

CASE REPORT

MELIOIDOTIC SPONDYLITIS MIMICKING TUBERCULOUS SPONDYLITIS

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Melioidosis, an infectious disease caused by *Pseudomonas pseudomallei*, may be disseminated, localized, or subclinical. It can imitate various common infectious diseases (Kosuwon *et al*, 1993). Melioidotic spondylitis is a relatively rare pyogenic infection of musculoskeletal system and its clinical presentation may mimic tuberculous spondylitis.

A 52-year-old man came to hospital with the chief complaint of backache for 4 months. The patient had had diabetes mellitus for 9 years. He had frequently traveled to northeastern Thailand. Four months prior to admission, he suffered from backache. The pain progressively increased in 2 months before admission with fever, weakness of both legs and difficult walking and urine voiding. Physical examination revealed body temperature 38°C, tenderness at T8 to T9 vertebrae, paraparesis with sensory loss (pinprick sensation) below T9 vertebral level. Both legs also showed increased deep tendon reflex, positive clonus and Babinski sign. Investigation showed hemoglobin 15.1 g/l, white blood cells 6,300 /mm³ with neutrophils 82.4% lymphocytes 14%, monocytes 3.6%, platelets 105,000 /mm². Blood chemistries showed fasting plasma glucose 262 mg/ dl, blood urea nitrogen 16 mg/dl, creatinine 1 mg/dl. Electrolytes showed Na 138 mEq/l, K 3.6 mEq/l, Cl 101 mEq/l, HCO₃ 21 mEq/l. Catheterized urine showed specific gravity 1.030, pH 5, red blood cells 30/hpf, white blood cells 8/hpf. Anti-HIV was negative. Chest film showed loss of delineation of T9 vertebral body with a paravertebral soft tissue mass (Fig 1). X-ray of the thoraco-lumbar spine showed bony destruction of T9 vertebral body with loss of end plate and narrow of disc space between T9 and T10 vertebrae (Fig 2). Computerized scan showed bony destruction of T9 vertebra with paravertebral soft tissue swelling and epidural abscess (Fig 3). The provisional diagnosis was tuberculous spondylitis with epidural abscess with



Fig 1- Chest film showed paravertebral soft tissue mass (arrowhead) from approximately T8 to T10 vertebral levels.



Fig 2- Lateral roentgenogram of thoraco-lumbar spine showed bony destruction of T9 vertebral body with loss of end plate and narrow of disc space between T9 and T10 vertebrae (arrowhead).



Fig 3—Computerized scan showed bony destruction of T9 vertebra with paravertebral soft tissue swelling.

spinal cord compression. Anterior debridement and fusion with vascularized rib graft was performed (Currier and Eismont, 1992). Hemoculture grew *Pseudomonas pseudomallei*. Pus from T9 spine revealed gram-negative bacilli with safety pin appearance and the pus culture grew *Pseudomonas pseudomallei*. Acid-fast bacilli were not found from the pus. Thus, the final diagnosis was melioidotic spondylitis. Intravenous ceftazidime 2g was given 8 hourly with oral augmentin 750 mg *tid*. Fever was reduced to normal by 2 weeks after treatment. Cetazidime was administered for 28 days. Augmentin was continuously given for 4 months.

Thus, the patient had a long history of back pain and the x-ray finding revealed thoracic spondylitis with soft tissue mass. The clinical presentation was considered most likely to be due to tuberculous spondylitis rather than common pyogenic spondylitis such as *Staphylococcus* spondylitis (Currier and Eismont, 1992). However, pus from the spine and hemoculture confirmed that the patient had melioidotic spondylitis, not tuberculous spondylitis.

Melioidosis is usually associated with other diseases, such as thalassemia or diabetes mellitus; concurrent disease involves lungs more often than other organs (Ashdown and Guard, 1984). This may be due to an abnormal cellular immune response in patients who have melioidosis (Tanphaichitra and Srimuang, 1984). Most musculoskeletal melioidosis is localized and does not involve other organs like this patient (Kosuwon *et al*, 1993). Although the clinical presentation of this patient mimicked tuberculous spondylitis, the underlying diabetes mellitus and history of working in northeast Thailand where melioidosis is endemic (Jayanetra *et al*, 1974; Leelarasamee, 1985) were clinical clues for diagnosis of melioidotic spondylitis.

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