Pulmonary embolism and the electrocardiogram

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CASE REPORT

A 54-year-old man presented with a short-lived episode of blurred vision followed by collapse while shopping. There were no precipitating causes or preceding symptoms. He remained unconscious for less than 1 minute. He recovered sufficiently to continue shopping but presented to casualty 5 hours later with increasing breathlessness. His past medical history included renal colic and a left leg deep vein thrombosis associated with cellulitis 3 years before. His mother had suffered from premature ischaemic heart disease and had died from a pulmonary embolus 25 years before. He took no medication, drank little alcohol but smoked 25 cigarettes per day.

On examination he was an overweight Caucasian man, with normal observations. Examination was unremarkable. Initial investigations revealed an elevated white cell count at 11.5 x10^9/litre (majority neutrophils) with normal urea, electrolytes and creatine kinase. The chest X-ray was normal. The electrocardiogram (ECG) showed a sinus rate of 90 beats per minute with T wave inversion in leads V1 to V4 and lead III.

An initial diagnosis of a non-Q wave myocardial infarction was made. The patient was admitted to coronary care for monitoring, aspirin and intravenous heparin. Over the following 24 hours serial ECG recordings showed progressive deep and symmetrical T wave inversion in both the inferior and anterior leads (*Figure 1*). Cardiac enzymes remained normal.

Diagnostic coronary angiography was performed within 24 hours of presentation. Left ventricular function was normal and no obstructive coronary lesions were seen. Right heart catheterization revealed a significantly elevated pulmonary artery systolic pressure (70 mmHg). Both the right and left main pulmonary arteries were shown to be partially occluded by large pulmonary emboli (*Figure 2*).

Intra-arterial tissue plasminogen activator was administered as an initial bolus of 10 mg followed by an infusion of 90 mg over 2 hours. Follow-up angiography 20 hours later showed complete resolution; the ECG changes resolved over the following 3 days. The patient remains well, taking lifelong warfarin.

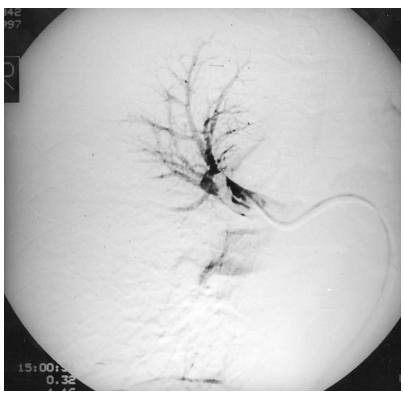
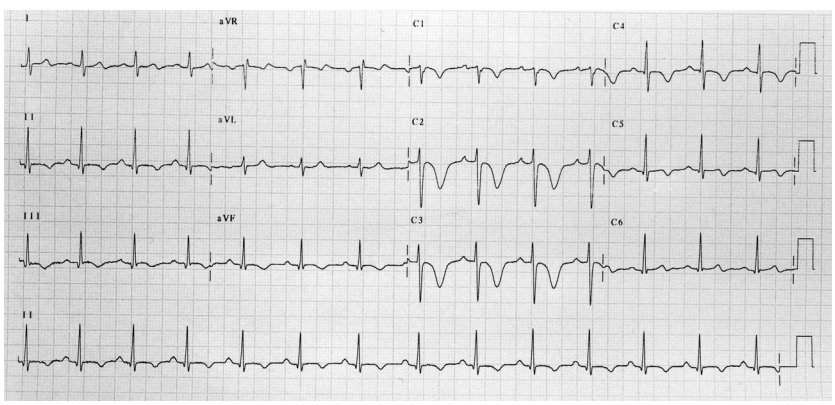


Figure 2. The pulmonary angiogram of the right lung revealing a large filling defect distal to the pig-tail catheter.

INTRODUCTION

Pulmonary embolism can be difficult to diagnose on clinical grounds alone as it often presents with non-specific symptoms (Miller, 1997). A definitive diagnosis may be reached by radioisotope ventilation perfusion imaging, conventional or spiral computed tomography angiography of the pulmonary arteries or transoesophageal echocardiography. However, the above investigations are rarely available on an emergency basis. In contrast, electrocardiography (ECG) is readily available in an emergency but the changes are non-specific. This article discusses the case of a patient who presented with massive pulmonary embolism and misleading ECG changes.

Figure 1. The electrocardiogram 24 hours after admission reveals deep symmetrical T wave inversion in the anterior leads, with further T wave abnormalities in the infero-lateral leads.



DISCUSSION

ECG changes associated with pulmonary embolism were first described by McGinn and White in 1935. They described the 'classical' $S_IQ_{III}T_{III}$ change familiar to medical students. A number of investigators (Stein et al, 1991; Sreeram et al, 1994; Ferrari et al, 1997; Stein and Henry, 1997) have subsequently studied patients presenting with pulmonary emboli to identify those ECG changes that correlate with the diagnosis or the effectiveness of treatment.

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In these studies 80–90% of patients presented in sinus rhythm. Between 2 and 4% had atrial fibrillation, with smaller numbers of patients presenting in atrial flutter, or with multiple atrial or ventricular ectopy. Significant anterior T wave inversion was found in 40–90% of cases, right bundle–branch block, both complete and incomplete, in 15–70%, $S_IQ_{III}T_{III}$ in 12–50%, small voltage QRS complexes in 6–29% and P pulmonale in 5–12%.

Smaller numbers of patients were noted to have left axis deviation, right axis deviation and pseudo-infarction changes. Significantly, the ECG was normal in 10–25%. One case of electrical alternans has been documented (Tighe et al, 1994). Most of these studies investigated small highly selected groups of patients referred to tertiary centres. Patients with preexisting cardiorespiratory disease were excluded. Therefore these data may be less applicable to general clinical practice.

Some investigators have examined the utility of the ECG as an indicator of the severity of pulmonary embolism (Ferrari et al, 1997; Stein and Henry, 1997). The most frequent ECG abnormality in severe embolism was found to be anterior T wave inversion (sensitivity 85%, specificity 81%, negative predictive value 65% and positive predictive value 93%) (Ferrari et al, 1997). The earlier the anterior changes occurred, the more severe the pulmonary embolism was likely to be. Resolution of these anterior changes by day six was a sensitive indicator of successful thrombolytic dissolution of the emboli as shown by pulmonary angiography. Although other ECG changes such as complete right bundle–branch block suggested increasing severity, their absence did not preclude it. At least 10% of patients with severe pulmonary emboli defined by pulmonary angiographic criteria had normal ECGs. This frequency increased as the severity decreased.

The cause of the anterior T wave changes seen in the most severe pulmonary emboli has been investigated (Ferrari et al, 1997). No ischaemia can be detected on coincident cardiac radio-isotope perfusion imaging and coronary artery flow has not been found to be compromised. It has been speculated that the changes may be a function of increased sympathetic drive, similar to the changes sometimes seen in subarachnoid haemorrhage. No overall conclusion has been reached as yet.

CONCLUSIONS

Many ECG changes are possible in pulmonary embolism. The ECG changes occur in the first 24–48 hours, after the change in right-sided pressures. A normal ECG can occur but the frequency decreases as the severity increases. A greater number of ECG changes implies a greater severity of pulmonary embolus. Anterior T wave inversion appears to be the best guide to severity and its resolution is a guide to the success of treatment. The cause of these changes is still elusive. HM

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