CASE REPORT

How ECG can cause confusion in pulmonary embolism and how echocardiogram can help

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A case of pulmonary embolism in which the diagnosis was aided by transthorasic echocardiography is described. Echocardiography may be helpful in emergency presentations, as ECG changes can be neither sensitive nor specific for the diagnosis of acute massive pulmonary embolism.

A 25 year old man presented with a 1 week history of non-productive cough and shortness of breath on exertion. Over the preceding month, he had experienced intermittent palpitations on several occasions. He had received antibiotics from his general practitioner for cellulitis in the left leg. There was no history of chest pain or haemoptysis. Past medical history was of mild asthma. There was no significant family history of medical problems. He was a non-smoker and occasionally drank alcohol.

Examination revealed a moderately built man, who was apyrexial, tachypnoeic, and tachycardic, but not in respiratory distress. Blood pressure was 135/85, pulse regular and 110 beats/min, respiration rate 20 breaths/min, O_2 saturation 94% (on 2 litres of O_2). Chest auscultation revealed a loud second heart sound. An ejection systolic murmur was heard at the left sternal edge radiating to the neck. The chest was clear. There was minimal oedema of the left leg, with a swollen but non-tender calf. There were no abnormal abdominal or CNS findings.

Blood results were: haemoglobin 16 g/l, white cell count 14.4×10^9/l, platelets 226×10^9/l, D-dimer 2 U (normal range 0–0.5). Troponin T and thrombophilia screens were negative. Arterial blood gases showed pO_2 of 10 kPa, pCO_2 of 3.9 kPa, and pH of 7.42. Chest *x* ray revealed enlarged pulmonary arteries, and ECG showed left axis deviation and anteroseptal ST/T depression (fig 1).

A diagnosis of pulmonary embolism (PE) was made and anticoagulation with tinzaparin was initiated, followed by warfarin treatment. An early transthoracic echocardiogram revealed dilated right heart chambers with good left and right ventricle function and mild to moderate tricuspid regurgitation with estimated pulmonary artery systolic pressure of 68 mmHg. There was paradoxical septal motion due to right heart dilatation. A subsequent V/Q scan detected a number of significant unmatched perfusion defects in the left lung and one on the right, suggesting a high probability of PE.

The patient made a good recovery and was allowed home with continuing warfarin treatment.

DISCUSSION

This case highlights the value of early echocardiogram in the diagnosis of acute massive PE. PE is both underdiagnosed and overdiagnosed. Acute PE causes up to 10% of all hospital deaths, with most occurring because of a delay in diagnosis, as PE is easily missed in elderly patients, patients with cardiorespiratory disease, and those presenting with isolated dyspnoea. A high index of suspicion is required to achieve an accurate diagnosis.

Most ECG features in PE lack specificity and sensitivity, and the value of ECG for the diagnosis of PE is debatable. ECG can be normal in pulmonary embolism, and other recognised features of include sinus tachycardia (heart rate >100 beats/min), negative T waves in precordial leads, S_1 Q_3 T_3, complete/incomplete right bundle branch block, right axis deviation, inferior S wave notch in lead V_1, and subepicardial ischaemic patterns. The mechanism for these ECG changes is acute right heart dilatation, such that the V leads that mostly represent the left ventricle now represent the right ventricle (RV). The presence of inverted T waves on precordial leads suggests massive PE.

Echocardiography can be helpful when acute lifethreatening PE is suspected.[2–4 8] However, it should not be used as a screening tool for diagnosing PE as it allows a firm diagnosis in only a minority of cases.[1 6]

RV dysfunction is an important consequence of a large PE. Not only can an echocardiogram identify dilated right heart and pulmonary hypertension, it will also exclude local hypokinesis consistent with myocardial infarction. RV dysfunction following PE helps to stratify patient risk, as RV afterload stress confers a worse prognosis than does normal RV function after PE.[4 7] On very rare occasions, a thrombus can be seen in the right heart en route to the lungs.

The presence of RV afterload stress detected by echocardiography has been shown to be a major determinant of short term prognosis in patients with clinically suspected acute pulmonary embolism.[7] However, RV dysfunction and other previously described echo findings may be observed in a variety of cardiovascular disease including primary and secondary pulmonary hypertension, tricuspid valve disease, dilated cardiomyopathy, RV infarction, and congenital heart disease, and are not specific for PE.

An acute massive PE leading to RV dilatation and failure may further compromise cardiac output and systemic

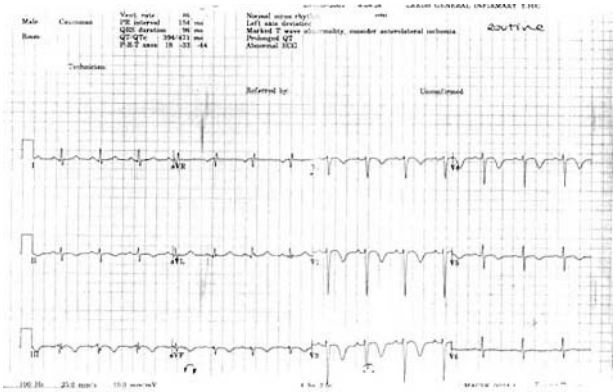

Figure 1 ECG showing left axis deviation and anteroseptal ST/T depression.

circulation through ventricular interdependence and reduced left verntricle filling. This may be associated with decreased coronary perfusion pressure resulting in ischaemia, worsening heart failure, progressing to RV infarction, circulatory arrest, and death. Treatment with thrombolytic and vasoactive agents may improve RV function and clinical outcome.

CONCLUSION

Most deaths from PE occur because of a delay in diagnosis. ECG changes suggesting cardiac ischaemia can cause confusion but are well recognised in massive PE. In these cases, early echocardiogram is important, especially in the presence of hypotension or collapse. Patients with clinically suspected PE have a poor prognosis if they present with right ventricular afterload stress as assessed by echocardiography.

Echocardiogram is a non-invasive, valuable, and sensitive technique for the rapid identification of RV overload in PE, enabling further investigation and treatment to be appropriately directed. It is, however, only sensitive in severe cases of PE, being poorly sensitive in other patients. The current BTS guidelines for the management of patients with suspected PE state that echocardiography is felt to be diagnostic only in massive PE, allowing a firm diagnosis in only a minority of others.[9] Therefore, echocardiogram should be used in the A&E department for haemodynamic assessment of patients when massive PE is suspected and not as a routine screening tool for PE.

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