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Osteomyelitis and Intervertebral Discitis Caused by *Pseudomonas pickettii*

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***Pseudomonas pickettii*, a nonfermenting, gram-negative rod, is rarely pathogenic. Previous reports of infection with *P. pickettii* have largely involved direct contamination of supplies presumed to be sterile. We describe a case of vertebral osteomyelitis and intervertebral discitis caused by *P. pickettii* in a debilitated patient. The aggressive nature of this infection demonstrates that *P. pickettii* may be a more invasive organism than previously noted, particularly in hosts with weakened immunity secondary to underlying disease.**

Pseudomonas pickettii is classified in the rRNA homology group II, with DNA homology to *P. solanacearum* (18). Unlike the other species in the genus *Pseudomonas*, however, it rarely is pathogenic. Cases in which infection has been reported generally involve simple bacteremia, or pyuria, related to foreign bodies or contamination of supplies presumed to be sterile (1, 5–8, 11, 14–16, 20). We describe a case of *P. pickettii* vertebral osteomyelitis and discitis in a patient on chronic hemodialysis. The case is unusual because of both the aggressive nature of the infection (osteomyelitis) and, unlike most previously reported infections with this organism, the absence of an obvious contaminating source for the infection.

Case report. The patient was a 71-year-old black male with chronic renal failure secondary to adult-onset diabetes mellitus and hypertension, undergoing chronic hemodialysis three times per week via a synthetic graft. He had a history of cirrhosis with ascites, presumed to be secondary to alcoholic liver disease. He was known to have a stable lung nodule which had been monitored for 5 years. He was admitted to the Ann Arbor, Mich., Veterans' Affairs Hospital on 15 October 1990 with atypical chest pain and shortness of breath. Myocardial infarction was ruled out, and cardiac catheterization revealed normal coronary arteries. He was given 125 mg of methylprednisolone intravenously (i.v.) for bronchospasm and then placed on a rapid prednisone taper, and his dyspnea improved. He was discharged from the hospital on 22 October, and he completed his prednisone taper at the end of the month. In the last week of October 1990, he developed progressive weakness of the lower extremities and watery diarrhea with loss of bowel control. He was admitted to the inpatient medicine service at the Ann Arbor VA Hospital on 2 November 1990 with a tentative diagnosis of spinal cord compression.

Physical examination on admission revealed a thin black male in no acute distress. He was unable to walk. His temperature was 99.3°F (37.4°C) orally, his blood pressure was 116/64, his heart rate was 98 beats per min, his respiratory rate was 16 per min, and his lungs were clear. A cardiovascular examination revealed a grade II/VI systolic murmur at the lower left sternal border which spread to the apex. An abdominal examination revealed a 14-cm liver, and ascites was once again noted. His stool was positive for occult blood. A neurological examination revealed 3/5 strength in both lower extremities; the patient had diminished sensation in response to pinpricks in both of the lower extremities and the perianal region and had diminished vibratory sense in the lower extremities. A Babinski reflex was observed in the right foot; deep tendon reflexes were not elicited. Laboratory examination was notable for a leukocyte count of 15,000 (71% segmented neutrophils, 2% band neutrophils, 14% lymphocytes, and 12% monocytes). A myelogram with computed axial tomography revealed obstruction to cerebrospinal fluid flow at the level of L4-L5; a destructive lesion extending from vertebral bodies L4 and L5 impinged upon the cauda equina. The patient was given 1 g of vancomycin and 2 g of ceftriaxone i.v. He also received dexamethasone at this time.

The patient underwent a decompressive laminectomy at L4-L5 with removal of the intervertebral disc between vertebrae L4 and L5. The bone and disc were friable, but no gross pus or neoplasm was found. Gram staining of these specimens revealed no organisms. Postoperatively, the patient did well, with swift recovery of neurologic function and return of bowel control. Five days postoperatively, the patient's leukocyte count was 14,200 and cultures of both the vertebral bodies and the intervertebral disc grew a gram-negative, lactose-nonfermenting bacillus. The isolate was oxidase positive and unable to grow on acetamide agar. Final identification as *P. pickettii* was determined by 48-h reactions in the API nonfermenting strip (Analytab Products, Plainview, N.Y.). In this system, the organism reduced nitrate to nitrogen and utilized D-glucose, L-arabinose, D-gluconate, L-malate, and citrate as single carbon sources. All other parameters tested in the API system were negative. The isolate was sensitive to ampicillin, ampicillin-sulbactam, piperacillin, cephalothin, cefoxitin, trimethoprim-sulfamethoxazole, ciprofloxacin, amoxicillin-clavulanic acid, cefuroxime, ceftriaxone, ceftazidime, imipenem, aztreonam, ticarcillin-clavulanic acid, cefotaxime, cefoperazone, and mezlocillin. A 6-week course of orally administered trimethoprim-sulfamethoxazole was begun. Nine days later, the patient's leukocyte count had fallen to 8,500 and he was discharged to a nursing home. He remained well, and when last seen in January 1991, he had normal neurologic function and a normal leukocyte count.

