Achromobacter xylosoxidans (Alcaligenes xylosoxidans subsp. xylosoxidans) Meningitis Associated with a Gunshot Wound

RICHARD F. D’AMATO,1,2* MOZAFAR SALEMI,3 ANNA MATHEWS,2 DENNIS J. CLERI,1,2 AND GADDAM REDDY3

Department of Pathology, Division of Microbiology,1 Department of Medicine,2 and Department of Pediatrics,3 The Catholic Medical Center of Brooklyn and Queens, Inc., Jamaica, New York 11432

Received 6 June 1988/Accepted 26 July 1988

The microbiological and clinical features of a case of Achromobacter xylosoxidans (Alcaligenes xylosoxidans subsp. xylosoxidans) meningitis associated with a gunshot wound are described. To our knowledge, this is the third confirmed case report of meningitis caused by this organism.

Achromobacter xylosoxidans (Alcaligenes xylosoxidans subsp. xylosoxidans) is a nonfermentative gram-negative rod described in 1971 by Yabuuchi and Ohyama (9), who isolated it from purulent ear discharges from seven patients with chronic otitis media. Spear et al. (8) recently reported that the organism is uncommonly encountered in clinical specimens. Only two microbiologically or clinically authenticated cases of meningitis caused by A. xylosoxidans have been published. Shigeta et al. (6) described a case of meningitis in a 9-year-old female, and Namnyak et al. (2) reported a case of neonatal meningitis caused by this organism. Shigeta et al. (7) also described six cases of ventriculitis associated with A. xylosoxidans. We report a case of A. xylosoxidans meningitis associated with a gunshot wound.

The patient was a healthy 14-year-old male who was admitted to Mary Immaculate Hospital, Jamaica, N.Y., in February 1988 after a gunshot wound to the chest. He was unresponsive, was bleeding from an entrance wound in the left delto-pectoral groove, and had no breath sounds over the left chest. The patient’s rectal temperature was 37.8°C, his pulse rate was 134/min and regular, his blood pressure was 50 mm Hg by palpation (1 mm Hg = 133.3 Pa), and his respiratory rate was 34/min. The patient was intubated and placed on a respirator, and a chest tube was inserted. His hematocrit at the time of admission was 41.0%, and the leukocyte count was 19,000/mm3 with a differential of 67% neutrophils and 9% bands. A chest X-ray revealed an endotracheal tube and chest tube in place, left hemothorax, a soft tissue density in the left perihilar area representing contusion, a bullet in the right middle thorax, a swallowed incisor in the esophagus, and subcutaneous emphysema in the left chest. Intravenous cefazolin (1 g every 8 h) was administered. The patient underwent exploratory thoracotomy, ligation of bleeding vessels, and repair of a lung laceration within 2 h of admission.

The following day, the patient was awake and paraplegic, with a sensory level at T7-T8 and loss of bladder and anal sphincter control. Computerized axial tomography of the thoracic spine (T4 to T10) that day showed fractures of the spinal pedicles, laminae, and the transverse process of T8 and several bony fragments lying within the spinal canal with extension into the inferior portion of T7. On the second hospital day, a totally transected spinal cord was found when the patient underwent laminectomy and debridement of the involved thoracic spine.

The patient’s temperature remained between 37.8 and 38.4°C, and his leukocyte count remained between 19,000 and 21,000/mm3 while he was on cefazolin during the first 14 days of his hospitalization. On hospital day 15, he complained of headache and photophobia. His temperature rose to 40.1°C, and his leukocyte count was 31,000/mm3. A lumbar puncture revealed cloudy cerebrospinal fluid with a leukocyte count of 19,470/mm3 and a differential of 100% neutrophils, an erythrocyte count of 515/mm3, a protein concentration of 365 mg/dl, and a glucose concentration of 2 mg/dl.

A Gram stain of the cerebrospinal fluid demonstrated ≥1 gram-negative rods, 10 to 15 erythrocytes, and 20 to 25 neutrophils per oil-immersion field. Latex agglutination tests (Directigen; BBL Microbiology Systems, Cockeysville, Md.) for Haemophilus influenzae type b, Streptococcus pneumoniae, group B streptococci, and Neisseria meningitidis groups A, B, C, Y, and W135 were negative.

The patient’s antimicrobial therapy was changed to intravenous nafcillin (2 g every 4 h), cefazidime (2 g every 8 h), and gentamicin (60 mg every 8 h).

