Epidural Abscess caused by *Streptobacillus moniliformis*.

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**Abstract**

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 delay may be inevitable when relevant clinical history is lacking.

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 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...
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 admission history. On examination, he was febrile. There was marked weakness of muscles innervated by L5 and S1 on both sides. Anal tone was absent.

An urgent MRI demonstrated the presence of a large epidural abscess anterior to L4, L5 and S1 vertebrae, compressing the thecal sac. There was also evidence of osteomyelitic involvement of these vertebrae along with septic arthritis of the facet joints between L2 and S1, figure 1.

The patient was placed on empirical IV flucloxacillin and an urgent surgical drainage of the epidural abscess was performed. The abscess fluid was negative for bacterial culture after 10 days incubation on sheep blood agar, chocolate agar (CO2 incubation at 37°C) and Fastidious Anaerobic Agar (anaerobic incubation at 37°C). An aliquot of the fluid was added to an aerobic blood culture bottle (Becton-Dickinson Bactec FX) and was negative after 10 days incubation. The aerobic bottle from a blood culture set (Becton-Dickinson Bactec FX, paired aerobic and anaerobic bottles) taken on admission was positive after 2 days with slender Gram negative bacilli seen on the Gram stain. The blood culture was sub-cultured on to sheep blood agar, chocolate agar (CO2 incubation at 37°C) and Fastidious Anaerobic Agar (anaerobic incubation at 37°C). Pinpoint cream coloured colonies grew on the blood agar and Fastidious Anaerobic Agar after a further 3 days incubation and the possibility of a \textit{Capnocytophaga} species was suggested, which was also consistent with the close contact with dogs in the patient history. At this stage there were no characteristic features in the
Gram stain suggestive of S. moniliformis, such as pleomorphism or a bulbous centre. The organism was oxidase negative and catalase negative. Further biochemical identification was not possible, largely because of its fastidious growth requirements.

The patient was commenced on IV ceftriaxone and oral ciprofloxacin because of the speculative diagnosis of Capnocytophaga infection. Gram stain of subsequent sub-cultures of the organism from the original blood culture began to reveal a more characteristic Gram stain with a bulbous centre, as shown in figure 2 and the organism was eventually presumptively identified after 21 days as a S. moniliformis. This was subsequently confirmed by 16s rRNA molecular testing of the culture. Primers F27 (AGAGTTTGATCMTGGCTCAG) and 1541R (AAGGAGGTGATCCAGCCGCA) were used to amplify a 16s PCR product of approximately 1.5kb. Comparison with sequences deposited in the EMBL-Bank database showed that a 1468bp sequence shared 100% homology with that of the 16s rRNA sequence from the S. moniliformis type strain (9901, EMBL-Bank accession CP001779).

At this stage 16s rRNA testing was also requested directly from the epidural abscess sample, the DNA being extracted using a column extraction kit (Roche Diagnostics, Switzerland) according to the manufacturer’s instructions. A 528bp section of the 16s rRNA gene was amplified using AGAGTTTGATCMTGGCTCAG forward primer and GWATTACCGGGCGGCTG reverse primer. The sequence was compared with those in the Genebank 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 0.012mg/l and a ceftriaxone MIC of 0.006mg/l (Etest, bioMerieux).

Further history from the patient was sought in light of the updated identification. On retrospective questioning, there was no clear history of a rat bite, although the patient did query 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 IV ceftriaxone. During this time, he clinically improved and regained his mobility. Repeat MRI scanning after treatment cessation showed complete resolution of the epidural abscess.

S. moniliformis is a rare, but well recognised cause of infection following exposure to rats, either directly or via a contaminated environment. Classic rat bite fever is a systemic illness generally characterised by fever, rash and polyarthritis. 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 stain. Culture requires microaerophilic conditions with the addition of blood or serum to the growth media. Growth may be inhibited by the concentrations of sodium
polynethol sulfonate found in many commercial blood culture bottles as an anticoagulant (1). Various studies have demonstrated carriage rates of the organism in the nasopharynx of both domestic and feral rats of up to 100% (3). Infections typically follow a bite but have also been associated with 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 (4). 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 due to the difficulty in culturing the organism and lack of 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 colonisation of the animal in an environment where rats are common (5). 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 and close contact with domesticated animals (6). The first reported case of rat bite fever in New Zealand was in 1919 and there have only been sporadic cases documented since then (7). Whilst 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 due to the 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 stain. The history of patient exposure to rats was only gained 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 Associated Laser Desorption and Ionisation-Time of Flight mass spectrometry (MALDI-TOF), it may be possible to expedite identification of colonies of fastidious organisms such as *S. moniliformis*. However, at the time of writing, none of the main commercial MALDI-TOF databases contains spectrophotometric data for this organism. Other technologies that allow rapid identification of bacteria directly from body fluids are developing rapidly (2). In the near future it may be possible to use spectrophotometric methods with or without prior PCR to allow rapid, sensitive and cost-effective diagnosis of fastidious bacteria directly from body fluids in a routine diagnostic laboratory.

Recognised complications of *S. moniliformis* infections include endocarditis, meningitis, pneumonia and focal abscesses. The mortality rate for untreated cases is estimated to be around 10% though may be as high as 53% in endocarditis (1). *S. moniliformis* is almost always susceptible to penicillin and there has been only one historical report of penicillin resistance reported in the literature (8). Ceftriaxone, erythromycin, clindamycin and tetracycline, amongst others, 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 delay may be inevitable when relevant clinical history is lacking. Gram stain morphology may not always be characteristic or recognised by staff unfamiliar with this rare organism. Rat bite fever may consequently be under-reported due to the difficulties associated with culturing the causative organism and its usual response to empiric treatment.

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**References**


Figure 1: MRI of patient demonstrating the large epidural abscess.
Figure 2: Gram stain from sub-culture of the organism, beginning to show the characteristic features of *Streptobacillus moniliformis* such as central bulbae and pleomorphism.