Severe haemoptysis associated with viral tracheitis

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We describe a case of influenza A tracheitis which was complicated by severe haemoptysis.

Case report

A 51-year-old woman was admitted to hospital after severe haemoptysis (about 200 ml fresh blood on the day of admission). For three days she had been coughing up some amounts of blood. She also complained of a substernal dull chest pain, which was aggravated by coughing. She had never smoked and had no history of malaise, chills, myalgia, rhinorrhea, sore throat, or production of purulent sputum, or of previous respiratory illness.

On admission the patient appeared well and in no respiratory distress. Her pulse rate was 90/min, the blood pressure was 130/80 mm Hg, and the respiratory rate 14/min. Her temperature was normal and she was cyanosed. The right calf appeared slightly swollen but was not tender or inflamed. Examination of the heart and cardiovascular system showed nothing abnormal. Crackles were heard over the right middle lobe.

The chest radiograph was normal apart from patchy infiltration in the middle lobe. The electrocardiogram was normal. Laboratory investigations showed the haemoglobin concentration to be 13.6 g/dl, white cell count 3.8 × 10⁹/l, and platelets 200 × 10⁹/l. The prothrombin index was 100% and the partial tromboplastin time 27-4 seconds (control = 29 s). Arterial blood gases were normal. Ascending contrast phlebography of the right leg carried out shortly after admission gave normal results. Despite the absence of any evidence of deep venous thrombosis or pulmonary embolism anticoagulant treatment with intravenous heparin was started before ventilation and perfusion radioisotopic lung scans were performed the next day. This was soon followed by haemoptysis of a further 250 ml of fresh blood. The partial tromboplastin time at this time was 112 seconds (control = 28-1 s). Heparin treatment was stopped and its effects reversed with protamine sulphate, and the haemoptysis ceased. The ventilation and perfusion lung scans carried out subsequently were normal.

Fibreoptic bronchoscopy was carried out and showed the trachea to be diffusely haemorrhagic with multiple bleeding sites. The mucosa was friable and bled easily. No distal bleeding site could be seen. Viral antibody studies were performed and repeated after 14 days. These showed an eight-fold rise in the titre of antibody to influenza A virus, from an initial titre of <1/8 to a titre of 1/64. There were no significant antibody titres to any other respiratory viruses. Infection due to many other micro-organisms was excluded by culture and serological tests. Tests for antinuclear and rheumatoid factors gave negative results.

The patient was treated symptomatically and had no further haemoptysis.

Discussion

While tracheitis is included in an extensive list of causes of haemoptysis compiled by the American Thoracic Society, we have been unable to find a reference to major haemoptysis attributable to this cause. Massive bleeding from the trachea has, however, been reported in association with tracheo-innominate-artery fistula, and after transtracheal aspiration.

Our patient had haemorrhagic tracheitis that resulted in major haemoptysis, which was subsequently exacerbated by anticoagulant treatment. The patchy infiltrate noted on the chest radiograph was interpreted as being due to aspiration of blood into the right middle lobe. The concomitant eight-fold rise in titre of antibody to influenza A virus is highly suggestive of acute influenza infection, which commonly causes tracheobronchitis. Major haemoptysis complicating this disease, however, has not to our knowledge been previously reported.

References