The cerebrospinal fluid was subcultured to an enriched chocolate agar plate, an anaerobe blood agar plate, and thioglycolate broth. The chocolate agar plate was incubated at 35°C in an atmosphere of 7.5% CO2, and the anaerobe blood agar plate was incubated at 35°C in an anaerobic jar (BBL). Smooth colonies consisting of gram-negative rods were present on the chocolate agar plate at 24 h of incubation, but no growth was present on the anaerobe blood agar plate at 96 h of incubation. The isolate was nonhemolytic on 5% sheep blood agar and grew on MacConkey agar. It was nonfermentative, producing an alkaline slant and butt on triple sugar iron agar, and was positive for acid production from oxidation-fermentation medium with glucose and xylose, catalase and cytochrome oxidase production, growth at 25 and 42°C, alkali production on Christensen citrate, nitrate and nitrite reduction, motility, and growth on cetrimide agar. Negative reactions were obtained for acid production from oxidation-fermentation medium with mannitol, lactose, sucrose, and maltose, hydrogen sulfide on triple sugar iron agar, lysine and ornithine decarboxylase, and arginine dihydrolase. Based on the aforementioned characteristics, the isolate was identified as A. xylosoxidans (5). This identification was confirmed by apiLAB (Analytab Products, Plainview, N.Y.). The isolate from the chest drain yielded identical reactions and was also identified as A. xylosoxidans.

Antimicrobial susceptibility tests were performed by the
NOTES

They were diffusion isolates had resistant to trimethoprim-sulfamethoxazole, cefotaxime, amikacin by chloramphenicol, tetracycline, gentamicin, and amikacin by the disk diffusion method. The MICs of those antimicrobial agents to which the isolates were susceptible, determined by the disk method, were as follows: azlocillin, ≤2 μg/ml; cefoperazone, 4 μg/ml; ceftazidime, 8 μg/ml; and trimethoprim-sulfamethoxazole, ≤1/19 μg/ml. The MBCs of trimethoprim-sulfamethoxazole and ceftazidime were >4/76 and 16 μg/ml, respectively.

Nafcillin was discontinued, and intravenous trimethoprim (20 mg/kg per day)-sulfamethoxazole (100 mg/kg per day) was added. Cerebrospinal fluid bactericidal activity was detected at a 1:16 dilution.

The patient’s temperature remained between 37.8 and 38.4°C, and he began complaining of low back pain in the region of the laminectomy during the fourth hospital week. A lumbar puncture revealed a sterile cerebrospinal fluid with 8 neutrophils per mm³, 200 erythrocytes per mm³, a protein concentration of 1,270 mg/dl, and a glucose concentration of 22 mg/dl. Computerized axial tomography showed well-defined loculated hypodense areas to the left of the thoracic spine (T7 and T8). The patient was transferred to another institution, where sterile clear fluid was aspirated from the area. He remained on intravenous ceftazidime for 2 additional weeks, at which time he became afebrile and was subsequently transferred to a rehabilitation facility. Multiple blood cultures, taken throughout the patient’s hospitalization, were negative.

A. xylosidans is not commonly involved in infectious disease processes. It is found in the aqueous environment and has been demonstrated as the etiologic agent in otitis media, pneumonia, septicemia, urinary and surgical wound infections, and peritonitis and in an infected orbit; it has been shown to cause nosocomial infections (1, 8). Shigeta et al. (7) reported six cases of cerebral ventriculitis associated with A. xylosidans in a neurosurgical ward. All patients had undergone craniotomy or cranial trephination before the infection. The source of the infection was believed to be contaminated chlorhexidine solution. The first confirmed case of meningitis caused by A. xylosidans was in a 9-year-old female who became infected after occipital trephination (6). Namnyak et al. (2) described a case of neonatal meningitis caused by A. xylosidans in a premature (33-week-old) male. This was the first report of meningitis caused by A. xylosidans in a patient who had not undergone neurosurgery. All but one reported and confirmed case of meningitis or ventriculitis caused by A. xylosidans has been associated with neurosurgery. The source of the organism, in our case, has not been definitely identified. It is possible that the patient’s skin or clothing may have been colonized with A. xylosidans at the time he received the gunshot wound to the chest. The bullet could have transmitted the organism to the pleural cavity, with fragmenting bone contaminating the subarachnoid space.

LITERATURE CITED