A fatal pulmonary embolus in a post-stroke patient

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A 68 year old male with a background of rheumatoid arthritis (on methotrexate), hypertension, type 2 diabetes mellitus and dyslipidaemia was repatriated to his local hospital 5 days following a left parieto-occipital infarct which was thrombolysed.

On repatriation he had mild expressive dysphasia and a dense right hemiplegia (0/5 power throughout), which improved to 1/5 during the first week of rehabilitation.

Initial investigations to determine underlying aetiology demonstrated normal sinus rhythm on his ECG (fig. 1) and complete occlusion of his left internal carotid artery on his carotid doppler.

He was commenced on intravenous fluids, intravenous antibiotics for hospital-acquired pneumonia, and loaded on digoxin for atrial fibrillation. The ECG (fig. 2) was interpreted as atrial flutter with variable AV block. He was afebrile and oxygen saturations as high as 96% on air. Chest X ray demonstrated right middle zone consolidation and patchy left basal consolidation (fig. 3). Blood tests demonstrated an acute kidney injury, transaminitis and markedly raised inflammatory markers.

Ten days after repatriation, he reported shortness of breath and a cough during the registrar ward-round. On examination he was found to have right mid-zone crepitations. The ECG (fig. 2) was interpreted as atrial flutter with variable AV block. He was afebrile and oxygen saturations were 96% on air. Chest X ray demonstrated right middle zone consolidation and patchy left basal consolidation (fig. 3). Blood tests demonstrated an acute kidney injury, transaminitis and markedly raised inflammatory markers.

CT pulmonary angiogram demonstrated large proximal bilateral pulmonary emboli and, following discussion with the consultant and review of post-thrombolysis CT head to ensure there was no evidence of post-thrombolysis haemorrhage, treatment was started with IV unfractionated heparin. However, one hour later he arrested and, despite thrombolysis and CPR for over an hour, successive blood gases demonstrated worsening respiratory and metabolic acidosis and CPR was stopped.

He was commenced on intravenous fluids, intravenous antibiotics for hospital-acquired pneumonia, and loaded on digoxin for atrial fibrillation thought to be secondary to this. His fluids were continued the next day when his acute kidney injury was mildly worse despite symptomatic improvement and a heart rate of 70 bpm.

The following day he deteriorated rapidly with decreasing oxygen saturations (89% on air down from 96% on air 3 hours previously). On examination he was tachypnoeic (28 respirations per minute), tachycardic (120 bpm) and peripherally cool with prolonged capillary refill time. Jugular venous pressure was elevated to the angle of the jaw with minimal bibasal crepitations and soft, non-tender calves with a trace of pedal oedema. The ECG (fig. 3) and chest X ray were repeated and arterial blood gas performed on 5 L of oxygen demonstrated:

- pH: 7.39
- pCO₂: 3.5
- pO₂: 23.4
- Lactate: 3.4
- Base excess: -7.3
- HCO₃: 15.7

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Given the metabolic acidosis, improvement in consolidation on the repeat chest X ray and right axis shift and S1Q3T3 sign on the ECG, the reviewing SHO considered the deterioration may be due to a pulmonary embolus and asked the registrar and HDU to review urgently.

Given the presence of ECG changes suggestive of PE at the time of treatment for pneumonia, it was thought that there may have been a PE present at this time alongside the pneumonia. It is unknown whether earlier treatment would have prevented mortality given the risk of intracranial haemorrhage with anticoagulation.

Pulmonary embolism is the leading cause of death two to four weeks after a stroke with an incidence of roughly 1% in the first 3 months following a stroke. This risk is higher in older and less mobile patients. Current NICE guidelines state that anticoagulation should not be used routinely post-stroke, but that it may be considered in certain subgroups such as those with complete paralysis of a leg.

Learning points
1. A PE can mimic pneumonia and the two can often co-exist in the high-risk patient. Both are common causes of death post-stroke.
2. PE can be associated with a number of ECG changes including new-onset AF, right axis shift, late R wave progression and RV strain. Direct comparison with previous ECGs can aid recognition.
3. Anticoagulation in the acute stroke patient is a challenging scenario with current NICE guidance recommending an individualised approach to prophylaxis and treatment.

References

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