Case Series – Headache

Campylobacter fetus Meningitis and Pyogenic Spondylodiscitis in a Healthy Young Woman

Kenta Ikeda^a Yasuhiro Manabe^a Shunya Fujiwara^a Yoshio Omote^a
Hisashi Narai^a Koji Abe^b

^aDepartment of Neurology, National Hospital Organization Okayama Medical Center, Okayama, Japan; ^bDepartment of Neurology, Graduate School of Medicine and Dentistry, Okayama University, Okayama, Japan

Keywords

Campylobacter fetus · Bacterial meningitis · Pyogenic spondylodiscitis · Healthy young female

Abstract

We report a rare case of *Campylobacter fetus* meningitis and pyogenic spondylodiscitis in a healthy young woman. A 35-year-old woman without significant medical history presented with fever, headache, and low back pain. *C. fetus* was detected from the blood culture. Cerebrospinal fluid analysis showed bacterial meningitis 2 days after onset. Although initial magnetic resonance imaging (MRI) did not reveal abnormal findings, repeated MRI showed a low-signal-intensity lesion on T1-weighted image (T1WI) and a high-signal-intensity lesion on T2WI between the L5 and S1 vertebral bodies 11 days after onset. The mode of infection was considered to be the consumption of raw chicken meat. After antibiotic treatment with 12 g/day ampicillin following 6 g/day meropenem, she was discharged 51 days after onset. As the inflammatory signs on MRI improved, oral antibiotic therapy was discontinued 85 days after onset. Although the initial MRI showed no abnormal findings, repeat MRI should be performed for patients who have persistent low back pain and fever.

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Introduction

Campylobacter fetus is a zoonotic pathogen that causes systemic infections in elderly or immunosuppressed people [1, 2]. Generally, bacterial meningitis is caused by *Streptococcus pneumoniae* or *Neisseria meningitidis*, and bacterial pyogenic spondylodiscitis is caused by *Strep-tococcus aureus* [2–8]. Bacterial meningitis and pyogenic spondylodiscitis are rarely caused by *C. fetus* [2–8]. Here, we report a rare case of *C. fetus* meningitis and pyogenic spondylodiscitis in a healthy young female.

Case Report

A 35-year-old woman without a significant medical history presented with fever, headache, and low back pain. Blood culture was collected, and she was treated with non-steroidal anti-inflammatory drugs. As the symptoms persisted, another medical check was performed at another hospital 2 days after onset. She was admitted to the hospital because the increased neutrophil count suggested bacterial meningitis. Cerebrospinal fluid (CSF) analysis showed a cell count of 472/μL (50.0% mononuclear cells), with an increased protein level of 52 mg/dL, and a normal glucose level of 59 mg/dL (blood glucose: 121 mg/dL). Lumbar magnetic resonance imaging (MRI) revealed no abnormal findings (Fig. 1a, b, arrows) 2 days after onset. At the beginning of her treatment, she received 4 g/day ceftriaxone and 2 g/day vancomycin. However, this was changed to 6 g/day meropenem, because *C. fetus* was detected from the blood culture. This treatment improved her fever and headache, but not right low back pain or right leg pain.

