CASE REPORT

Septic thrombophlebitis of the superior mesenteric vein with bacteraemia caused by *Bacteroides fragilis* and *Streptococcus intermedius* as a complication of diverticulitis

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SUMMARY

A 68-year-old Japanese man with alcoholic liver cirrhosis was admitted to our hospital because of fever and haematemesis. On day 3, his blood culture became positive for Gram-negative bacilli and Gram-positive cocci, and contrast-enhanced abdominal CT revealed acute septic thrombophlebitis of the superior mesenteric vein with caecal diverticulitis. Antimicrobial therapy with ampicillin–sulbactam and anticoagulant therapy were started and the blood culture grew *Bacteroides fragilis* and *Streptococcus intermedius*. On hospital day 7, the patient's condition began to improve in response to the therapy, therefore, the ampicillin–sulbactam and anticoagulant therapy was continued for 42 days. The patient was discharged home on hospital day 45. *B. fragilis* bacteraemia of unknown source should caution the physician to search for an intra-abdominal focus, such as thrombosis of the portal vein or mesenteric vein.

BACKGROUND

Septic thrombophlebitis (pyephebitis) of the superior mesenteric vein is a rare complication, and is often associated with intra-abdominal diseases such as diverticulitis, appendicitis, inflammatory bowel disease, etc.¹ Early clinical diagnosis of septic thrombophlebitis is sometimes difficult because of the non-specific symptoms, and depends on a high index of clinical suspicion, and appropriate use of imaging studies.² In this article, we report the case of a patient with septic thrombophlebitis of the superior mesenteric vein with bacteraemia caused by *Bacteroides fragilis* (*B. fragilis*) and *Streptococcus intermedius* (*S. intermedius*) as a complication of diverticulitis, who was successfully treated with antibiotic and anticoagulant drugs.

CASE PRESENTATION

A 68-year-old Japanese man with alcoholic liver cirrhosis (Child-Pugh class A) and hypertension was admitted to our hospital because of fever and haematemesis. Two days prior to admission, he developed fever, chills and generalised fatigue, and acetaminophen had been prescribed for some kind of presumed viral infection; however, no response to this treatment was observed, and the patient's complaints of fever and generalised fatigue persisted. Then, the patient developed acute haematemesis and was brought to our emergency department. An emergency endoscopic examination

revealed the presence of a haemorrhagic gastric ulcer. The patient was admitted to our hospital after haemostasis was accomplished by an endoscopic procedure. In regard to the treatment history, the patient was under regular treatment with an ACE inhibitor. He did not smoke tobacco or use illicit drugs, but drank 1000 ml of beer per day. He denied any history of allergies.

INVESTIGATIONS

Examination of the vital signs revealed the following: blood pressure 118/66 mm Hg, pulse 110 bpm, temperature 38.2°C, respiratory rate 32/min and peripheral arterial oxygen saturation 96% while breathing ambient room air. Physical examination revealed no abnormalities of the eyes, nose, oral cavity or throat. The lungs and heart were normal. The abdomen was soft, with no tenderness, no hepatosplenomegaly and normal bowel sounds. Liver knock pain was negative. Rectal examination revealed no masses or tenderness, and only a scant amount of brown stool was positive for occult blood. Neurogenic examination was unremarkable.

Laboratory data obtained at admission revealed a white blood cell count of 19 900/μl with 90% neutrophils. Haemoglobin was 9.9 mg/dl, with a mean corpuscular volume of 96.8 fl. The platelet count was 165 000/μl. Examination of the blood biochemical parameters revealed the following: serum sodium 136 mEq/l, potassium 4.5 mEq/l, chloride 100 mEq/l, blood urea nitrogen 66.1 mg/dl, serum creatinine 1.2 mg/dl, blood glucose 74 mg/dl, serum albumin 3.2 g/dl, serum total protein 6 g/dl, serum aspartate transaminase 962 IU/l, serum alanine aminotransferase 294 IU/l, serum total bilirubin 1.4 mg/dl, serum lactate dehydrogenase 770 IU/l and serum C reactive protein 32.8 mg/dl. The serum tests for hepatitis B antigen and hepatitis C antibody were negative. Chest radiography revealed no evidence of pneumonia and no cardiomegaly, and abdominal ultrasound examination revealed mild hepatosplenomegaly, but no evidence of infectious origin.

