Acute septicaemic melioidosis with pulmonary hilar prominence: a case report with a unique chest radiographic pattern¹

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In acute septicaemic melioidosis chest radiographs most commonly show nodular densities, upper lobe pneumonitis with cavity formation, and irregular mottled densities with dissemination throughout both lungs.

A rarely reported but unique radiographic pattern of bilateral pulmonary hilar prominence in a patient with acute septicaemic melioidosis is described. A triad of pulmonary hilar adenopathy, pyrexia, and suppurative lymphadenitis in a patient with *Pseudomonas pseudomallei* bacteraemia is emphasised.

Case report

A 73-year-old man, a known case of diabetes mellitus, was admitted to hospital on 14 August 1978 with a history of fever and chills for three weeks. When questioned he admitted to slight cough and decreased appetite. On admission he was febrile. There was suppurative lymphadenitis of the right side of the neck and fine rales over the left upper chest. A chest radiograph showed bilateral pulmonary hilar prominence suggestive of tuberculosis (figure). The white

cell count was $9.2 \times 10^9/l$ (86% neutrophils, 2% monocytes, 12% lymphocytes). Results of serum electrolyte and blood urea tests were unremarkable. Blood sugar measured 300 mg/dl. He was treated with isoniazid, ethambutol, and streptomycin, which was discontinued after five days because right cervical lymph node biopsy and culture showed no evidence of tuberculosis, but *Ps pseudomallei*, which was sensitive to amikacin, kanamycin, chloramphenicol, and Septrin.

A change to chloramphenicol, 6 g, Septrin, 2 g, and amikacin, 1.5 g, a day brought clinical response within three days. Three blood culture specimens on 14 August showed the same *Ps pseudomallei* as from the cervical lymph node. Antibiotics were continued for four weeks.

In-vitro and in-vivo cellular immunity evaluation by using E-rosette formation (E) and 2,4 dinitrochlorobenzene reaction (D) in this case showed subnormal values: E-46% and D-negative reaction, as compared with E-77.1 $\pm$ 4.1% and D-80% positive reaction in 20 healthy subjects. Based on the depressed cellular immunity, levamisole, 150 mg was given twice a week for four weeks together with the combined antibiotic treatment. After the immunopotential his cellular immunity reached normal levels within two weeks. A

¹Supported in part by the Rockefeller Foundation, National Research Council, Bangkok, Thailand.

Chest radiographs showing bilateral hilar adenopathy.

chest radiograph during the third week in hospital showed resolution of the hilar adenopathy. Since his discharge from hospital he has remained well.

Discussion

Melioidosis is a rare disease of man caused by *Ps pseudomallei*, which may appear acutely with septicaemia and resemble typhoid fever, staphylococcal bacteraemia, tuberculosis, or systemic mycosis. In acute septicaemic melioidosis there is a spectrum of chest radiographic abnormalities ranging from obviously mild infiltrates to bilateral nodular densities. The pattern may comprise nodular densities, upper lobe pneumonitis with cavity formation, and irregular mottled densities with dissemination throughout both lungs. These abnormalities are basically acute pneumonia with lung abscess, macroscopically resembling miliary tuberculosis. A rarely reported but unique pattern of pulmonary hilar prominence seen in acute septicaemic melioidosis has been omitted in many standard textbooks of pulmonary disease (Mitchell, 1974; Sanford, 1974). The differential diagnosis of hilar adenopathy includes tuberculosis, histoplasmosis, coccidioidomycosis, sarcoidosis, silicosis, lymphoma, bronchial adenoma, and bronchogenic carcinoma. For the treatment of disseminated melioidosis at least two antibiotics have been recommended for 30 days (chloramphenicol, 9–12 g, kanamycin, 2–4 g, and sulphonamide, 4–6 g, daily). In recent years we have seen several cases of disseminated melioidosis; almost all received multiple antibiotics (Septrin, 4–6 g, kanamycin or amikacin, 1–2 g, and chloramphenicol, 4–9 g, daily) for four weeks despite the fact that in-vitro studies have shown antagonism between these antibiotics. The mortality rate, however, was up to 95% in acute septicaemic melioidosis treated with a single agent.

Based on the depressed cellular immunity shown in vitro and in vivo in disseminated melioidosis, levami-

sole, a cellular immunopotentiating agent, was given along with multiple antibiotics. Shortly after the immunopotentialization the patient's cellular immunity reached the normal level. Better awareness of this disease in the tropics and earlier clinical identification of patients with systemic involvement will lead to the appropriate treatment with a greater prospect of a favourable outcome (Tanphaichitra *et al*, 1978a, b). The practical relevance of this case is that a combination of a unique pattern of pulmonary hilar prominence, cervical lymphadenopathy, and pyrexia in a seriously ill patient from whom tubercle bacilli cannot be isolated needs special recognition, since earlier diagnosis of acute septicaemic melioidosis should lower the mortality.

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