Case Report

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Chronic meningitis caused by *Erysipelothrix rhusiopathiae*

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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 erysipieloid, 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 septicaemia 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

Abbreviation: CSF, cerebrospinal fluid.

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 anti-tuberculosis 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 risk of human infection with *E. rhusiopathiae* is known to be closely related to the opportunity for exposure to the organism, and many reported human cases have been related to 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 the causative organism. His general medical condition was fine before infection, and he did not have a history of chronic alcohol consumption. Underlying diseases such as renal failure and malignancies including acute/chronic leukaemia are commonly associated with *E. rhusiopathiae* infection, and chronic alcohol ingestion is accepted as the most common underlying medical condition in systemic infection (Schuster *et al.*, 1993; Ko *et al.*, 2003). Although there are a few case reports of infected patients who were previously healthy (Vallianatos *et al.*, 2003; Beristain *et al.*, 2002), this case still raises questions about the contributing factor of *E. rhusiopathiae* intracranial infection.

There is a very old report of *Erysipelothrix* meningitis. The organism was detected in CSF from a 24-year-old Italian soldier, who manifested with high fever and altered mentality (Dumont & Cotoni, 1921). Interestingly, after 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 bacteraemia.

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

- Artz, A. L., Szabo, S., Zabel, L. T. & Hoffmeister, H. M. (2001). Aortic valve endocarditis with paravalvular abscesses caused by *Erysipelothrix rhusiopathiae*. *Eur J Clin Microbiol Infect Dis* **20**, 587–588.
- Beristain, X., Lopez-Lopategui, M. C., Bidaguren, J., Fernandez, C. & Idigoras, P. (2002). *Erysipelothrix rhusiopathiae* bacteremia without endocarditis or evidence of the entry point in an immunocompetent woman. *Enferm Infecc Microbiol Clin* **20**, 137–138.
- Brooke, C. J. & Riley, T. V. (1999). *Erysipelothrix rhusiopathiae*: bacteriology, epidemiology and clinical manifestations of an occupational pathogen. *J Med Microbiol* **48**, 789–799.
- Clarridge, J. E. & Spiegel, C. A. (2003). *Erysipelothrix*. In *Manual of Clinical Microbiology*, 8th edn, pp. 467–468. Edited by P. R. Murray, E. J. Baron, J. H. Jorgensen, M. A. Pfaller & R. H. Tenover. Washington, DC: American Society for Microbiology.
- Dumont, J. A. & Cotoni, L. (1921). Bacille semblable au bacille du rouge du porc rencontré dans le liquide céphalo-rachidien d'un méningitique. *Ann Inst Pasteur (Paris)* **35**, 625–633.
- Dunbar, S. A. & Clarridge, J. E. (2000). Potential errors in recognition of *Erysipelothrix rhusiopathiae*. *J Clin Microbiol* **38**, 1302–1304.
- Ko, S. B., Kim, D. E., Kwon, H. M. & Roh, J. K. (2003). A case of brain infarction associated with *Erysipelothrix rhusiopathiae* endocarditis. *Arch Neurol* **60**, 434–436.
- Reboli, A. C. & Farrar, W. E. (1989). *Erysipelothrix rhusiopathiae*: an occupational pathogen. *Clin Microbiol Rev* **2**, 354–359.
- Schuster, M. G., Brennan, P. J. & Edelstein, P. (1993). Persistent bacteremia with *Erysipelothrix rhusiopathiae* in a hospitalized patient. *Clin Infect Dis* **17**, 783–784.
- Silberstein, E. B. (1965). *Erysipelothrix* endocarditis: report of a case with cerebral manifestations. *JAMA* **191**, 862–864.
- Vallianatos, P. G., Tilentzoglou, A. C. & Koutsoukou, A. D. (2003). Septic arthritis caused by *Erysipelothrix rhusiopathiae* infection after arthroscopically assisted anterior cruciate ligament reconstruction. *Arthroscopy* **19**, E26.