Neisseria sicca Osteomyelitis

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Received 12 February 1982/Accepted 25 May 1982

Neisseria sicca was identified as the cause of vertebral osteomyelitis in a male patient who had previously suffered a nonpenetrating, traumatic back injury. The identifying characteristics and antimicrobial susceptibility patterns are presented for this rare human pathogen, which heretofore has not been reported as a cause of infection localized to bone.

Neisseria sicca is normally considered a harmless commensal of the upper respiratory tracts of humans (6). Infrequently, however, this organism has been identified as the cause of significant human infections (5). These include meningitis (2, 7), endocarditis (4), and pneumonia (3). Described herein is a patient shown to have N. sicca vertebral osteomyelitis. The organism was recovered from a vertebral biopsy specimen and from blood cultures. N. sicca has been reported once previously as the cause of intervertebral disc space infection (8). To our knowledge, the present case represents the first report of N. sicca infection localized to bone.

Case report. A 52-year-old man was admitted to the University of Massachusetts Medical Center for evaluation of back pain and fever. Six weeks before admission, the patient fell from a moving truck and incurred right-sided lumbosacral back pain and paraesthesias of the right leg. His symptoms subsided with rest until 7 days before admission, when his symptoms recurred, along with a temperature of 39°C. He was admitted to a local hospital because of difficulty in voiding and severe back pain. On examination, spasms of the lumbar back musculature and limited range of motion of both legs were noted. A peripheral leukocyte count revealed 10,400 leukocytes per mm3, with 82% polymorphonuclear leukocytes, 1% band forms, and 17% lymphocytes. The erythrocyte sedimentation rate was normal. Multiple blood cultures were performed, and the patient was treated initially with cephalothin, 2 g given intravenously every 6 h. Lumbosacral spine X rays revealed degenerative changes at the L5-S1 level. A technetium-99 bone scan and myelogram were normal. After 7 days, he was transferred to the University of Massachusetts Medical Center because of persistent fever and severe back pain.

Upon admission, the physical examination was normal except for a temperature of 39.3°C, muscle spasms, and fullness and tenderness localized at the L5-S1 level. A peripheral leukocyte count demonstrated 9,900 leukocytes per mm3, with 51% polymorphonuclear leukocytes, 11% band forms, 18% monocytes, and 20% lymphocytes. The erythrocyte sedimentation rate was elevated to 121 mm/h. The serum alkaline phosphatase level was 218 U/liter (normal level, 30 to 115 U/liter), with bone isozyme predominance. Blood cultures done 7 days previously at the local hospital remained negative. Cephalothin therapy was discontinued. Repeat X ray and tomography of the lumbosacral spine both demonstrated degenerative narrowing of the L5-S1 disc space, with tapering of the posterior aspect of the fifth vertebral body. A technetium-99 bone scan showed increased uptake in the fifth vertebral body.

Seven days after admission, a Craig needle biopsy of the L5 vertebral body and aspiration of the L5-S1 disc space were performed. Nonspecific, chronic bony and paravertebral soft tissue inflammation was observed histologically. A gram smear of the vertebral biopsy material was negative; however, cultivating yielded small numbers of colonies identified as N. sicca. A culture of the disc space aspirate was negative for N. sicca. The patient became afebrile and noted significant improvement in back pain, mobility, and voiding coincident with the needle biopsy and aspiration. Multiple blood cultures done during the previous week remained negative. The serum alkaline phosphatase level was normal, and the erythrocyte sedimentation rate fell to 58 mm/h.

During the next 7 days, the patient’s back pain continued to improve; however, a report was received from the local hospital that three out of three blood cultures done on day 1 of admission, 3 weeks previously, had just yielded N. sicca.
Blind subculturing of these blood cultures to enriched chocolate agar had been performed only on day 2 of incubation and were negative. Macroscopic examinations, which had been performed daily for the first 7 days, were also considered negative. A comparison of the antibiogram of this organism with that of the strain recovered from the vertebral biopsy specimen demonstrated that the two organisms were identical, and we elected to treat the patient with cephalothin, 2 g given intravenously every 4 h, based on the results of in vitro susceptibility testing. Peak serum levels 3 days after initiation of cephalothin therapy demonstrated bactericidal activity at a dilution of 1:4. Trough levels were less than 1:4. Because of this information, intravenous cefamandole (2 g every 4 h) therapy was initiated. Peak and trough serum bactericidal levels measured 2 days later were 1:16 and <1:4, respectively. Serum bactericidal levels were determined with normal human serum as a diluent and a final inoculum of 10^2 to 10^6 colony-forming units per ml (1). The patient was treated with cefamandole for 5 weeks and discharged from the hospital free of symptoms. Follow-up clinical and laboratory evaluations 1 year later were normal, with X rays revealing residual sclerotic bony densities but no additional destruction of the L5 vertebral body.

