Fatal Spontaneous Haemothorax in a Child With Acute Leukaemia: A Case Report

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ABSTRACT

A 6-year old boy with acute leukaemia developed spontaneous bleeding and presented with spontaneous massive haemothorax. One litre of blood was drained at tube thoracostomy and another 300mls over the next 3 hours. The patient continued to deteriorate and died despite replacement of lost blood. The rarity of spontaneous haemothorax is highlighted and the importance of early diagnosis and treatment emphasised.

KEY WORDS: Acute leukaemia, spontaneous haemothorax.

Introduction

Haemothorax is most commonly due to trauma, pulmonary infarction, pulmonary neoplasia or a complication of surgery and diagnostic procedures.1 Spontaneous haemothorax is uncommon but has been reported in patients on anticoagulants2 and patients with vascular disease.3,4 This is a report of spontaneous haemothorax in a child with leukaemia.

Case Report

A 6-year-old boy was referred from a peripheral hospital with epistaxis and bleeding from injection sites for one month. This was associated with easy fatigability and gradual pedal, facial and abdominal swelling. He also had a dry cough, associated with dyspnoea on exertion but no orthopnoea, paroxysmal nocturnal dyspnoea, haemoptyis or fever. There were occasional episodes of haematochezia. There was no history of administration of anticoagulants and the patient had no past history of tendency to easy bleeding. No family history of bleeding diathesis was obtained.

The findings at initial examination were; temperature of 36.0°C; no pallor; no cyanosis; generalised lymphadenopathy; bilateral pitting pedal oedema and
facial oedema. There were no evidences of bleeding into the skin. The pulse rate was 120 per minute and of small volume and blood pressure 80/60 mmHg. The apex beat was not displaced and there were no added heart sounds. Respiratory rate was 30 cycles per minute and there was no evidence of pleural collection. There was hepatosplenomegaly of 7cm and 5cm respectively.

Haemogram was 7gm/dl, white cell count 14.3 x 10^9/L with lymphocytes of 65%, lymphoblasts 16%, neutrophils 11%, myeloblasts 6% and eosinophils 2%, and platelet count 117 x 10^9/L. These features suggested acute lymphoblastic leukaemia, which was confirmed by bone marrow examination. Bedside whole blood clotting time was over 1 hour and prothrombin time was prolonged by 22 seconds. Liver function tests were normal.

Anaemia was corrected with transfusion of fresh whole blood and appropriate cytotoxic chemotherapy was commenced. The patient however suddenly became dyspnoeic. There was no trauma at this time. There was cyanosis and pallor increased. The trachea was deviated to the left, the right hemithorax was stony dull and breathe sounds were absent in the same area. The patient was in shock and the apex beat was displaced to the seventh left intercostal space along the midaxillary line. A chest radiograph showed massive right pleural effusion, which was confirmed to be haemothorax by thoracocentesis. Five hundred millilitres of the haemothorax was aspirated to improve respiration and 2 units of whole blood were transfused to replace loss. The haemothorax was subsequently drained by tube thoracostomy (1000mls over 30 minutes) under local anaesthesia and oxygen administration by facemask. The tube drained 300mls of blood over the next 3 hours. Cytology of the pleural fluid did not show malignant or blast cells. Blood transfusion was continued but the patient continued to deteriorate. Thoracotomy was considered at this stage (as there were no facilities for thoracoscopy), however the patient died before this could be carried out. Consent for post-mortem examination was not given by the parents.

Discussion

Spontaneous haemothorax is uncommon, though it has been reported in patients on anticoagulants and those with Osler-Weber-Rendu disease and renovascular hypertension. Spontaneous bleeding in patients with acute leukaemia is not unusual but bleeding is usually from skin and mucosal surfaces and is frequently due to thrombocytopenia. Spontaneous bleeding is the reason for presentation in 10% of patients with acute leukaemia, however, haemorrhage into large body cavities is unusual. Persistent cough in this patient could have caused tearing of pleural capillaries; these would normally seal but in the presence of coagulation defects (thrombocytopenia, prolonged prothrombin and clotting times) bleeding would continue. Previous reports have implicated violent emesis and paroxysmal cough in the aetiology of spontaneous haemothorax and haemomediastinum.

Early diagnosis and prompt drainage is necessary to avoid mortality. Coagulation defects must be corrected by transfusion of deficient components where available or fresh whole blood. Treatment of the predisposing factor is the ultimate remedy. Where the cause of bleeding is unknown, selective angiography may be helpful in identifying the bleeding vessel and the cause. Thoracoscopy where available is also helpful in such cases.
Haemorrhage is a frequent cause of death in acute leukaemia,\(^5\) accounting for up to 52% of the death\(^6\) but fatal intrapleural or intraperitoneal haemorrhage is rare.\(^6\) However, as illustrated by this case, intrapleural haemorrhage may be massive, compromise respiration and lead to death.

References


