Corynebacterium Group JK pathogen in cerebrospinal fluid shunt infection

Report of two cases

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The clinical and laboratory findings in two cases of aerobic Corynebacterium Group JK infection of cerebrospinal fluid (CSF) shunts are described. These organisms have occasionally been reported as a cause of serious infections in man but have not been reported as a cause of shunt infection. In both cases, CSF pleocytosis was limited to 20 or 60 cells with variable protein and sugar values. Fever was a constant finding, frequently accompanied by signs of central nervous system dysfunction. Corynebacterium Group JK organisms are common contaminants of the normal skin flora. When isolated from the blood and/or the CSF of a patient with a CSF shunt who has symptoms and signs compatible with infection, the organism should not be dismissed as a contaminant. A significant feature of this group is its resistance to almost all presently available antibiotics including penicillin, the cephalosporins, and the aminoglycosides. These organisms are, however, sensitive to vancomycin.

KEY WORDS • Corynebacterium • vancomycin • infection, bacterial • cerebrospinal fluid shunt • hydrocephalus

Infections are serious complications of cerebrospinal fluid (CSF) shunts. A wide variety of microorganisms have been responsible for these infections; however, to our knowledge aerobic Corynebacterium Group JK (CJK) organisms have not been reported so far as a cause of shunt infection. We describe the clinical and laboratory findings in two cases with CJK infection of a CSF shunt.

Case Reports

Case 1

This 10-month-old girl was the product of a clomiphene-induced pregnancy in which quadruplets were born in the 34th week of gestation. Her birth weight was 1465 gm. At the age of 2 weeks, she suffered from nosocomial septicemia and meningitis with Enterobacter cloacae and Pseudomonas aeruginosa while being nursed in an incubator. Secondary to these infections, she later developed hydrocephalus. A ventriculoperitoneal (VP) shunt was inserted at the age of 5 months. At the age of 10 months, she was admitted for evaluation of fever spikes of 1 week’s duration, as well as vomiting and poor oral intake which began 24 hours before her admission. Her rectal temperature was 39.6°C, pulse 126/min, blood pressure 100/60 mm Hg, and respiration rate 36/min. She appeared pale and restless, and had a bulging fontanel. The remainder of the physical examination was normal.

The subcutaneous canal of the VP shunt appeared unremarkable. The hemoglobin was 10.2 gm/dl, and the total white blood cell (WBC) count was 14,500/cu mm with 41% polymorphonuclear cells, 53% lymphocytes, 3% band forms, 3% monocytes, and an adequate number of platelets. A CSF sample obtained by puncture of the proximal portion of the VP shunt demonstrated 60 WBC’s (mainly neutrophils). The glucose level was 38 mg/dl (simultaneous blood glucose 77 mg/dl), protein 65 mg/dl, and lactic acid 14 mg/dl. No organisms were seen on Gram staining, but cultures of the CSF and blood grew small white glistening pure colonies of aerobic Corynebacterium, susceptible to vancomycin and amikacin but resistant to penicillin, the cephalosporins, and the other aminoglycosides. The
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minimal inhibitory concentration (MIC) and minimal bactericidal concentration (MBC) of vancomycin is 1.25 and 2.5 μg/ml, respectively.

Initial therapy included vancomycin, 50 mg/kg/day, given intravenously at 6-hour intervals. Within 72 hours after therapy, the patient became afebrile and improved clinically. While she was receiving antibiotic therapy, the VP shunt was removed, but no external ventricular drainage was left. Three days later, another VP shunt was inserted. She was discharged from the hospital with negative blood and CSF cultures. On follow-up examination 18 months later, she is well with no apparent sequelae.

