CASE REPORT

An unusual cause of spontaneous bacterial peritonitis due to *Campylobacter fetus* with alcoholic liver cirrhosis

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SUMMARY

A 40-year-old man with severe alcoholic liver cirrhosis with a 2-day history of fatigue and abdominal pain was admitted. He reported eating sushi and sliced raw chicken a few days previously. His abdomen was distended, with shifting dullness. Based on the patient's history, physical examination and the results of abdominocentesis, he was diagnosed as having spontaneous bacterial peritonitis; blood and ascitic fluid cultures were positive for *Campylobacter fetus*. The patient was started on treatment with cefotaxime, which was switched after 1 week to ampicillin for an additional 3 weeks. The patient was successfully treated with the 4-week course of intravenous antibiotic therapy.

BACKGROUND

Campylobacter fetus (*C fetus*) is a slender, curved microaerophilic Gram-negative bacillus. *C fetus* is well known to cause blood stream infection in patients with underlying diseases such as cancer, liver disease and diabetes^{1 2}; however, it is an unusual causative bacterium for spontaneous bacterial peritonitis (SBP). In this article, we report a case of SBP caused by *C fetus* in a patient with severe alcoholic liver cirrhosis.

CASE PRESENTATION

A 40-year-old Japanese man with severe alcoholic liver cirrhosis (Child–Pugh: grade C) was admitted to our hospital with a 2-day history of fatigue, abdominal pain and slight diarrhoea. He reported eating sushi and sliced raw chicken a few days back. His medical history revealed that he had been suffering from alcoholic cirrhosis and oesophageal varices. His daily medications included furosemide, spironolactone and ursodeoxycholic acid. He did not smoke cigarettes or indulge in illicit drug use, but drank heavily. He denied any history of allergies.

INVESTIGATIONS

Upon physical examination, the patient appeared ill; his blood pressure was 105/42 mm Hg, pulse was 130 beats/min and regular, temperature was 37.3 °C and the respiratory rate was 24 breaths/min, with a peripheral arterial oxygen saturation of 96% in room air. His Glasgow Coma Scale score was evaluated as E4, V5 and M6. His abdomen was soft, but distended, with shifting dullness; the bowel sounds were hyperactive. He complained of slight abdominal pain in the upper quadrants of both sides. Liver knock pain and Murphy's sign

were negative. Rectal examination revealed no mass or tenderness and a scant amount of brown stool that was positive for occult blood. Neurological examination was unremarkable.

Laboratory examination revealed a white blood cell (WBC) count of 13 100/μl with 97% neutrophils. The haemoglobin was 9.9 mg/dl, with a mean corpuscular volume of 109.8 fl. The platelet count was 50 000/μl, the prothrombin time and international normalized ratio (PT–INR) was 2 s, and the activated partial thromboplastin time (APTT) was 57.7 s. The results of the analysis of the blood chemistry were as follows: blood urea nitrogen 24.3 mg/dl, serum creatinine 1 mg/dl, serum albumin 2.4 g/dl, serum total protein 6.2 g/dl, serum aspartate transaminase 126 IU/l, serum alanine transaminase 38 IU/l, serum total bilirubin 5 mg/dl, serum direct bilirubin 3.7 mg/dl, serum lactate dehydrogenase 204 IU/l and serum C reactive protein 10 mg/dl. Abdominal CT showed significant ascites, with no evidence of perforation of the gastrointestinal tract. Paracentesis was performed and analysis of the ascitic fluid specimen revealed a WBC count of 4150/μl with 84% neutrophils and a protein level of 1.4 g/dl. Gram staining of the ascitic fluid revealed polymorphonuclear leucocytes, but no evidence of any bacteria was found.

TREATMENT

Specimens for blood culture, ascitic fluid culture and stool culture were taken, and the patient was started on treatment with cefotaxime 2 g administered every 8 h, based on a presumptive diagnosis of spontaneous bacterial peritonitis, pending culture results.

Figure 1 Gram staining of the blood culture isolate shows spiral Gram-negative and spiral bacilli.

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OUTCOME AND FOLLOW-UP

On day 3, the blood culture and ascitic fluid culture turned out to be positive, and faintly staining slender, spiral, Gram-negative bacilli were isolated (figure 1), which were identified as *C fetus* by the BacT/Alert 3D system (Sysmex bioMérieux, Tokyo, Japan). Samples were then subcultured onto 5% sheep blood agar and chocolate agar, and the organism was identified as *C fetus* based on the morphology on wet preparations, a positive catalase test, resistance to nalidixic acid and susceptibility to cephalothin and inability to grow at 42 °C. The disk sensitivity test results using the breakpoints recommended by the CLSI for *Campylobacter* spp revealed that the isolates of *C fetus* were susceptible to ampicillin, ceftriaxone, erythromycin, ciprofloxacin, gentamicin and imipenem, but resistant to minocycline. On day 4, the follow-up blood culture was negative and the stool culture grew only normal flora, and the patient showed improvement of the clinical symptoms. On day 7, a follow-up paracentesis was performed and the analysis of the ascitic fluid specimen revealed a WBC count of 525/μl, with 66% neutrophils and a protein level of 1.1 g/dl, and the ascitic fluid culture was negative. On day 8, the antimicrobial therapy was changed to ampicillin 2 g administered every 6 h for an additional 21 days. The patient was successfully treated with a 4-week course of intravenous antibiotics without any side effects; no evidence of relapse of the infection was noted at the 1-year follow-up.

