Epidural Abscess Caused by *Streptobacillus moniliformis*

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We present an interesting case of a patient who developed an epidural abscess caused by *Streptobacillus moniliformis*. This is the first report in the medical literature of a spinal epidural abscess associated with this organism. Diagnosis of *S. moniliformis* infection requires a high degree of suspicion, and a delay may be inevitable when a relevant clinical history is lacking.

**CASE REPORT**

We present an interesting case from New Zealand of a patient who developed an epidural abscess caused by *Streptobacillus moniliformis* on a background of previous spinal surgery.

The patient, a 58-year-old male, presented with a 2-week history of right-sided flank pain, fevers, and lower limb weakness to the extent that he was unable to walk. He had a decreased sensation of bladder filling.

He had undergone spinal laminectomy for an L4/L5 radiculopathy 6 months prior to presentation. Other past medical history included hypertension, hypercholesterolemia, and gout. A month before his admission, the patient recalled sustaining a minor abrasion on the back of his hand, which was licked on several occasions by his dog. No history of contact with rats was elicited from the patient history. At that stage, there were no characteristic features from the original blood culture began to reveal a more characteristic Gram stain with a bulbous center, as shown in Fig. 2, and the organism was eventually presumptively identified after 21 days as *S. moniliformis*. This was subsequently confirmed by 16S rRNA molecular testing of the culture. Primers F27 (AGAGTTTGATCMTGGCTCAG) and 1541R (AAGGAGGTGATCCAGCCGGA) were used to amplify a 16S PCR product of approximately 1.5 kb. Comparison with sequences deposited in the EMBL-GenBank database showed that a 1,468-bp sequence shared 100% homology with the 16S rRNA sequence of the *S. moniliformis* type strain (9901; EMBL-GenBank accession no. CP001779).

At that stage, testing of the 16S rRNA gene directly from the epidural abscess sample was also requested and the DNA was extracted with a column extraction kit (Roche Diagnostics) according to the manufacturer’s instructions. A 528-bp section of the 16S rRNA gene was amplified with the AGAGTTTGATCMTGGCTCAG forward primer and the GWATTACCGGCCKGCTG reverse primer. The sequence was compared with those in the GenBank BLAST database and showed 99% homology with *S. moniliformis* DSM 12112 (accession number CP001779.1), thus confirming the presence of *S. moniliformis* in both the epidural abscess and the blood culture.

The organism gave a penicillin MIC of 0.012 mg/liter and a ceftriaxone MIC of 0.006 mg/liter (Etest; bioMérieux).

Further history was sought from the patient in light of the updated identification. On retrospective questioning, there was no clear history of a rat bite, although the patient did recall being woken by a “bite” some months prior to his presentation. Also of note was that numerous wild rats had been caught and poisoned on his property within the previous year.

The patient received a total of 5 weeks of i.v. ceftriaxone. During that time, he improved clinically and regained his mobility. Repeat MRI scanning after treatment cessation showed complete resolution of the epidural abscess.

*S. moniliformis* is a rare but well-recognized cause of infection following exposure to rats, either directly or via a contaminated...
environment. Classic rat bite fever is a systemic illness generally characterized by fever, rash, and polyarthralgias. To our knowledge, this is the first report in the medical literature of a spinal epidural abscess associated with this organism.

*S. moniliformis* is a fastidious Gram-negative bacillus with a highly pleomorphic appearance on Gram staining. Culture requires microaerophilic conditions with the addition of blood or serum to the growth medium. Growth may be inhibited by the concentrations of sodium polyanethol sulfonate found in many commercial blood culture bottles as an anticoagulant (2). Various studies have demonstrated carriage rates of the organism in the nasopharynxes of both domestic and feral rats of up to 100% (4). Infections typically follow a bite but have also been associated with the ingestion of contaminated food or water. Illness acquired by this route has been termed Haverhill fever after the location of the first recorded outbreak in 1926 (5). A similar syndrome, termed sodoku and more commonly found in Asia, may also follow a rat bite but is caused by the spirochaete *Spirillum minus*.

The true incidence of *S. moniliformis* infection is unknown. Case reports originate from most parts of the world, but the actual number of infections is hard to determine because of the difficulty in culturing the organism and lack of a specific history of rat exposure. Rat bite fever is also generally not a notifiable illness. Rats appear to be the main reservoir of infection. Cases have been associated with contact with other animals such as dogs, but this is likely due to transient colonization of the animals in an environment where rats are common (6). Historically, cases have been associated with social deprivation and contact with wild rat populations, but there are an increasing number of reports of infections following bites from and close contact with domesticated animals (1). The first reported case of rat bite fever in New Zealand was in 1919, and there have been only sporadic cases documented since then (7). While contact with feral rats has likely diminished, the increasing popularity of rats as pets may well lead to a rise in the number of infections due to this organism.

Delays in diagnosis often occur because of the organism’s fastidious growth requirements and the lack of specific exposure to rats in the clinical history. In our case, although the organism was successfully isolated from blood cultures taken on admission, the isolate did not initially demonstrate the classic pleomorphic appearance on Gram staining. The patient’s history of exposure to rats was gained only retrospectively in this particular case. Fortunately, empirical therapy was sufficient to cover the eventual diagnosis of *S. moniliformis* made by 16S rRNA gene sequencing. In the future, with the advent of rapid identification techniques such as matrix-assisted laser desorption ionization–time of flight (MALDI-TOF) mass spectrometry, it may be possible to expedite the identification of colonies of fastidious organisms such as *S. moniliformis*. However, at the time this was written, none of the main commercial MALDI-TOF databases contained spectrophotometric data for...
this organism. Other technologies that allow rapid identification of bacteria directly in body fluids are developing rapidly (3). In the near future, it may be possible to use spectrophotometric methods with or without prior PCR to allow the rapid, sensitive, and cost-effective identification of fastidious bacteria directly in body fluids in a routine diagnostic laboratory.

Recognized complications of S. moniliformis infections include endocarditis, meningitis, pneumonia, and focal abscesses. The mortality rate of untreated cases is estimated to be around 10%, though it may be as high as 53% in cases of endocarditis (2). S. moniliformis is almost always susceptible to penicillin, and there is only one historical report of penicillin resistance in the literature (8). Ceftriaxone, erythromycin, clindamycin, and tetracycline, among other drugs, have also been shown to be effective. Ceftriaxone was continued in this patient’s case for logistical reasons.

Diagnosis of S. moniliformis infection requires a high degree of suspicion, and a delay may be inevitable when a relevant clinical history is lacking. Gram stain morphology may not always be characteristic or recognized by staff unfamiliar with this rare organism. Rat bite fever may consequently be under-reported because of the difficulties associated with culturing the causative organism and its usual response to empirical treatment.

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REFERENCES