Case 2

This 15-month-old boy was admitted to the hospital with a 48-hour history of irritability, bulging fontanelles, and spiking fever. He was the product of a 27-week pregnancy. On the 10th day of his life, he developed generalized convulsions due to intraventricular hemorrhage. At the age of 6 weeks, a VP shunt was inserted because of hydrocephalus secondary to this intraventricular event. Ten months later, malfunction of this shunt necessitated the insertion of a new VP shunt. Significant physical findings at this admission included a blood pressure of 110/65 mm Hg, a rectal temperature of 39.2°C, a barely palpable spleen, and bulging fontanel. The optic fundi were normal. There were no posturing or lateralizing signs. His head circumference was 53.0 cm (> 97th percentile). Investigations showed a hemoglobin concentration of 9.6 gm/dl and a WBC count of 13,000/cu mm. Withdrawal of CSF for examination showed an opening pressure of 11.0 cm H₂O. The CSF contained 20 WBC's/cu mm (mainly neutrophils); the protein level was 10 mg/dl and the glucose content was 41 mg/dl (blood glucose 75 mg/dl). Blood cultures were sterile, but aerobic culture of the CSF yielded slow-growing small gray colonies identified biochemically as CJK. These organisms were resistant to penicillin, erythromycin, the cephalosporins, and gentamicin, but susceptible to vancomycin and amikacin.

The patient was treated with vancomycin, 50 mg/kg/day, administered intravenously four times daily. Repeat CSF cultures were negative 72 hours following therapy. At this time, serum vancomycin levels were: peak 13.0 μg/ml, trough 1.8 μg/ml. His fever resolved on the 4th hospital day. No apparent sequelae were noted on physical examination at the time of discharge and 12 months later.

Discussion

Shunt infections are the major cause of morbidity and mortality in patients with CSF shunts. The frequency of shunt infections varies from 7.0% to 41.0%. 6 Coagulase-negative Staphylococci are the most common infecting organisms, followed by Staphylococcus aureus. 1,3 Other organisms, including the heterogeneous group of the corynebacteria, Bacillus cereus, 1 and anaerobic diphtheroids, 1,3,5,22 have also been reported. This is the first report of CSF shunt infection caused by CJK organisms.

In 1976, Hande, et al., 10 described "sepsis with new species of corynebacterium" in four patients. This "new species" was later identified as Corynebacterium Group JK. In 1979, Riley, et al., 20 published a list of characteristics for the identification of CJK, based on 95 cultures received at the Centers of Disease Control over a 15-year period. The morphology of the CJK organisms shows Gram-positive coccobacillary or coccal nonmotile organisms. Their growth is strictly aerobic, and colonies are slow-growing, small, gray to white, and glistening; they are usually nonhemolytic on sheep blood agar. Biochemical reactions that differentiate CJK from other corynebacteria are the inability to produce urease, reduce nitrate, or readily ferment most carbohydrates except glucose and galactose under special conditions. All CJK organism isolates have been susceptible to vancomycin. Many strains similar to the present isolates were reported to be resistant to penicillin, the cephalosporins, and aminoglycosides. Human infections with CJK organisms have been mainly described in immunocompromised patients with hematological malignancies 10,18,21 and in patients with infected prosthetic devices, 2,10,16 including prosthetic valves 19 and an epicardial pacemaker. 8 Additional CJK infections include pneumonitis, pyelonephritis, peritonitis, and osteomyelitis in normal hosts. 8,13,21,25 This organism has been found on the skin of 25% to 35% of hospitalized patients. 7,11,23

The exact pathogenesis of CSF shunt infections has not been elucidated, but they may originate from colonization of the shunt at the time of surgery or later, following asymptomatic bacteremia. 19 In the present two cases, infection was evident 5 months following shunt insertion, raising the question of whether colonization of the shunt occurred at the time of surgery with an organism of low pathogenic potential, or whether the shunt was colonized later during bacteremia which was unnoticed. In both cases, intravenous vancomycin resulted in cure of the infection. Although the penetration of vancomycin into the CSF through noninflamed meninges is inadequate, 9,15,17 in cases of meningitis it appears to reach the CSF in sufficient concentration, although this is poorly documented. 9,14 Several cases of cure of methicillin-resistant S. aureus shunt infections have been reported; however, treatment failure when vancomycin was used alone under those circumstances has also been documented. 14,17,24 In our cases, vancomycin concentrations in the CSF were not assayed, but the MIC and MBC of one of the two infecting CJK organisms documented values that are comparable to those observed in Staphylococci. Nevertheless, the rapid therapeutic success in these two cases with an additional foreign body as a confounding factor suggests that further experience with vancomycin administered intravenously is warranted in cases of CSF shunt infections caused by vancomycin-susceptible mi-
cidoorganisms. These two cases further underline the need for reappraisal of the isolation of CJK organisms in blood and CSF cultures of patients with CSF shunts.

References


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