CASE REPORT

Spondylodiscitis Complicated by an Epidural Abscess and Meningitis Caused by *Bacteroides fragilis*

Takeshi Kawakami, Hiromichi Suzuki, Masatsune Suzuki and Yumi Hirose

**Abstract**

*Bacteroides fragilis* is a rare causative agent of spondylodiscitis. The pathophysiology of *B. fragilis* in spondylodiscitis remains largely unclear because of its rare occurrence. We herein report a case of spondylodiscitis complicated by an epidural abscess and meningitis; *B. fragilis* was detected in the blood of the patient. Moreover, the patient had a splenic abscess that was confirmed on magnetic resonance imaging. The patient completely recovered with antimicrobial therapy alone.

**Key words:** *Bacteroides fragilis*, spondylodiscitis, vertebral osteomyelitis

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**Introduction**

Spondylodiscitis is a common manifestation of osteomyelitis in adults (1). *Staphylococcus aureus* is the causative agent in approximately half of all cases, followed by coagulase-negative staphylococci, streptococci and *Enterobacteriaceae* (2-4). On the other hand, anaerobic bacteria rarely cause spondylodiscitis. Saeed et al. conducted a literature review of anaerobic spondylodiscitis and found only 33 reported cases of anaerobic spondylodiscitis (5).

In the present study, we describe a case of spondylodiscitis complicated by an epidural abscess and meningitis caused by *Bacteroides fragilis*, an obligatory anaerobic gram-negative bacillus.

**Case Report**

A 77-year-old, previously self-supporting, Japanese man was hospitalized due to pyrexia and shivering. The patient had experienced a cerebral infarction caused by atrial fibrillation one year previously and had been taking antihypertensive and anticoagulant agents since then. Six months prior to hospitalization, he developed intestinal strangulation, the cause of which was unknown, and underwent a partial resection at our facility. On admission, no remarkable physical findings, such as skin lesions, neck stiffness or tenderness on the back were apparent. The findings of blood tests revealed a normal leukocyte count (5,700/μL) and elevated serum C-reactive protein levels (14.4 mg/dL) (Fig. 1). Abdominal computed tomography (CT) showed a low attenuation area with mild ring enhancement in the spleen. The administration of ceftriaxone (2 g/day) did not resolve the pyrexia. On day 5 of hospitalization, the antimicrobial therapy was changed to ampicillin and sulbactam (8 g/day of ampicillin) because *B. fragilis* was identified on Gram stain and BD BBLCRYSTAL ANR (Nippon Becton Dickinson, Tokyo, Japan) of three sets of blood cultures obtained on admission. On day 7, the patient developed a disturbance of consciousness and neck stiffness. A cerebrospinal fluid analysis showed pleocytosis (mononuclear cells: 61/μL; polymorphonuclear cells: 53/μL), an increase in the total protein level (331 mg/dL) and a decrease in the glucose level (17 mg/dL). T2-weighted magnetic resonance imaging (MRI) of the spine showed high signals on the vertebrae (L4, L5 and S2), an intervertebral disc (L4/5) and in the epidural space posterior to the vertebrae, thus indicating the presence of spondylodiscitis and an epidural abscess (Fig. 2). The administration of meropenem (2 g for the first dose followed by 3 g/day) promptly improved the patient’s condition. Transthoracic echocardiography performed on day 8 showed the absence of cardiac vegetation. Diffusion-weighted MRI of the abdomen performed on day 9 confirmed the presence of a splenic abscess (Fig. 3). The results

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Correspondence to Dr. Hiromichi Suzuki, hsuzuki@tmch.or.jp
of a blood culture obtained on day 14 were negative for any bacteria. The antimicrobial therapy was switched to ampicillin with sulbactam (6 g/day of ampicillin) and metronidazole (1.5 g/day, orally) on day 21 and subsequently to amoxicillin with clavulanic acid, ceftriaxone, MEPM: meropenem, MNZ: metronidazole.

Figure 1. The patient’s clinical course. The closed squares, triangles and circles indicate maximum body temperatures, white blood cell counts (WBC) and the serum levels of C-reactive protein (CRP), respectively. ABPC/SBT: ampicillin with sulbactam, AMPC/CVA: amoxicillin with clavulanic acid, CTRX: ceftriaxone, MEPM: meropenem, MNZ: metronidazole.

Penicillin and cephalosporin are considered to be ineffective for the treatment of B. fragilis infection because B. fragilis produces beta-lactamase that inactivates these drugs (6). In addition, recent studies have shown that only 85%, 64% and 53% of B. fragilis strains are susceptible to cephaparin, clindamycin and moxifloxacin, respectively (7). Therefore, the antibiotics used empirically to treat serious infections that may involve B. fragilis are limited to penicillin combined with a beta-lactamase inhibitor, carbapenem and metronidazole (7). In the present case, the patient was initially treated with ceftriaxone; however, the isolated strain was resistant to this antibiotic. We believe that the inappropriate choice of antibiotics in this case may have exacerbated the infection. Moreover, the patient received ampicillin with sulbactam starting from day 5 after admission for two days; however, this treatment did not show any clinical effectiveness. The susceptibility result for B. fragilis was preserved (MIC ≤1/0.5 μg/mL) and found to be identical to most of the current B. fragilis strains (7). As ampicillin with sulbactam is known to penetrate bone tissue (8) and cerebrospinal fluid (9), it could be used as a therapeutic option for the treatment of meningitis. However, in this case, the patient’s altered mental status and the possibility that the meningitis was caused by other organisms prompted us to change the antibiotic therapy from ampicillin with sulbactam to meropenem. Therefore, it was difficult to judge the clinical effectiveness of ampicillin with sulbactam due to the short treatment period. Additionally, the antimicrobial dose of 8 g of ampicillin used in this study might not be sufficient to achieve therapeutic concentrations in the cerebrospinal fluid. Therefore, once the symptoms of meningitis had subsided, a surgical treatment was necessary.

