Posttraumatic Brain Abscess Caused by Aeromonas hydrophila

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Posttraumatic brain abscesses are usually caused by Gram-negative bacilli, notably Enterobacteriaceae and Staphylococcus aureus. Although Aeromonas hydrophila is a recognized cause of trauma-related sepsis, it has not been previously isolated from posttraumatic brain abscesses. We describe the first case of Aeromonas hydrophila brain abscess.

CASE REPORT

A 30-year-old male presented to his local primary health care clinic following an assault during which he sustained a right frontal scalp laceration. The details surrounding the injury were vague, and the information was obtained from a referral letter. It was unknown whether there was contamination of the wound with water or soil. After the laceration was sutured, the patient was sent home.

Two days later, he developed signs of a left hemiplegia with associated seizures. He was then transferred to a tertiary institution for neurosurgical intervention. When examined by the neurosurgeon, the patient had a Glasgow coma scale (GCS) score of 8/15 (E2 M5 V1), which was in keeping with severe brain injury and coma. A laceration measuring 5 cm was noted over the right frontal area, and the clinical features of a left hemiplegia were confirmed. The examination of all other systems was normal.

A noncontrasted computed tomography (CT) scan of the brain (Fig. 1) revealed a right frontal hypodense lesion with midline shift suggestive of an early brain abscess or a resolving contusion. A depressed skull fracture was also noted on the right. A contrasted CT scan of the brain confirmed the presence of a right frontal brain abscess. There was surrounding vasogenic edema with mass effect and subfalcine herniation.

The patient underwent an emergency craniectomy with drainage of the abscess and debridement of the wound. During the operation, a right frontal depressed skull fracture with gray foul-smelling pus was detected. The dura and cortical surface were lacerated. The abscess was located in the right frontal lobe and extended from the cortical surface to the periventricular region. It measured 5 cm by 4.5 cm by 4 cm.

A pus swab collected from the abscess cavity was sent for microbiological investigation. The Gram stain of the pus swab demonstrated moderate leukocytes and Gram-negative bacilli. The swab was cultured onto chocolate agar, 5% horse blood agar, MacConkey agar, and colistin nalidixic acid agar and was also cultured anaerobically using 10% blood agar and 10% blood agar with amikacin. The following day, a Gram-negative bacillus was isolated in pure culture on blood, chocolate, and MacConkey agar plates. The blood plate demonstrated large, beta-hemolytic colonies resembling Aeromonas spp. The organism did not ferment lactose. It was oxidase positive and indole positive. The Vitek 2 system (bioMérieux SA, France) identified the organism as Aeromonas hydrophila complex with 98% probability by using software version 5.04. The key biochemical reactions were fermenta-

tion of glucose, maltose, and sucrose, ornithine decarboxylase negative, lysine decarboxylase positive, resistance to vibriostatic agent (O/129), citrate negative, and urea negative. Susceptibility testing was also conducted using Vitek 2. The organism was resistant to ampicillin but was susceptible to amoxicillin-clavulanic acid, cefuroxime, cefotaxime, cefazidime, cefepime, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin, and trimethoprim-sulfamethoxazole. The MIC to extended-spectrum cephalosporins was ≤1 μg/mL. Blood cultures were not done.

Postoperatively and before microbiology results were complete, empirical antimicrobial therapy with ceftriaxone and vancomycin was commenced. Two days after the drainage of the brain abscess, the identification and susceptibility results were available. Despite being treated with ceftriaxone, to which the organism was susceptible, and aggressive surgical treatment, the patient’s condition deteriorated. An urgent CT scan of the brain demonstrated massive vasogenic edema with marked midline shift and transtentorial herniation. A few hours later, the patient had a cardiorespiratory arrest and expired. The postmortem confirmed the presence of pus over the right parietal area and a midline brainstem hemorrhage.

Trauma or neurosurgical procedures cause almost 10% of brain abscesses, and Staphylococcus aureus, Clostridium spp., and Enterobacteriaceae are common pathogens in these cases (1, 2). Although numerous Gram-negative bacilli have been isolated from posttraumatic brain abscesses, A. hydrophila has not been reported previously.

A. hydrophila, true to its name, “water-loving,” is found in a variety of aquatic environments worldwide. These range from groundwater to drinking water, reservoirs, rivers, and lakes. It causes skin and soft tissue infection, gastroenteritis, and septicemia, especially in immunocompromised patients (3). Skin and soft tissue infections usually occur in patients who have been ex-
posed to contaminated water (3, 4) or following leech therapy, as $A. \ hydrophila$, found in the gut of medicinal leeches, aids the digestion of blood (3). Trauma-related wound infections and osteomyelitis have occurred following injuries in aquatic environments (5–7).

$A. \ hydrophila$ is an aggressive pathogen, and a number of virulence factors may play a role in wound infections. Colonization of the wound occurs through pili. In addition, proteases (metalloproteases and aminopeptidases) are produced which degrade connective tissue, and, hypothetically, the degradation products can be used as a source of energy for multiplication. The organisms may then spread by chemotactic motility to deeper tissues which have a higher concentration of nutrients, like proteins, which are required for further replication and spread (3).

In our patient, we hypothesize that when the assault occurred, the laceration may have been contaminated with water or mud harboring $A. \ hydrophila$, resulting in colonization of the wound. The compound skull fracture, which was most likely not detected at the clinic initially, may have facilitated the development of the brain abscess by allowing the infection to extend rapidly to the brain. The patient received appropriate antimicrobial and surgical therapy for the brain abscess and, despite this, he expired. This may be due the infection having progressed significantly over a longer period than the 2 days reported, that the patient had not sought medical attention timeously, and that the initial management of the patient at the clinic had been suboptimal.

$A. \ hydrophila$ infections of the central nervous system are rare, and a few cases of meningitis have been documented. In one case, meningitis occurred in an adult after a craniotomy following head trauma (8). That report is similar to ours in which a preceding head injury may have allowed access of the organism into deeper tissues. Other cases of meningitis in adults have occurred in association with medicinal leech therapy (9) and following exposure to compost, where inhalation of the organism was the proposed mechanism of entry (10). Two reports of meningitis in children occurred in a 3-month-old baby who ingested contaminated water (11) and in a child with sickle cell disease with no known exposure to the organism (12).

The ideal management of $A. \ hydrophila$ central nervous system infections is unknown at present due to the small number of cases in the literature. We postulate that if the organism is susceptible, an extended-spectrum cephalosporin is a suitable choice to treat meningitis because it passes the blood-brain barrier. Extended-spectrum cephalosporins also achieve high concentrations in brain abscess cavities, and they could be used in combination with surgical drainage for the management of $A. \ hydrophila$ brain abscesses (1).

**Conclusion.** We describe the first case of $A. \ hydrophila$ brain abscess that occurred following a head injury. Although $A. \ hydrophila$ is not an unusual cause of trauma-related sepsis, it was surprising that this was the first case of posttraumatic brain abscess. The case illustrates that one should be vigilant of new clinical manifestations of well-recognized pathogens. It also highlights the importance of detecting underlying skull fractures in patients who present with head injuries so that complications may be prevented.

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We have no conflicts of interest to declare.

Informed consent was obtained from the hospital manager. Ethical approval from the institutional ethics board was not required.

**REFERENCES**