Cerebellar abscess and meningitis, caused by *Shewanella putrefaciens* and *Klebsiella pneumoniae*, associated with chronic otitis media

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*Shewanella putrefaciens* is a facultatively anaerobic, non-motile, Gram-negative, non-fermentative bacterium. It is found in various environments and has been isolated worldwide. *S. putrefaciens* is a rare cause of brain abscesses and meningitis. This is a case report of a cerebellar abscess and meningitis caused by *Shewanella putrefaciens* and *Klebsiella pneumoniae* in a river trap fisherman.

**Introduction**

The incidence of intracranial complications such as meningitis and brain abscesses in patients with chronic otitis media has been reported as 0.24–0.45% (Kangsanarak et al., 1993). Antibiotics and early mastoid surgery have dramatically decreased intracranial suppurative complications of otitis media (Goldstein et al., 1998; Kurien et al., 1998). Most abscesses are seen in the temporal lobe or the cerebellum (Kurien et al., 1998). Early diagnosis of intracranial complications may be life-saving (Schwager & Carducci, 1997).

The bacteriology of a brain abscess is diverse and usually consists of a complex mixture of aerobes and anaerobes (Tunkel et al., 2000). *Shewanella putrefaciens*, also known as *Pseudomonas putrefaciens*, is a rare cause of brain abscesses and meningitis (Suzuki et al., 2004). It is a Gram-negative, facultatively anaerobic, non-fermentative bacillus and a ubiquitous organism that has been isolated from many foods, sewage, and both freshwater and salt water (Kueh et al., 1992).

In the present report, a case of cerebellar abscess and meningitis, caused by *S. putrefaciens* and *Klebsiella pneumoniae*, secondary to chronic otitis media is described.

**Case report**

A 28-year-old man was admitted to our clinic with a 4-day history of fever, frontal headache and severe pain of the left ear. The patient was known to have had chronic suppurative otitis media since childhood. He had no history of any congenital or acquired immune deficiency. The patient was living in an overcrowded environment and had poor personal hygiene. He was a trap fisherman and frequently swam in the river.

At admission the patient was lethargic, his skin was pale and his body temperature ranged between 39 and 40°C. He had meningism with no other accompanying abnormal neurological findings. Otoscopic examination of ears revealed the exudation of fresh pus from the left ear. His peripheral white blood cell count was 14.1 × 10⁹ l⁻¹ with 86% neutrophils and 10% lymphocytes. The rest of the blood count findings and serum chemistry were normal. His erythrocyte sedimentation rate was 65 mm h⁻¹ and C-reactive protein was 21 mg dl⁻¹. The cerebrospinal fluid (CSF) had pleocytosis (4100 cells mm⁻³ with 90% neutrophils), a low concentration of glucose (43 mg dl⁻¹) and an elevated protein level (292 mg dl⁻¹). The blood level of glucose was 118 mg dl⁻¹. A magnetic resonance imaging (MRI) scan of the left temporal bone revealed mastoiditis. An MRI scan of the brain showed a cerebellar abscess (3 × 1 cm) (Figs 1 and 2).

Microscopic examination of Gram-stained CSF revealed polymorphonuclear neutrophils, but no bacteria. *S. putrefaciens* and *K. pneumoniae* were isolated from CSF culture (Bactec 9240; Becton Dickinson). These microorganisms were identified with the help of the Phoenix system (Becton Dickinson). *S. putrefaciens* and meticillin-sensitive coagulase-negative staphylococci were isolated from the culture of exudate from the ear in fluid thioglycollate medium. The blood culture was sterile. Antimicrobial susceptibility of the isolates was determined by the automated Phoenix system. *K. pneumoniae* was only susceptible to amikacin, imipenem, meropenem and...
Ciprofloxacin, whereas *S. putrefaciens* was sensitive to all antibiotics except for ampicillin.

According to sensitivity tests the patient was treated with meropenem (3 × 2 g per day). Ten days later, the patient underwent a left lateral posterior fossa craniotomy and 8 ml thick pus was obtained with the drainage of the cerebellar abscess. Aerobic and anaerobic cultures obtained from the abscess were sterile. The symptoms resolved partially after drainage of the cerebellar abscess. Three days later, a mastoidectomy was performed. The mastoid bone and the middle ear were invaded by an extensive cholesteatoma. Exposure of the posterior fossa revealed an epidural abscess. The purulent secretion was drained, the granulation tissue was curedt away and the devitalized bone fragments in the vicinity were removed. The materials of operation were sterile.

He was discharged in a good clinical condition after 8 weeks, without any neurological deficit.

### Discussion

Culture of the abscess fluid or pus is the most important microbiological investigation in the management of a brain abscess (Carpenter *et al.*, 2007). The causative pathogens of brain abscesses vary with time period, geographical distribution, age, underlying medical and/or surgical conditions, and mode of infection (Lu *et al.*, 2002). Limitations of anaerobic culture techniques also influence pathogen prevalence in different studies (Lu *et al.*, 2002). In patients who are operated on with a delay but antibiotics have been started, abscess fluid culture may not yield growth, and early blood culture and CSF culture may contribute helpful information. However, blood culture and CSF culture are helpful only in a small percentage of patients (Carpenter *et al.*, 2007). In our case, the reason for the sterility of abscess material despite isolation of bacteria from CSF and ear exudate cultures may be attributed to the delay in surgery and abscess drainage during administration of antibiotics.

