Corynebacterium xerosis as a Cause of Vertebral Osteomyelitis

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We report a patient who developed Corynebacterium xerosis vertebral osteomyelitis 6 months following a decompressive laminectomy. Prolonged parenteral and subsequent oral therapy for 11 months resulted in apparent cure. This is the first reported case of vertebral osteomyelitis caused by C. xerosis.

Diphtheroid bacteria belonging to the genus Corynebacterium are part of the normal flora of the skin, mucous membranes, and gastrointestinal tract; they occasionally produce serious infections in the compromised host (5-8, 13). Generally considered a commensal of the human conjunctival sac (13), Corynebacterium xerosis is an irregularly staining, club-shaped rod with occasional granules. The organism ferments carbohydrates and reduces nitrates but does not liquefy gelatin or hydrolyze urea. Isolates of C. xerosis are generally susceptible to the penicillins and cephalosporins and variably resistant to the aminoglycosides and chloramphenicol. Previous reports have described C. xerosis endocarditis, pneumonitis, and cutaneous infections in compromised hosts (4, 7, 8, 15). To our knowledge, this is the first case of C. xerosis vertebral osteomyelitis.

A 61-year-old afebrile male was admitted to the Johnson City Medical Center in December 1987 with complaints of low back and left leg pain. The patient had a history of L4 vertebral disk prolapse documented by myelogram and computed tomography scan and treated by decompressive laminectomy in June 1987 with freeing of the L4 nerve root. The surgery was uneventful, and the patient did well for 6 months postoperatively, after which time pains recurred. Plain radiographs and a magnetic resonance imaging scan at this time revealed destructive changes in the bodies of L4-L5 and swelling and destruction of the intervening disk (Fig. 1). The patient was admitted for biopsy of the lesion.

Blood pressures were 110/70 mm Hg lying and 70/40 mm Hg standing. The patient had masklike facies. Chest examination was normal, and there were no cardiac murmurs. Examination of the back revealed point tenderness over the L4 vertebral spine. Neurological examination revealed bradykinesia but no rigidity. The patient was evaluated by a neurologist who felt he had manifestations of the Shy-Drager syndrome (primary autonomic failure with central neurological disease).

The patient underwent three Craig needle biopsies of L4-L5. A Gram stain of biopsy material revealed small gram-positive rods. Histopathologic material was consistent with osteomyelitis. Cultures of all three biopsy materials grew penicillin-, gentamicin-, trimethoprim-sulfamethoxazole-, and ampicillin-susceptible C. xerosis. A few colonies of Alcaligenes odorans were identified from one needle biopsy specimen. Identification as C. xerosis was based on morphological, cultural, and biochemical characteristics. The organism was gram positive and nonmotile, and it demonstrated alpha-hemolysis on blood agar. Biochemical assays were performed with RIM sugars (Austin Biological Labs, Austin, Tex.) and the API 20S identification system (Analytab Products, Plainview, N.Y.). The organism was catalase positive, fermented glucose and sucrose, and utilized arginine; utilization of mannitol was weak. The organism generated the code number 0000102 on the API 20S system. The patient’s hemoglobin was 13.8 g/dl, the leucocyte count was 9,700/mm³ with a normal differential, and the Westergren erythrocyte sedimentation rate was 50 mm/h. Blood cultures were negative. Serum protein electrophoresis was normal.

The patient was initially treated with cefazolin (2 g intravenously every 6 h) and aztreonam (1 g intravenously every 8 h). A long-term indwelling (Hickman) catheter was placed,

FIG. 1. Magnetic resonance imaging showing destructive changes in L4-L5 and the intervening disk (arrows). V, Normal vertebra; D, normal disk.

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and since the \( A. \) odorans and \( C. \) xerosis were susceptible to trimethoprim-sulphamethoxazole and ceftriaxone, the patient was discharged on six tablets daily of trimethoprim-sulphamethoxazole (160/800 mg) and 2 g of ceftriaxone intravenously daily for a total of 3 months. He was then continued on oral trimethoprim-sulphamethoxazole at the same dosage for 8 months. His condition steadily improved during therapy. One year after diagnosis, the patient had no recurrence of symptoms and a repeat magnetic resonance imaging showed marked improvement.

In immunocompromised hosts, \( C. \) xerosis has demonstrated significant pathogenic potential, producing septicemia, endocarditis, pneumonia, and serious skin infections (2, 4, 7, 8, 10, 13, 15). Although septic arthritis caused by \( C. \) xerosis has been described (14), spinal osteomyelitis caused by the organism in an immunocompetent host has not been previously reported.

Diphtheroid organisms have been isolated from cultures of osteomyelitis and shown to be pathogenic in these instances (1, 3, 9, 11, 12). Morrey et al. (9) emphasized the importance of compromised host resistance, either local or systemic, in the pathogenesis of bone and joint infection caused by corynebacteria. One patient with \( Corynebacterium \) haemolyticum infection of the spine had diabetes mellitus (1). Another patient with \( Corynebacterium \) group JK was the organism implicated (3). Propionibacterium acnes has been incriminated as the causative organism of vertebral osteomyelitis (11, 12). In our immunocompetent patient, seeding of the spine by \( C. \) xerosis may have been a direct result of surgery, but a later hematogenous spread to the site is conceivable.

**LITERATURE CITED**