TREATMENT

On hospital day 1, the patient was started on an empiric therapy with cefotaxime 2 g administered every 8 h. On day 3, his blood culture became positive for Gram-negative bacilli and Gram-positive

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Figure 1 Contrast-enhanced CT scan showing findings suggestive of caecal diverticulitis.

cocci, and contrast-enhanced abdominal CT revealed findings suggestive of caecal diverticulitis (figure 1) and evidence of acute septic thrombophlebitis of the superior mesenteric vein; a thrombus was visible in the lumen of the vein, along with perivascular inflammatory changes (figures 2 and 3). From day 3, treatment was started with ampicillin–sulbactam and anticoagulant drugs after confirming haemostasis in the region of the gastric ulcer. On day 4, the blood culture grew *B fragilis* and *S intermedius*, and a second follow-up blood culture on day 5 was negative. Serological examination for the detection of any concomitant medical condition predisposing to septic thrombophlebitis revealed negative test results for protein C, protein S, anticardiolipin antibody and lupus anticoagulant.

OUTCOME AND FOLLOW-UP

On hospital day 7, the patient's condition began to improve in response to the antibiotic and anticoagulant therapy, therefore, the ampicillin–sulbactam and anticoagulant drug therapy was continued for 42 days. The patient was discharged home on hospital day 45.

Figure 2 Sagittal view of thrombosis in the superior mesenteric vein.

Figure 3 Coronal view of thrombosis in the superior mesenteric vein.

DISCUSSION

Septic thrombophlebitis of the portal vein and mesenteric vein is termed pylephlebitis, and it is often associated with an intra-abdominal inflammatory process. The most common symptoms are fever, which occurs in 86–100% of the cases, and abdominal pain occurring in 74–82% of cases^{1–3}; however, clinical diagnosis is difficult because the clinical features are non-specific, especially in cases without abdominal pain.⁴ Physical examination often reveals splenomegaly, hepatomegaly and ascites, however, the features of each of these were non-specific. Regarding the aetiology, the most common underlying diseases associated with septic thrombophlebitis are diverticulitis (30%), appendicitis (19%) and inflammatory bowel disease (6%); other aetiologies include pancreatitis (5%), gastric/duodenal ulcer (2%) (including the present case), etc.¹ Liver cirrhosis and hepatocellular carcinoma are also well-known risk factors for septic thrombophlebitis.⁴ In the present case, the diagnosis was made by radiographic imaging and not by the clinical findings. In fact, ultrasonography and CT play important roles in establishing the diagnosis of septic thrombophlebitis. Regarding the site of involvement, according to a report, thrombosis of the superior mesenteric vein was detected in 42%, of the portal vein in 39%, and of the inferior mesenteric vein in 2% of the cases.¹ With respect to the causative organism, the common isolates are *B fragilis*, followed by *Escherichia coli* and *Streptococcus* spp., with the infection being polymicrobial in 44% of the patients (including the present case).^{1–3} *B fragilis* is the most common anaerobic blood isolate; Liappis *et al*⁵ reported that *B fragilis* bacteraemia is associated with portal and mesenteric venous thromboses because of the transient production of anticardiolipin

antibodies by this organism. Ni *et al*⁶ suggested that *B fragilis* bacteraemia of unknown source should caution the physician to search for an intra-abdominal focus, such as thrombosis of the portal or mesenteric veins. It is important for these patients to be treated initially with appropriate broad-spectrum antibiotics and anticoagulant drugs. Patients treated with anticoagulant drugs showed a better outcome as compared to those treated with antibiotics alone (death rate 5.7% vs 22.2%).¹ In the present case, anticoagulation was deemed to be contraindicated owing to the presence of gastric ulcer; however, we administered anticoagulant therapy after confirming haemostasis in the region of the gastric ulcer by endoscopy.

In conclusion, we report a case of septic thrombophlebitis of the superior mesenteric vein with bacteraemia caused by *B fragilis* and *S intermedius* occurring as a complication of caecal diverticulitis. It is important to keep in mind the possibility of septic thrombophlebitis associated with *B fragilis* bacteraemia as a rare complication of diverticulitis.

Learning points

- Diverticulitis can be associated with septic thrombophlebitis as a rare complication.
- *Bacteroides fragilis* is associated with portal and mesenteric venous thromboses, because of its transiently eliciting the production of anticardiolipin antibodies.
- It is important to treat these patients initially with appropriate broad-spectrum antibiotics and anticoagulant drugs to obtain improved outcomes.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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