Case Report

Correspondence
Nam Yong Lee
micro.lee@samsung.com

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Chronic meningitis caused by *Erysipelothrix rhusiopathiae*

Suk Ran Kim, Min Jung Kwon, Jang Ho Lee and Nam Yong Lee

Department of Laboratory Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

A 47-year-old man presented with headache, nausea, vomiting and fever. Laboratory findings including analysis of cerebrospinal fluid suggested bacterial meningitis. *Erysipelothrix rhusiopathiae* was identified in cultures of cerebrospinal fluid. The patient recovered without any neurological sequelae after antimicrobial treatment. It is interesting that intracranial infection by *E. rhusiopathiae* reappeared after scores of years and that it presented with absence of an underlying cause or bacteraemia.

Introduction

*Erysipelothrix rhusiopathiae* is a Gram-positive, nonsporulating, rod-shaped bacterium that is worldwide in distribution, and it has primarily been seen as a veterinary pathogen (Reboli & Farrar, 1989). Human infection is uncommon, the main infection being erysipeloid, a localized cutaneous disease that is closely related to the occupational exposure of persons who are in intimate contact with animals, their products or waste (Brooke & Riley, 1999). Recently, cases of septicaemia with or without endocarditis or arthritis have been reported presenting more diverse clinical syndromes (Dunbar & Clarridge, 2000; Schuster et al., 1993; Artz et al., 2001; Vallianatos et al., 2003). Only a few cases of cerebral manifestation of *E. rhusiopathiae* infection have been reported, associated with septicemia and endocarditis (Ko et al., 2003; Silberstein, 1965). However, intracranial infection or meningitis by this organism has hardly been reported. Herein we report a case of chronic meningitis with hydrocephalus and isolation of *E. rhusiopathiae* from cerebrospinal fluid (CSF), suggesting the potential of the bacterium to be the cause of bacterial meningitis.

Case report

A 47-year-old man was admitted because of a chronic headache that had lasted for 2 months. He had no particular previous medical history. He was a manufacturer and did not work with animals or keep pets. The headache was associated with night fever, and was insidious and fluctuating in character. Twenty days before admission, he had developed nausea and vomiting with progressive weight loss. Physical examination revealed neck stiffness, suggesting meningitis. He showed no focal neurological deficit.

CSF analysis was first done on the day of hospitalization. The specimen was slightly yellow, and the pressure was 13.6 cmH₂O. The glucose level was 8 mg dl⁻¹, protein was 527.6 mg dl⁻¹ and the white blood cell count was 1110 µl⁻¹ (polymorphonuclear cells 16%, lymphocytes 57% and others 27%). The adenosine deaminase (ADA) level was 40 U l⁻¹ (reference 1–10 U l⁻¹). Suspected as having tuberculous meningitis, the patient had been previously treated with anti-tuberculosis medications (isoniazid, rifampicin, ethambutol and pyrazinamide) since the day before admission. Laboratory tests such as acid-fast stain and culture, PCR and a hybridization method for tuberculosis detection were performed subsequently. The brain MRI scan was compatible with meningitis. On the 10th day of admission, follow-up CSF tapping was carried out. The pressure was 13.0 cmH₂O, the glucose level was 3 mg dl⁻¹ and protein was 408.3 mg dl⁻¹. The white blood cell count was 980 µl⁻¹ (polymorphonuclear cells 67%, lymphocytes 20% and others 13%). The ADA level was 19.9 U l⁻¹. The patient’s headache worsened despite administration of anti-tuberculosis medication, and on the 12th day of admission, hydrocephalus developed, showing an enlarged ventricle size on a brain computed tomography scan. A ventriculo-peritoneal shunt was inserted for 10 days, and simultaneously intravenous ceftriaxone (1 g every 12 h) was administered for a week.

A primary Gram stain was performed on the CSF specimens but no micro-organism was seen initially. However, after culturing the specimens for 2 days in a 5% CO₂ incubator, several clear, circular colonies of about 0.5 mm diameter were distinguished showing alpha-haemolysis on blood agar media. By Gram staining, the organisms were Gram-positive, non-branching filamentous rods resembling mycelial formations. Microscopically, they showed variable length; some were >40–50 µm in length. Isolates were non-motile, and negative for catalase, oxidase and indole reactions. Isolates were typically positive for...
H₂S production on triple-sugar iron medium. These biochemical tests were compatible with characteristics of *E. rhusiopathiae*. The isolates were designated *E. rhusiopathiae* by the Vitek-II System (bioMérieux). The presence of this organism was confirmed again by culture of CSF taken on the 10th day of admission. Blood cultures drawn on the 1st and 5th days of hospitalization were negative. Tuberculosis was not detected by any of the methods previously mentioned. The patient kept taking antituberculosis medication, but after confirming the isolate as *Erysipelothrix*, intravenous ceftriaxone (1 g every 12 h) was additionally administered with ventriculo-peritoneal shunt insertion for the hydrocephalus. A week later, symptoms including the headache subsequently subsided and follow-up CSF analysis was nearly normal [glucose 52 mg dl⁻¹, protein 107.1 mg dl⁻¹, white blood cell count 9 µl⁻¹ (polymorphonuclear cells 3%, lymphocytes 82% and others 15%)]. After removal of the ventriculo-peritoneal shunt, the patient was discharged without further treatment.

**Discussion**

*E. rhusiopathiae* and infections due to this organism are worldwide in distribution. The bacterium has been found as a commensal or pathogen in a wide variety of animal species. It appears that the organism may live long enough to cause infection weeks or months after initial soil contamination, and also it is resistant to many environmental influences. The infection weeks or months after initial soil contamination, and it appears that the organism may live long enough to cause occupational exposure (Reboli & Farrar, 1989; Brooke & Riley, 1999). The route of infection is thought to be scratches or puncture wounds of the skin, but also it appears that the organism has the ability to penetrate intact skin, and cases reported recently describe no contact history with animals. In our patient, his occupation seemed to be irrelevant to this probable first case report, there have been only a few reports about intracranial manifestation of *E. rhusiopathiae* infection. These described brain infarction (Artz et al., 2001; Ko et al., 2003) and possible superficial cerebral metastasis of septic emboli (Silberstein, 1965). In the latter case, the patient showed meningitis symptoms as a complication of *E. rhusiopathiae* endocarditis, but CSF culture was negative. It is interesting that intracranial infection with *E. rhusiopathiae* has reappeared after scores of years and that it presented with absence of an underlying cause or bacteremia.

Identification of the organism is based on the results of Gram staining, lack of motility, H₂S production, indole production, catalase activity and haemolysis on blood agar. H₂S production in particular can be the clue to confirming the identity of Gram-positive rods isolated in the presence of polymorphonuclear cells from a normally sterile site (Dunbar & Clarridge, 2000; Clarridge & Spiegel, 2003). Most strains of *E. rhusiopathiae* are highly susceptible to penicillins, cephalosporins, erythromycin and clindamycin (Reboli & Farrar, 1989). In this case, it is suggested that intravenous ceftriaxone treatment during ventriculo-peritoneal shunt insertion was effective.

To conclude, although systemic *E. rhusiopathiae* infection is rare and intracranial manifestation in particular is extremely rare, we could consider the organism as one of the possible causes of intracerebral infection, including meningitis.

**References**


