Vertebral osteomyelitis presenting with pleuritic chest pain and bilateral pleural effusions

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Abstract

Pyogenic non-tuberculous vertebral osteomyelitis presented with pleuritic chest pain and basal shadowing on the chest radiograph.

Pyogenic non-tuberculous vertebral osteomyelitis may present with non-specific symptoms, which make diagnosis difficult. We describe a patient whose initial symptom was pleuritic chest wall pain associated with bilateral basal shadowing on the chest radiograph.

Case report

A 64 year old man presented with bilateral infrascapular pleuritic chest pain and breathlessness. He had had occasional night sweats but no other symptoms and previously he had been active and well. He had never smoked. On examination he was febrile (37.6°C) and tachypnoeic with signs suggesting bilateral pleural effusions. There was no spine tender-

ness and straight leg raising was normal.

A chest radiograph showed bilateral basal pleural shadowing but the thoracic and lumbar spines were normal. Pleural aspiration was attempted but no fluid was obtained. He had microcytic anaemia (haemoglobin 9 g/dl), an erythrocyte sedimentation rate of 117 mm in one hour, and a white cell count of $9 \times 10^9/l$. Blood cultures were sterile. A ventilation-perfusion scan showed matched defects.

With a presumptive diagnosis of pneumonia he was treated with intravenous benzylpenicillin and erythromycin and the radiographic appearances improved. After discharge he developed increasingly severe pain and required opiate analgesics. He became short of breath at rest and complained of frequent drenching night sweats. Chest radiographs then showed recurrent bilateral basal shadowing and the lateral film showed destruction and collapse of the body of the 7th thoracic vertebra with narrowing of the T7-8 disk space and sclerotic changes in the upper margin of T8. Computed tomography of the thorax showed degeneration of the trabecular pattern and loss of the cortical margin of the T7 vertebral body and bilateral pleural thickening. There was some basal atelectasis but the lungs were otherwise normal. Pleural aspiration under ultrasound control produced only 5 ml of a bloodstained exudate, which contained numerous neutrophils and which grew no organisms. The pleura was thickened and a biopsy specimen consisted of fibrinous material and haemorrhagic granulation tissue. Osteomyelitis was suspected and costo-transversectomy and vertebral bone biopsy were performed. Gelatinous material was removed. The histological appearances were those of acute osteomyelitis with no evidence of neoplasia. *Staphylococcus aureus* was cultured from the bone biopsy specimen. Culture for mycobacteria was negative. Blood cultures produced no growth.

The patient was treated with a prolonged course of flucloxacillin and fucidic acid and his pain resolved after six weeks.

Discussion

Non-tuberculous vertebral osteomyelitis may be a subacute disorder with an insidious onset.¹ Two thirds of cases are not preceded by identifiable sepsis elsewhere² and blood cultures are frequently negative.³ Radiographs of the spine may be normal at the time of presentation⁴ and it may take up to eight weeks before radiological changes become apparent.⁵ The erythrocyte sedimentation rate is usually raised, often substantially, but the white cell count may remain within normal limits.⁶ There is often a delay in the diagnosis of vertebral osteomyelitis, partly because it is a disease of very low incidence in Britain⁷ and partly because the cardinal symptom, back pain, is common in the normal population.

This patient had no history of central back pain and the original symptoms and signs suggested a respiratory disorder. We suggest that the early symmetrical chest pains were

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Computed tomogram of the thorax showing destruction of the T7 vertebra and associated pleural changes.

caused by nerve root compression and that bilateral empyema developed as a consequence of direct spread from the infected vertebrae.

A literature search showed one previous report of pleural effusion associated with osteomyelitis.⁴ In that case the effusion was small and not associated with respiratory symptoms. This is the first reported case of prominent pleural pathological features associated with shortness of breath occurring as the presenting feature of pyogenic vertebral osteomyelitis.

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Immune thrombocytopenia complicating pulmonary tuberculosis: case report and investigation of mechanisms

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Immune thrombocytopenia and pulmonary tuberculosis presented concurrently in a 20 year old Thai man as a bleeding diathesis. Intravenous immunoglobulin rapidly corrected the thrombocytopenia. Immunofluorescence and immunoblot studies with platelets and mycobacteria showed the presence of platelet surface membrane IgG.

Isolated thrombocytopenia is an uncommon complication of pulmonary tuberculosis.^{1,2} The pathogenesis is believed to be immune destruction of platelets, though only one previous report has provided evidence to support this hypothesis.³ We report a case of immune thrombocytopenia associated with tuberculosis and discuss mechanisms of platelet sensitisation.

Case report

A 20 year old Thai man presented with a four day history of recurrent mucosal haemorrhages and rectal bleeding with bright blood. He had been unwell for two weeks with a cough producing small amounts of purulent sputum, intermittent night sweats, fevers, and

rigors. In the preceding four months he had noted lethargy and a 9 kg weight loss. There was no personal or family history of tuberculosis. A drug and toxin exposure history was non-contributory. He had emigrated from Thailand in 1980. A chest radiograph in 1982 had been reported as normal.

On examination he was febrile and weighed 42 kg. Bilateral cervical lymphadenopathy was present, the glands ranging in size from 1 to 3 cm. There was no hepatosplenomegaly. He had numerous cutaneous petechiae and haemorrhages in the oropharynx and there was bright blood on rectal examination. Scattered early inspiratory crackles were heard over the left upper zone of the chest.

A chest radiograph showed patchy left upper lobe and right upper and middle lobe opacities. The platelet count was $5 \text{ (normal range } 140\text{--}400) \times 10^9/\text{l}$; the white cell count was $12.7 \times 10^9/\text{l}$ with a slight monocytosis. A microcytic anaemia of 11.7 g/l with a mean corpuscular volume of 73.3 fl was present. Haemoglobin E trait was noted. The erythrocyte sedimentation rate was 38 mm in one hour and the coagulation profile was within normal limits. Bone marrow biopsy showed a normal number of megakaryocytes and no evidence of mycobacterial infection. An autoantibody screen and an enzyme linked immunosorbent assay for HIV antibody gave negative results. Smears of sputum were positive for acid fast bacilli. Sputum cultures grew *Mycobacterium tuberculosis* sensitive to isoniazid, ethambutol, rifampicin, and pyrazinamide but resistant to streptomycin. Cultures of bone marrow were negative.

Intravenous immunoglobulin G (Intragam-CSL, Melbourne) infusion, 0.5 g kg/day , was started on day 1 and continued for four days. Antituberculosis chemotherapy with isoniazid 5 mg/kg , pyrazinamide 35 mg/kg , rifampicin 10 mg/kg , and ethambutol 25 mg/kg was started on the third day. Pyrazinamide and ethambutol were stopped after two months. Owing to problems over compliance isoniazid and rifampicin were continued for nine months.

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