She was transferred to our hospital for continuation of antibiotic therapy and investigation into the cause of the persisting low back pain 11 days after onset. On admission, a physical examination revealed a fever of 37.1°C with a Glasgow Coma Scale score of 15 (E4V5M6). There was pain in the region from the right low back to the inside of the right thigh and tenderness in the low back. On neurological examination, her deep tendon reflexes were hypoactive in the right Achilles tendon without pathological reflexes. She had no stiff neck, but Kernig's signs were positive. At this point, further history taking revealed that she had consumed raw chicken 4 days before onset. Laboratory tests showed a white blood cell count of 7,300/μL and a C-reactive protein (CRP) level of 3.08 mg/dL. CSF analysis showed a cell count of 12/μL (97.2% mononuclear cells), with an increased protein level of 103 mg/dL and a normal glucose level of 41 mg/dL (blood glucose: 98 mg/dL). All CSF cultures, including those performed on her first admission, were negative. Lumbar MRI showed a low-signal-intensity lesion on T1-weighted image (T1WI) and a high-signal-intensity lesion on T2WI between the L5 and S1 vertebral bodies (Fig. 1c, d, arrows). Brain MRI did not show any parenchymal abnormalities. We diagnosed bacteremia of *C. fetus*, sequencing bacterial meningitis, and pyogenic spondylodiscitis. We de-escalated 6 g/day meropenem to 12 g/day ampicillin 26 days after onset. Her fever, low back pain, white blood cell count, and CRP gradually improved and became stable. However, the inflammatory signs on MRI worsened 35 days after onset (Fig. 1f, arrow); thus, antibiotic therapy was continued (oral 1.5 g/day ampicillin). She was discharged 51 days after onset and treated as an outpatient. Finally, the inflammatory signs on MRI improved (Fig. 1g, h, arrows), and antibiotic therapy was discontinued 85 days after onset.

Discussion

C. fetus, a zoonotic pathogen found in the intestinal tracts of cattle and sheep, is transmitted via enteral tracts either orally or by contact, resulting in systemic infection [1, 2]. *C. fetus* infection may develop by eating contaminated food, such as milk or beef, or direct contact with infectious animals [1, 2]. *C. fetus* has resistance to complement-mediated killing due to its surface layer proteins, which enable it to enter blood vessels from enteral tracts, causing bacteremia and systemic infections [1, 2]. Bacterial meningitis is rarely caused by *C. fetus* (0.02 per million people) [2], and bacterial pyogenic spondylodiscitis is even more rarely caused by *C. fetus* with only a few case reports [4–9]. In our case, *C. fetus* was detected only in the first blood culture. *C. fetus* usually infects a compromised host. Diabetes mellitus steroid therapy, malignancies, HIV infection, alcoholism, or aging are risk factors for the disease [1, 2]. There are only a few reports of *C. fetus* infection in healthy people [3]. In our case, oral infection was suspected because of the consumption of raw chicken. A combination of meningitis and pyogenic spondylodiscitis caused by *C. fetus* in a healthy young female has not been previously reported.

We used MRI to diagnose pyogenic spondylodiscitis. However, the high signal usually observed on T2WI is sometimes absent in the initial stage of symptoms [7]. In our case, no evident abnormal findings on T1 and T2WI were noted 2 days after onset. MRI was repeated 11 days after onset because the low back pain persisted. Repeat lumbar MRI showed a low-signal-intensity lesion on T1WI and a high-signal-intensity lesion on T2WI between the L5 and S1 vertebral bodies. We could diagnose these symptoms as pyogenic spondylodiscitis. Although she continued to display fever symptoms, her low back pain, white blood cell count, and CRP gradually improved. We decided to continue antibiotic therapy because the inflammatory signs on MRI worsened 35 days after onset. Antibiotic therapy was discontinued 85 days after onset because the inflammatory signs on MRI improved. MRI is useful for the evaluation of disease progression and judgement of the treatment. Although the initial MRI showed no abnormal findings, repeat MRI should be performed for patients with persistent low back pain and fever.

Statement of Ethics

The patient gave informed consent for the publication of this case report.

Disclosure Statement

The authors state that they have no conflicts of interest.

Funding Sources

The authors have nothing to disclose.

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Fig. 1. Lumber magnetic resonance imaging (MRI) on T1-weighted image (T1WI) and T2WI showing no abnormal findings 2 days after onset (**a, b**, arrows). Lumber MRI showing a low signal intensity on T1WI and a high-signal-intensity lesion on T2WI between the L5 and S1 vertebral bodies 11 days after onset (**c, d**, arrows). The inflammatory signs on MRI worsened 35 days after onset (**e, f**, arrows). The lesion disappeared 85 days after onset (**g, h**, arrows).