTS

- Ischaemic stroke may be caused by pulmonary embolism in the presence of a patent foramen ovale.
- Screening for patent foramen ovale in patients with pulmonary embolism is not currently routine, but this may be necessary if there is associated pulmonary artery hypertension given the increased risk of stroke in these patients.

Subhepatic appendicitis presenting with right upper quadrant pain

Subhepatic appendicitis is a rare presentation and is often confused by clinicians in the emergency department. A 32-year-old man re-presented with right upper quadrant pain and a working diagnosis of cholecystitis just 2 days after being sent home with buscopan after suspected gastroenteritis. Diagnosis was confirmed only after computed tomography, which revealed a high riding appendix secondary to fetal malrotation (*Figure 1*).

Although appendicitis is largely a clinical diagnosis, this highlights the importance of maintaining a low threshold for performing an abdominal computed tomography scan and/or laparoscopy even in younger patients who deteriorate or present atypically. Laparoscopy in these cases of colonic malformation acts as a valuable adjunct to radiological investigations and helps to guide the surgeon's approach, minimizing the need for a large incision. Using these methods establishes an earlier diagnosis, thus shortening the hospital length of stay and further reducing morbidity and mortality outcomes. BJHM



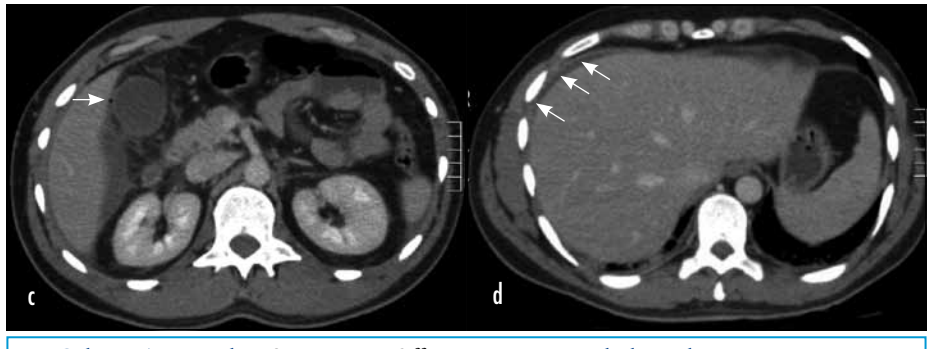


Figure 1. a. Contrast-enhanced computed tomography showing a thickened and distended appendix containing an appendicolith (arrow) and fat stranding and inflammation (black arrow). b. Coronal reconstruction showing a high appendix seen in the right upper quadrant (short white arrow) and the duodenum (long white arrow) and pancreas (black arrow). c. Free locule of air seen adjacent to the gall bladder suspicious of perforation (arrow). d. Small amount of free fluid seen in the right subphrenic space and pelvis.

Dr Othman Z Kirresh *is Senior House Officer,* **Mr Ioannis Nikolopoulos** *is Registrar in General Surgery,* **Miss Tayo Oke** *is Consultant in Colorectal Surgery and* **Miss Susan Koshy** *is Registrar in General Surgery in the Department of General Surgery, Queen Elizabeth Hospital, London SE18 4QH*

Correspondence to: Dr OZ Kirresh (othmankirresh@doctors.org.uk)