

# ACUTE SEPTICAEMIC MELIOIDOSIS

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Melioidosis is an uncommon disease caused by a Gram-negative bacillus, *Pseudomonas pseudomallei*. It is endemic mainly in Southeast Asia (1) where the causative organism is readily isolated from wet rice fields and recently cleared areas after heavy rainfall (2). Man is presumably infected by contamination of abraded skin or penetrating wounds, or by inhalation of dust containing the organism (3, 4). The disease has a wide spectrum of presentation. The common subclinical infection noted in the healthy endemic population is detected by a positive haemagglutination titre (5). The less common clinical melioidosis may be classified into an acute fulminant septicaemia, a subacute disease or a chronic suppurative disease (3, 6). The acute septicaemic variety is most dreaded and, if untreated, ends in death within two weeks.

Acute septicaemic melioidosis can occur in previously healthy individuals but numerous reports have documented that it was more likely in debilitated patients with diabetes mellitus, chronic alcoholism, malnutrition, leprosy, cancer or trauma (3, 4, 7-9). There is no pathognomonic clinical feature and almost all organ systems may be involved, notably the lungs, skin, soft tissue, liver, urinary tract, bone and joints. The disease presents with sudden high fever, chills, prostration, vomiting, abdominal pain, watery diarrhoea, confusion, cough, tachypnoea and headache. Jaundice, hepatosplenomegaly, septic arthritis, skin abscesses, pustules, lymphadenopathy and muscle tenderness may be detected (3, 4, 6-9).

Routine laboratory investigations are generally not helpful. The blood leucocyte count is slightly raised or normal. If liver abscesses are present, liver function tests may show a raised serum bilirubin, alkaline phosphatase and transaminases. Serological tests have a limited role in fulminant septicaemia. Paired sera demonstrating a four-fold or greater rise in titre over two weeks are rarely obtained because death intervenes within two weeks in the absence of appropriate vigorous treatment. Furthermore a single low-titre positive haemagglutination test does not distinguish between active and past infections.

A serological test that may be valuable in diagnosing active clinical melioidosis is immunoglobulin M – indirect fluorescent antibody test (IgM-IFA test) (10). The chest radiograph may be normal. However when chest radiographic abnormalities are present, they fall into two categories: nodular shadows disseminated throughout the lung fields, and consolidation sometimes with pleural effusion and cavitation within the consolidation (9, 11).

The definitive diagnosis of acute melioidosis rests solely on isolating *Ps. pseudomallei* from blood, pus, sputum, urine, synovial fluid or cerebrospinal fluid. If the disease is suspected, it is good sense to forewarn the laboratory. *Ps. pseudomallei* may be missed by overgrowth of other bacteria and the use of a selective culture medium may increase the recovery (12). A tentative identification of *Ps. pseudomallei* can be entertained when these bacteriological characteristics are present: (i) an unidentified pseudomonad that is gentamicin-resistant but tetracycline-sensitive; (ii) on Gram's, Wright's or Giemsa's stain, the bacillus has darkly stained ends and a clear centre (13); and (iii) after several days on glycerol-nutrient agar, the colonies exhibit a wrinkled appearance with radial folds, and produce a musty odour (1, 3).

Treatment of acute melioidosis is directed at antimicrobial therapy, supportive measures and care of co-existing debilitating disease. The optimal antimicrobial regimen has not been established. Nonetheless prompt antimicrobial therapy is crucial and should be used in combination in huge doses (3, 4, 6, 8, 13). The commonly used drugs are tetracycline, chloramphenicol, co-trimoxazole, kanamycin and novobiocin (3,6). Doxycycline, a tetracycline analogue, is better tolerated and has been used successfully (8). In one report, the most active antibiotics *in vitro* were ceftazidime, piperacillin and the new monobactam carumonam (14). However only ceftazidime has been documented to be used successfully (15). The earlier penicillins, aminoglycosides (except kanamycin), first and second generation cephalosporins are all ineffective. Based on these considerations and personal experience, the recommended regimen here is ceftazidime 2g 8-12 hourly plus doxycycline 6 mg/kg/day plus one of the following: co-trimoxazole (trimethoprim 9 mg/kg/day, sulphamethoxazole 45 mg/kg/day) or chloramphenicol 80 mg/kg/day. Treatment duration is 3-6 months but ceftazidime may be stopped after 1-2 months. With increased awareness and better treatment, it is hoped that the present mortality rate of 50% (8) may be reduced.

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