As in this case, polymicrobial organisms may be identified in patients having a chronic otitis media related brain abscess. It should be taken into account that in addition to aerobic bacteria such as *K. pneumoniae*, anaerobic bacteria may be an additional causative agent in cases like this. The material acquired from these cases should be meticulously evaluated and aerobic and anaerobic cultures should be obtained.

*S. putrefaciens* is found in various environmental and animal sources including all forms of water (fresh, stagnant, sea, lake, river and sewage), fish, oily foodstuffs and soil (Tsai & You, 2006; Holt *et al.*, 2005). It is a facultatively anaerobic, non-motile, Gram-negative, non-fermentative bacterium that hydrolyses oxidase. *S. putrefaciens* has only rarely been isolated from such clinical materials as various body wounds, ear discharge, sputum, skin wounds, faeces, conjunctiva, urine, CSF, bile, ascitic fluid, pleural fluid and stored blood (Kueh *et al.*, 1992; Tsai & You, 2006; Holt *et al.*, 2005). This bacterium is isolated worldwide.

Most of the infections reported pertained to contact with contaminated water or injuries or occurrences in which the integrity of the skin was compromised to some extent. *S. putrefaciens* has been associated with biliary tract infections, empyema, bacteraemia, endocarditis, skin and soft tissue infections, including fulminant periorbital-facial

**Fig. 1.** T1-weighted MRI scan demonstrating the cerebellar abscess with enhancement of the lateral sinus.

**Fig. 2.** T2-weighted image showing collection of air in the cerebellopontine angle and diffuse oedema in the cerebellar parenchyma.
cellulitis, dacyrocystitis, perianal abscess, finger abscess, septic arthritis or osteomyelitis, peritonitis, ear infections and ventilator-associated pneumonia in humans (Suzuki et al., 2004; Tsai & You, 2006; Holt et al., 2005). Since S. putrefaciens was first isolated from humans by King (1964), the number of case reports describing this organism as a human pathogen has increased in recent years (Tsai & You, 2006). However, most human isolates of S. putrefaciens occur as part of a mixed bacterial flora, which may overshadow the clinical significance of infections caused by this organism. Most of these patients have predisposing factors such as malignancy, hepatobiliary disease, neutropenia, prematurity, diabetes mellitus, poor living conditions or receiving immunosuppressive drugs (Tsai & You, 2006; Holt et al., 2005). Renal failure may also represent a potential predisposing risk factor. Our patient was living in an overcrowded environment and had poor personal hygiene. Moreover, he was also a trap fisherman and frequently swam in the river. It has been reported that S. putrefaciens may be transmitted via river water (Holt et al., 2005). We think that the source of infection in our patient may have been either river water or poor living conditions.

There are no definitive guidelines for the treatment of S. putrefaciens infections. S. putrefaciens is characteristically susceptible to aminoglycosides, carbapenems, trimethoprim–sulfamethoxazole and quinolones, but resistant to penicillin. Susceptibility to ampicillin and cephalosporins is variable, with more isolates being susceptible to the third- and fourth- than to the first- and second-generation cephalosporins (Tsai & You, 2006; Holt et al., 2005). According to the literature, most Shewanella infections are treated easily by a combination of surgical therapy, drainage and antibiotics (Goldstein et al., 1998; Kurien et al., 1998; Schwager & Carducci, 1997; Tunkel et al., 2000; Suzuki et al., 2004). Treatment options include β-lactams, aminoglycosides, quinolones and carbapenems. We gave meropenem to our patient because K. pneumoniae was resistant to antibiotics except for amikacin, imipenem, meropenem and ciprofloxacin, whereas the identified S. putrefaciens was sensitive to many antibiotics. Treatment of a brain abscess requires a combination of antimicrobials, surgical intervention and eradication of primary infected foci (Lu et al., 2002). There are few indications for the non-operative management of brain abscesses. Occasionally the abscess is inaccessible or there are multiple abscesses. In some cases in which there is a small lesion and the infecting organism is known, medical treatment alone is considered appropriate (Carpenter et al., 2007). Since there was a large brain abscess in our case, surgical drainage was necessary. The basic reason for the delay in surgery in our case was that the patient and his relatives only consented to surgery 10 days after his hospitalization.

The present case may help to redefine the role of S. putrefaciens in human intracranial infections. The association of S. putrefaciens with a cerebellar abscess further extends the clinical spectrum of this rare pathogen. This unusual case highlights that in patients presenting with severe intracranial complications of chronic otitis media, early diagnosis and radical surgical intervention may be life-saving. In conclusion, in chronic otitis media patients with a history of river water exposure, S. putrefaciens should also be considered as a possible causative agent.

References


