Massive pulmonary embolus in a 14 year old boy

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Abstract
Pulmonary embolus in children is rare. A case of massive pulmonary embolus, after surgery, in a child of 14 years is described. Accident and emergency doctors should be aware that pulmonary embolus can occur in children and exercise a high index of suspicion for the diagnosis in those patients with risk factors for the condition who present acutely with typical symptoms such as dyspnoea, chest pain, haemoptysis, or collapse.

Keywords: pulmonary embolus; children

Case report
A 14 year old boy attended the accident and emergency (A&E) department having suddenly experienced a feeling of lightheadedness, dyspnoea, and substernal chest pain. According to his mother he had become cyanosed and had lost consciousness for a few seconds. The general practitioner had been called and immediately requested an emergency ambulance as the child was unwell and clinically appeared to have suffered a pulmonary embolus.

Three weeks earlier, the patient had had bilateral slipped upper femoral epiphyses treated with internal fixation. He had since been mobilising slowly on crutches but had been on no anticoagulant treatment.

His mother had previously suffered with three pulmonary emboli but the cause of these had not been investigated and she was no longer on anticoagulant treatment.

On arrival in the A&E department, he was short of breath at rest, with a respiratory rate of 28 breaths/min despite high flow oxygen. He was tachycardic at 138 beats/min with a blood pressure of 112/68 mm Hg. His heart sounds were normal and his chest was clear. There was no clinical evidence of deep venous thrombosis in either leg. He was noted to be obese, weighing 85 kg (on the 98th centile for his age).

A clinical diagnosis of pulmonary embolism was made. Electrocardiography showed a classic S1, Q3, T3 pattern (fig 1) as well as sinus tachycardia. Arterial blood gases (on air) were oxygen tension 8.50 kPa, carbon dioxide tension 4.08 kPa, and pH 7.487. Chest radiography was normal. Venous blood was drawn for a thrombophilia screen before anticoagulation treatment was started with intravenous heparin.

Subsequent spiral computed tomography of the chest showed a large embolus at the bifurcation of the main pulmonary outflow tract, extending into both the right and left pulmonary arteries (fig 2). The embolus was removed by surgical embolectomy. The patient was anticoagulated with warfarin postoperatively and has made a full recovery.

Subsequent analysis of the patient’s and his mother’s blood showed that both were deficient in antithrombin III (an anticoagulant protein).

Discussion
Thromboembolic disease is rare in childhood.1 In postmortem examinations the incidence of pulmonary embolism in children was 3.7%.2 Of these only about half had had clinical symptoms of pulmonary embolus and in only a third of these symptomatic cases was the diagnosis considered before death. The mortality rate for untreated pulmonary embolus in childhood approaches 30%.3 Symptoms include pleuritic chest pain, dyspnoea, cough, haemoptysis, sweats, and syncope. Unfortunately, no such findings alone can accurately diagnose pulmonary embolus and it is essential that medical staff are alert to the possibility of the condition in children if the diagnosis is to be made. Overall, the incidence of venous thromboembolism has been estimated to be 0.6/100 000/year in those aged 0–14 years, and this rises to

Figure 1 Electrocardiography limb leads showing S1, Q3, T3.
The incidence of pulmonary embolism after general surgical procedures to be around 20%\(^2\) and show that the risk persists for more than one month.\(^6\) Prophylactic anticoagulation is of proven benefit for adults at high risk of thrombosis,\(^7\) but no such evidence is available for children.\(^1\)

It is important to take a family history, as this may raise the possibility of an inherited thrombophilia. These include defects of the protein C pathway, protein S deficiency, antithrombin III deficiency, or the presence of antiphospholipid antibodies.\(^5\) In published reports, 65%–70% of children with documented thrombosis had one of these defects.\(^6\)\(^8\) Furthermore, they were found in 90% of children who had proved thrombosis but no obvious acquired risk factors.\(^5\) Children with suspected thrombosis should, therefore, be tested for inherited coagulation disorders, particularly of the protein C pathway. Blood should be drawn for these analyses before anticoagulant treatment is started.

In summary, pulmonary embolus is a rare but potentially fatal condition in children. The risk factors for thromboembolic disease that apply to adult patients also apply to children, but children who suffer from pulmonary embolus often have additional inherited defects of the endogenous anticoagulant proteins. Many cases are missed before death because the diagnosis is not considered. Heightened awareness among A&E staff should aid earlier diagnosis and therefore reduce fatalities.

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