Electrocardiographical case. Elderly woman with sudden onset of post-operative dyspnoea

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**CLINICAL PRESENTATION**

A 70-year-old Chinese woman was admitted for elective total hip replacement for a dysplastic right hip secondary to osteoarthritis. She was a hypertensive and had a past history of burr hole surgery for subdural haematoma. In the immediate post-operative period, the patient’s recovery was unremarkable. She was not started on deep vein thrombosis prophylaxis with heparin because of her past history of subdural haematoma. However, on the fourth post-operative day, she developed a sudden onset of shortness of breath, tachycardia and hypotension. Her blood gas levels were: pH 7.445, pO₂ 44.5 mmHg, pCO₂ 30.6 mmHg, base excess -2.1 mmol/L, standard bicarbonate 22.4 mmol/L, and oxygen saturation 83%. What does the electrocardiogram (ECG) show (Fig. 1)? What is your diagnosis?
ECG INTERPRETATION
The ECG (Fig. 1) shows sinus tachycardia, with ST segment elevation in lead V1 and ST depression in leads I, II and V5-6, along with a qR complex in V1. In addition, there is an S wave in lead I and a Q wave with T inversion in III. In an elderly patient developing breathlessness post-operatively, these findings suggest acute pulmonary embolism.

DIAGNOSIS
Acute pulmonary embolism.

CLINICAL COURSE
Spiral computed tomography (CT) of the thorax showed a large thrombus in the right main pulmonary artery that extended into the upper and lower lobe pulmonary arteries. Thrombus was also visualised in the segmental branches. A smaller thrombus was seen at the bifurcation of the left main pulmonary artery, with thrombus extension into the upper and lower lobe pulmonary arteries. There was no lung consolidation or collapse. Urgent bedside echocardiography showed a dilated right ventricle with impaired right-sided ejection fraction. There was hypokinesia on the right side with sparing of the right apical regions. The visually-estimated ejection fraction of the left ventricle was 60%. There were no regional wall motion abnormalities.

The patient was also screened for thrombophilia. Her protein C, protein S levels, antithrombin III and factor V (Leiden) levels were normal. Lupus anticoagulant and antiphospholipid antibodies were not demonstrable. Duplex Doppler ultrasonography of her lower limbs showed acute partial thrombosis in right popliteal (below the mid-knee), posterior tibial and peroneal veins. Right lower saphenous veins were also partially thrombosed.

In view of her recent surgery, she was not given thrombolytic therapy. She was started on intravenous heparin and anticoagulation with warfarin. An inferior vena cava filter was also inserted 1 cm below the renal vein ostia to prevent recurrent pulmonary embolism. Her subsequent recovery was uncomplicated and she was discharged 10 days later with an international normalised ratio (INR) target of 2.0-3.0.

DISCUSSION
The classical ECG features of acute pulmonary embolism are SIQ3T3. These were originally described by McGinn and White in 1935(1). However, these findings occur in only 12-30% of cases(2). Transient right bundle branch block occurs in 9% and this is due to the compression on the right bundle branch secondary to increased pressure within the right ventricle(3). The ECG findings of acute pulmonary embolism are varied and therefore cannot be used alone. A normal-looking ECG does not exclude the diagnosis.

Uncommon findings include right (7%) or left axis deviation (97%) and peaked P waves. Other common but non-specific features include ST and T wave changes such as T wave inversion in the anterior precordial leads as well as displacement of the transition zone to the left. Alternatively, the only abnormality may be sinus tachycardia (13%). Recently, QT prolongation (456-521ms) with global T wave inversion has been reported as a novel electrocardiograph phenomenon(4). Atrial arrhythmias, such as atrial extrasystoles, atrial flutter, atrial fibrillation and atrial tachycardia, may also co-exist.

The differential diagnosis to consider is that of acute inferior myocardial infarction in the presence of Q waves in lead III, coved ST elevation in lead III, and T wave inversion in lead III and aVF. Another great mimic is that of anteroseptal infarction. However, the Q wave in pulmonary embolism is always followed by an ensuing R wave forming part of the qR complex(5). Furthermore, the Q wave does not have the typical features of a pathological Q wave in that it is not 0.04 seconds or longer and the magnitude is not 25% more than the ensuing R wave.

On the other hand, the ST elevation in inferior myocardial infarction involves leads II, III and aVF, and is more marked in leads II than III. In acute pulmonary embolism, the ST segments are usually isoelectric in standard lead III and aVF, and may in fact be depressed in standard lead II. Concomitant acute anteroseptal injury or ischaemia is a common feature in acute pulmonary embolism but not in acute inferior myocardial infarction.