DISCUSSION

SBP caused by *C fetus* is rare. The first case report of SBP caused by *C fetus* was presented in 1976 by Targan *et al.*³ Since then, five cases have been reported in the literature.⁴ Three of the five patients had concomitant peritonitis and bacteraemia, like the present case. Patients with Budd–Chiari syndrome, end-stage renal disease and continuous ambulatory peritoneal dialysis can develop peritonitis owing to *C fetus*.^{1 5 6} The main underlying conditions reported are liver disease (34%), including alcohol abuse (27%), cirrhosis (20%), cancer (33%), diabetes (21%) and chemotherapy (19%).¹ In the present case, the patient was at a high risk for *C fetus* infection as he had severe alcoholic liver cirrhosis and had eaten sushi and raw chicken 5 days prior to admission.

Although current guidelines suggest the use of a third-generation cephalosporin, such as cefotaxime, for empiric treatment of SBP,⁷ it is noteworthy that the susceptibility of the infection to cefotaxime was not so high. A susceptibility rate to cefotaxime of 62% was reported from a study in France.² Another study reported a lower bactericidal activity of cefotaxime as compared to that of amoxicillin or imipenem, and also cases of treatment failure with cefotaxime.⁸ Empirical treatment

with imipenem has been reported to be associated with good outcomes in cases of *C fetus* bacteraemia.² With respect to the optimal duration of treatment, a minimum of 3–4 weeks of intravenous antimicrobial therapy is recommended, especially for systemic infection.^{1 2 9}

In conclusion, we report a case of SBP caused by *C fetus*. SBP owing to *Campylobacter* spp mainly occurs in patients with severe underlying alcoholic liver cirrhosis, and blood culture is also often positive. Further studies are needed to describe the typical characteristics of this infection.

Learning points

- ▶ *Campylobacter fetus* can be a causative pathogen of spontaneous bacterial peritonitis among immunocompromised patients, especially those with underlying alcoholic liver cirrhosis.
- ▶ A history of eating raw chicken or sushi should urge ruling out the possibility of campylobacter infection.
- ▶ Third-generation cephalosporins, such as cefotaxime, may be ineffective for the treatment of *C fetus* infection.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Pacanowski J, Lalande V, Lacombe K, *et al.* *Campylobacter* bacteremia: clinical features and factors associated with fatal outcome. *Clin Infect Dis* 2008;47:790–6.
- 2 Gazonne L, Legrand P, Renaud B, *et al.* *Campylobacter fetus* bloodstream infection: risk factors and clinical features. *Eur J Clin Microbiol Infect Dis* 2008;27:185–9.
- 3 Targan SR, Chow AW, Guze LB. Spontaneous peritonitis of cirrhosis due to *Campylobacter fetus*. *Gastroenterology* 1976;71:311–13.
- 4 Calonge Raventos L, Len Abad O, Bartolomé R, *et al.* Spontaneous peritonitis induced by *Campylobacter* spp in patients with liver cirrhosis. Report of 2 cases and review of the literature. *An Med Interna* 2002;19:23–6.
- 5 Barrio J, Castiella A, Gil I, *et al.* Spontaneous bacterial peritonitis by *Campylobacter fetus* in Budd–Chiari syndrome without liver cirrhosis. *Liver* 1999;19:69–70.
- 6 Lee YC, Huang YT, Sheng WH, *et al.* Simultaneous peritoneal dialysis-associated peritonitis and bacteremia due to ceftriaxone-resistant *Campylobacter fetus*. *Perit Dial Int* 2011;31:366–8.
- 7 Pigraud C, Bartolomé R, Almirante B, *et al.* Bacteremia due to *Campylobacter* species: clinical findings and antimicrobial susceptibility patterns. *Clin Infect Dis* 1997;25:1414–20.
- 8 Francioli P, Herzstein J, Grob JP, *et al.* *Campylobacter fetus* subspecies *fetus* bacteremia. *Arch Intern Med* 1985;145:289–92.
- 9 Pigraud C, Bartolomé R, Almirante B, *et al.* Bacteremia due to *Campylobacter* species: clinical findings and antimicrobial susceptibility patterns. *Clin Infect Dis* 1997;25:1414–20.

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