Letters to the Editor

Melioidosis presenting as septic arthritis in Bengali men in East London

SIR, Melioidosis can be a fatal disease [1] and is endemic in northeastern Thailand [2] and northern Australia. Melioidosis is not endemic in the Indian subcontinent, but it has been described in travellers from Bangladesh [3, 4]. It is caused by the bacterium *Burkholderia pseudomallei*, which is found in moist soil and water in the tropics [5], and can enter the host by inoculation, and perhaps by ingestion and inhalation [6]. The incubation period is from a few days to many years, and immunosuppression, diabetes mellitus and thalassaemia can occur concurrently [7]. The disease has protean manifestations ranging from localized abscess formation to disseminated abscesses, septicaemia and shock [8–11]. Subacute melioidosis presents with fever, weight loss and suppurative abscesses involving numerous anatomical sites. Musculoskeletal melioidosis cannot be clinically differentiated from other infective causes or rheumatoid disorders [7].

In May 1998, a 47-yr-old Bengali man presented with a painful right elbow and a 3-month history of cough, sweats and weight loss. He had been resident in the UK since 1978 and had a past history of successfully treated pulmonary tuberculosis. He had returned 2 weeks previously from a 5-month holiday in Sylhet, in rural Bangladesh. On examination, he had a fever of 38.5°C and a hot swollen elbow (Fig. 1). Investigation revealed newly diagnosed diabetes mellitus (random glucose 22 mmol/l), a normal peripheral white blood cell count (8 × 10⁹/l) and a grossly elevated C-reactive protein (>180 mg/l). The elbow X-ray was normal and the chest X-ray showed cavitation at the right apex. A
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A ‘Pseudomonas-type’ organism with a characteristic antibiotic sensitivity profile was grown and a diagnosis of melioidosis was made.

In August 1998, a 59-yr-old Bengali man presented with a 3-week history of myalgia, arthralgia and fever. He had been resident in the UK for 20 yr with a past medical history of non-insulin-dependent diabetes mellitus. He had returned within the week from a 2-month holiday in Sylhet. On examination, he had a fever of 38°C and during the third week of his admission developed a swelling of his left elbow, both wrists and right knee joint. Investigation revealed a raised peripheral white blood cell count (16 × 10⁹/l) and a grossly elevated serum C-reactive protein (180 mg/l). The joint X-rays were all normal and further imaging revealed only a prostatic abscess. Approximately 5 ml of pus were aspirated from each affected joint and this, together with blood cultures, prostatic aspirate and urine, all grew a ‘Pseudomonas-type’ organism with a characteristic antibiotic sensitivity profile and a diagnosis of melioidosis was made.

In all three of the patients described, the joint aspirates contained many pus cells, but no organisms were seen on microscopy. All the aspirates grew B. pseudomallei within 48 h of culture. However, the organisms were not identified immediately with routine laboratory tests. The microbiological diagnosis was made presumptively (later confirmed by the Public Health Laboratory Service, Colindale) because of the antibiotic sensitivity pattern (resistant to gentamicin, ciprofloxacin and colistin, and sensitive to co-trimoxazole and chloramphenicol), clinical presentation and the travel history. In isolation, this ‘Pseudomonas-type’ organism with this antibiotic profile may be considered an environmental contaminant.

In conclusion, these cases demonstrate that melioidosis should be considered as part of the differential diagnosis in patients presenting with septic arthritis who have travelled to southeastern Asia, northern Australia and the Indian subcontinent.

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