Pulmonary embolism is generally associated with hypoxemia, hypocapnia and raised oxygen alveolo-arterial difference. However, up to 20% of patients have normal partial oxygen pressure and alveolo-arterial difference. Plasma D dimer derived from the ELISA method in excess of 500 microgram/litre is more than 99% sensitive for acute pulmonary embolism and deep venous thrombosis. Though D dimer is specific for fibrin, the specificity of D dimer for venous thromboembolism is poor, particularly in the elderly and hospitalised patients. This together with its low positive predictive value, limits its usefulness in ruling in pulmonary embolism. Hence, it is most useful in emergency room patients.

Pulmonary angiography is widely regarded as the reference method for the diagnosis and exclusion of pulmonary embolism. However, the procedure is invasive and carries with it the attendant risk of an invasive procedure. More complications arise if there
is concomitant pulmonary hypertension. Insufficient opacification of the subsegmental pulmonary arteries may reduce the sensitivity as well as specificity of pulmonary embolism involving the subsegmental arteries, an area that has remained controversial.

Two other widely-used non-invasive imaging modalities include ventilation-perfusion (VQ) scan as well as the spiral CT angiography. A VQ scan showing a normal or low probability has a high negative predictive value when the clinical suspicion of pulmonary embolism is low, and a high-probability scan has a high positive predictive value when the clinical suspicion is high. Unfortunately, only 34% of cases correspond to these two categories(6). In some studies, the use of spiral CT angiography has demonstrated acute PE in 14% to 44% of patients with nondiagnostic VQ scans(7).

Treatment for acute pulmonary embolism is stratified according to whether it is a massive pulmonary embolism. In acute massive pulmonary embolism consisting of shock and/or hypotension with a blood pressure of less than 90 mmHg that is not due to new onset arrhythmia, hypovolemia or sepsis, thrombolytics is the treatment of choice. The aim is to relieve increase in the right ventricle afterload that is seen in massive pulmonary embolism. In non-massive forms of acute pulmonary embolism, anticoagulation should be started. Either unfractionated or fractionated heparin is used while waiting for the INR to reach therapeutic levels of 2.0-3.0. Higher INR is associated with higher risk of bleeding.

For first presentation of acute pulmonary embolism, the duration of anticoagulation should be six months. Long-term anticoagulation is also recommended in patients with recurrent pulmonary embolism. An inferior vena cava filter should also be inserted in patients who have contraindications to anticoagulation. Although it was widely recommended initially that the insertion of an inferior vena cava filter precludes the use of anticoagulation, adjunctive anticoagulation has now also been recommended to reduce vena cava occlusion as well as reduce the risk of thrombosis that may occur proximal to the site of insertion.

This case illustrates the typical clinical presentation of pulmonary embolism in the high-risk population of post-operative elderly patients. A high index of suspicion with a classical history is required. The diagnosis of pulmonary embolism is often difficult, and is frequently missed with serious outcomes. Mortality in untreated pulmonary embolism is approximately 30% but with adequate anticoagulation, this can be reduced to 2-8%(8).

**ABSTRACT**

A 70-year-old Chinese woman developed breathlessness, tachycardia and hypotension on the fourth day after total hip replacement. 12-lead electrocardiogram (ECG) showed sinus tachycardia with ST depression in I, II, V5 and V6. The ECG changes of sinus tachycardia along with a typical history is suggestive of pulmonary embolism. Diagnosis, treatment and the use of IVC filter for pulmonary embolism are discussed.

**Keywords:** acute pulmonary embolism, warfarin

**REFERENCES**

### Question 1. The following are risk factors for venous thrombosis:

(a) Protein C deficiency.  
(b) Protein S deficiency.  
(c) Advanced age.  
(d) Non-pregnant woman.  

### Question 2. The following statement is true of ECG characteristics of pulmonary embolism:

(a) A normal ECG excludes the diagnosis of pulmonary embolism.  
(b) There may be concomitant atrial fibrillation.  
(c) Transient left bundle branch block.  
(d) S1Q3T3 is a common finding.  

### Question 3. Which of the following statements regarding the treatment of pulmonary embolism is false?

(a) Target INR range is 2.0-3.0.  
(b) Duration of therapy for first episode of pulmonary embolism is six months.  
(c) Duration of therapy for recurrent pulmonary embolism is lifelong.  
(d) Target INR range is 3.0-4.0.  

### Question 4. Investigations for pulmonary embolism should include the following:

(a) Pulmonary angiography.  
(b) VQ scan.  
(c) Spiral CT.  
(d) Arterial blood gas.  

### Question 5. Which of the following statements about warfarin is false?

(a) Warfarin is contraindicated during the first trimester as it is tetratogenic.  
(b) Warfarin is contraindicated during the last six weeks.  
(c) Warfarin does not interact with ciprofloxacin.  
(d) In patients with an IVC filter inserted warfarin should never be given.  

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