Figure 2. T2-weighted magnetic resonance imaging of the vertebrae performed on day 10 showed L4, L5 and S2 spondylitis (arrowheads), L4/5 discitis (white arrow) and an epidural abscess (black arrow).

Figure 3. Diffusion-weighted magnetic resonance imaging of the abdomen performed on day 9 showed a splenic abscess (arrowhead).

Discussion

Penicillin and cephalosporin are considered to be ineffective for the treatment of B. fragilis infection because B. fragilis produces beta-lactamase that inactivates these drugs (6). In addition, recent studies have shown that only 85%, 64% and 53% of B. fragilis strains are susceptible to cephaparin, clindamycin and moxifloxacin, respectively (7). Therefore, the antibiotics used empirically to treat serious infections that may involve B. fragilis are limited to penicillin combined with a beta-lactamase inhibitor, carbapenem and metronidazole (7). In the present case, the patient was initially treated with ceftriaxone; however, the isolated strain was resistant to this antibiotic. We believe that the inappropriate choice of antibiotics in this case may have exacerbated the infection. Moreover, the patient received ampicillin with sulbactam starting from day 5 after admission for two days; however, this treatment did not show any clinical effectiveness. The susceptibility result for B. fragilis was preserved (MIC ≤1/0.5 μg/mL) and found to be identical to most of the current B. fragilis strains (7). As ampicillin with sulbactam is known to penetrate bone tissue (8) and cerebrospinal fluid (9), it could be used as a therapeutic option for the treatment of meningitis. However, in this case, the patient’s altered mental status and the possibility that the meningitis was caused by other organisms prompted us to change the antibiotic therapy from ampicillin with sulbactam to meropenem. Therefore, it was difficult to judge the clinical effectiveness of ampicillin with sulbactam due to the short treatment period. Additionally, the antimicrobial dose of 8 g of ampicillin used in this study might not be sufficient to achieve therapeutic concentrations in the cerebrospinal fluid. Therefore, once the symptoms of meningitis had subsided, a surgical treatment was necessary.
we reinitiated treatment with ampicillin and sulbactam to treat the splenic abscess and spondylodiscitis. Metronidazole was used concurrently as a precaution against failure to achieve a clinical response to ampicillin and sulbactam.

To the best of our knowledge, only 16 cases of B. fragilis spondylodiscitis have so far been reported (10-13). In these cases, the organism was thought to have spread hematogenously and contagiously in 10 (63%) and 3 (19%) cases, respectively. In the present case, we attempted to determine other sites of infection with MRI of the brain, CT of the trunk, echocardiography and gallium scintigraphy of the entire body. Consequently, the only lesion identified was the splenic abscess, which was ultimately diagnosed using abdominal MRI nine days after admission. There was a possibility that the splenic abscess had thus been the primary infective source; however, primary splenic abscesses are rare and splenic abscesses usually occur as a result of other primary infective sources (14). During the investigation, we performed only transthoracic echocardiography (TTE) and did not perform transesophageal echocardiography. Therefore, taking into consideration the spondylodiscitis and splenic abscess, the possibility of infective endocarditis could not be excluded, although no vegetation was observed on TTE. In addition, the patient had undergone a partial intestinal resection six months previously. We did not detect any intra-abdominal abscesses during the patient’s hospitalization; however, his clinical history might be associated with the infective cause, as B. fragilis is one of the most common bacteria found in the human intestines. Despite these possibilities, the precise mechanism of infection in this case remains unknown.

In the present case, the patient also developed an epidural abscess and meningitis, most likely as progressive manifestations of spondylodiscitis. Although we confirmed the presence of pleocytosis and an elevation of the protein concentration in the cerebrospinal fluid when the patient showed meningal symptoms, Gram staining did not reveal the presence of any organisms. Moreover, B. fragilis was not detected. There are several possibilities for this result. First, there could have been inappropriate management of the specimen, including prolonged exposure to room air until initiation of the anaerobic culture. Second, ampicillin with sulbactam, which was determined to be effective according to in vitro susceptibility testing, was administered starting from day 5 and may have influenced the culture results. Third, the epidural abscess induced the meningal symptoms. A previous report stated that pleocytosis and an elevation of the protein concentrations in the cerebrospinal fluid commonly occur in patients with epidural abscesses (15). Therefore, we believe that indirect inflammation in the present case because the pleocytosis was relatively mild.

A large part of the pathophysiology of B. fragilis spondylodiscitis remains unclear because of its rare occurrence. Publishing further such case reports will provide more clarity regarding the clinical significance of the disease, including associated risk factors and appropriate treatment.

The authors state that they have no Conflict of Interest (COI).

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References