P. pickettii is a nonfermenting, gram-negative, aerobic bacillus. The organisms *P. thomasii*, group IVd, and group Va-1, which have been independently described, appear to

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TABLE 1. Previously reported infections with *P. pickettii*, *P. thomasii*, group IVd, and group Va-1

Reference(s)	Type of infection	Organism(s)	Comment
2	Bacteremia	*P. pickettii*	Diabetes mellitus; unknown source
8	Bacteremia, wound	*P. pickettii*	3 cases; one i.v. catheter contamination
11	Bacteremia	*P. pickettii*	Contaminated chlorhexidine solution
14	Bacteremia	*P. pickettii*	Cancer patients; contaminated catheter flush
5	Bacteremia	*P. pickettii*	i.v. catheter contamination
6, 16	Respiratory (colonization)	*P. pickettii*	Saline contamination
15	Bacteremia	*P. pickettii*	Contaminated narcotics
1		*P. thomasii*	Water contamination
20	Various; different patients	*P. thomasii*	Contaminated supplies
13	Respiratory (colonization)	Group IVd	Chronic lung disease
7	Endocarditis	Group IVd	Parenteral drug use
4	Meningitis	Va-1	
10	Bacteremia	Va-1	
21	Various	Va-1	Both water contamination and clinical specimens
19	Gastrointestinal tract	Va	Cell wall-defective bacteria; possible link to Crohn's disease
3, 9	Pseudo-outbreak	Multiple, predominantly *P. pickettii*	Contaminated transport medium
17	Oropharynx	Multiple, predominantly *P. pickettii*	Myelosuppressed cancer patients; colonization

be strains of the same species on the basis of biochemical testing and DNA base pair composition (12, 21, 22), although *P. pickettii* and *P. thomasii* have been differentiated on the basis of sodium dodecyl sulfate-polyacrylamide gel electrophoresis (3). While these organisms are frequently found in the water supply, they have not previously been considered to be aggressive pathogens. This is the first reported case of osteomyelitis (and discitis) caused by *P. pickettii*. Previously, infection with *P. pickettii* has largely been associated with contamination of hospital supplies (Table 1). Sputum colonization in hospitalized patients, secondary to contamination of nebulized saline, has been reported, although this did not lead to clinical illness (6, 16). Fujita et al. reported a case of *P. pickettii* bacteremia associated with an intravascular catheter (5), with the infection clearing following removal of the catheter and administration of a parenteral antibiotic to which the organism displayed resistance in vitro. Lacey and Want reported an outbreak of *P. pickettii* bacteremia in seven pediatric oncology patients which was attributed to contaminated Hickman catheter flush solution (14); all infections were cleared with antibiotics and discontinuation of the particular batch of flush solution. Kahan et al. reported a series of six episodes of *P. pickettii* septicemia in a coronary care unit that were associated with contaminated chlorhexidine skin-cleansing solution (11). Hansen et al. described two cases of *P. pickettii* bacteremia and one case of a wound infection in hospitalized patients; although no source was identified in two, one had intravenous catheter contamination (8). Chomarat et al. reported a case of bacteremia with *P. pickettii* in a hospitalized diabetic without a source noted (2). Recently, Maki et al. described an outbreak of *P. pickettii* bacteremia in surgical patients which was traced to contamination following tampering with fentanyl supplies in the hospital pharmacy (15). Group IVd and *P. thomasii* have been reported to cause respiratory tract colonization, urinary tract infection, and catheter-related bacteremia, all associated with contaminated hospital supplies (1, 13, 20). One case of fatal endocarditis in a user of parenteral narcotics was attributed to group IVd (7). Pickett and Greenwood reported isolation of group Va-1 from several sites, including clinical specimens (knee aspirate, pericardial fluid, and eye), hospital water supplies, and nonhospital sources (cosmetics) (21). Whether identifiable inoculation of the clinical specimens with contaminated supplies occurred was not discussed. A case of septicemia caused by Va-1 in a patient with neurologic sequelae of encephalitis was reported by Japp et al. (10); no source was identified, although note was made of intravenous catheter phlebitis and an indwelling central venous catheter. One case of meningitis attributed to group Va-1 has been described (4); there was no obvious contamination in this report. Lastly, Parent and Mitchell postulated cell wall-defective group Va bacteria as an etiology of Crohn's disease after isolating the bacteria from the guts and mesenteric lymphatic tissues of several patients with the disease (19). However, there has been no subsequent confirmation of this correlation.

The most unusual feature of this case is the invasive nature of the patient's infection. Previous infections described can predominantly be linked to direct inoculation of the blood or another site of infection and, other than the case of meningitis and two cases of endocarditis (4, 7, 11), were not associated with localized, invasive disease. Therefore, the bone and disc destruction seen in this patient represents notably more virulent behavior than that generally associated with this organism. *P. pickettii* was unlikely to be a contaminant, as the specimens were taken intraoperatively by using a sterile technique and the organism grew from multiple sites. Additionally, the patient's leukocytosis cleared after administration of antibiotics to which the organism was susceptible. One potential portal of entry for *P. pickettii* in this patient was the hemodialysis machines and catheters used to dialyze him three times per week. However, through the subsequent months, no case of *P. pickettii* infection was uncovered at the Ann Arbor VA Hospital, despite regular, continued use of the hemodialysis equipment. The patient may have been infected during the preceding hospitalization, by introduction of *P. pickettii* through an i.v. catheter or during cardiac catheterization. This patient's situation was complicated by underlying diabetes, liver disease, and uremia and by administration of corticosteroids, all of which increased his risk of infection. The multifactorial immune suppression in this patient may have facilitated the aggressive course following infection with this unusual pathogen. This case shows that *P. pickettii* may be a more virulent pathogen than previously believed. In the setting of a debilitated patient, destructive, localized

infection may occur at sites distant from the site at which the organism was originally introduced.

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