**Microbiology.** The organisms recovered both from the vertebral biopsy specimen and from blood culture fluid were oxidase-positive, gram-negative diplococci. Both strains grew well on enriched chocolate agar and 5% sheep blood agar incubated at 35°C in an atmosphere of 5 to 7% CO_2_. Nonpigmented, heaped-up colonies, 1 to 3 mm in diameter, were observed after 24 h of incubation. The colony morphologies of both strains were identical. They failed to grow on modified Thayer-Martin medium at 35°C in CO_2_, but grew well on nutrient agar slants incubated at room temperature in atmospheric air for 24 h. Acid was produced from glucose, maltose, and sucrose but not from lactose in New York City fermentation medium. Nitrate were not reduced. Both strains were identified as *N. sicca* according to the criteria of Morello and Bohnhoff (9).

Agar dilution quantitative susceptibility testing was performed with an inoculum of 10^4 to 10^5 colony-forming units per spot on Mueller-Hinton agar supplemented with 5% chlozaholized sheep blood (10). The minimum inhibitory concentrations (MICs [in micrograms per milliliter]) of various drugs for the organism recovered from the vertebral biopsy specimen were as follows: penicillin G, 0.5; ampicillin, 0.8; vancomycin, 3.1; erythromycin, 12.5; trimethoprim/sulfamethoxazole, <0.05/1.0; chloramphenicol, 3.1; rifampin, 0.8; gentamicin, 0.2; cephalothin, 3.1; cefamandole, 0.8; cefotaxime, <0.2; and moxalactam, 0.4. The MICs for the isolate recovered from blood culture fluid were identical with two exceptions. The MIC of vancomycin for this strain was 1.6 µg/ml; the rifampin MIC was 1.6 µg/ml. *Staphylococcus aureus* ATCC 25923 was used as a control strain for all MIC testing. Because of the variation inherent in agar dilution quantitative susceptibility testing, these differences of one twofold dilution were not considered significant. Thus, the identifying characteristics and antimicrobial susceptibility patterns of the *N. sicca* strains isolated from the bone and blood of our patient were identical.

**Discussion.** *N. sicca* is usually found as part of the normal microflora of the upper respiratory tracts of humans. Despite its apparent limited pathogenic potential, *N. sicca* has been identified as the cause of serious, life-threatening infections. These include meningitis (2, 7), endocarditis (4), and pneumonia (3). In addition, *N. sicca* has been reported as the cause of intervertebral disc space infection (8).

The clinical and laboratory findings for our patient were consistent with the diagnosis of *N. sicca* vertebral osteomyelitis. The organism was recovered both from vertebral biopsy material and from blood. That the organism was recovered only from a vertebral biopsy specimen and not from an aspirate of the adjacent disc space possibly reflects either lack of disc space involvement or sampling error due to small numbers of organisms in this site. Clinical, radiological, and laboratory parameters of infection responded promptly to surgical curettage of the involved vertebra and intravenous cefamandole therapy.

The pathogenesis of this patient’s disease is uncertain. It is possible that, despite normal dentition and the absence of a history of gross dental manipulation, transient seeding of the bloodstream from the upper respiratory tract led to hematogenous seeding of the vertebral body injured during the fall. Another possibility is that the back injury resulted in direct traumatic inoculation of the vertebra. This would seem less likely since the skin adjacent to the infected bone remained intact after the fall and because *N. sicca* is not considered a normal inhabitant of skin.

It is clear from this and other reports that *N. sicca* should be considered a potential human pathogen capable of causing serious infections, including osteomyelitis.

**LITERATURE CITED**