A cirrhotic patient with fever and abdominal pain in the presence of ascites

Vincent Wai-Sun Wong, Henry Lik-Yuen Chan

INTRODUCTION

Among patients with liver cirrhosis, infection is a common cause of morbidity and mortality. Spontaneous bacterial peritonitis, chest infection and urinary tract infection are the commonest infections in these patients. Secondary bacterial peritonitis, defined as infection of the ascitic fluid with a contagious source of sepsis, accounts for 3% of all infections (Fernandez et al, 2002). The contagious source is usually a perforated viscus or intra-abdominal abscess.

The diagnosis of secondary bacterial peritonitis is often difficult as the ascitic fluid separates the parietal peritoneum from the viscus and masks any peritonism. On the other hand, early diagnosis of this condition is important because mortality is high if timely surgical intervention is not performed.

DISCUSSION

Diagnostic abdominal paracentesis must be performed early in patients with ascites who present with fever. Even in cases of perforated viscus, delayed paracentesis results may be misleading. For example, Runyon and Hoefs (1984) proposed the use of three parameters (ascitic fluid total protein >1 g/dl, glucose <50 mg/dl, and lactate dehydrogenase greater than the upper limit of normal for serum) to differentiate between spontaneous bacterial peritonitis and gut perforation. Although subsequent studies confirmed the high sensitivity of these criteria, the ascitic fluid neutrophil counts of two-thirds of the patients dropped after antibiotic treatment for 2 days (Akriviadis and Runyon, 1990). In the present case, the ascitic fluid cell count became normal despite presence of ruptured viscus 6 days after the commencement of antibiotics.

Another study in Taiwan reported the use of ascitic fluid carcinoembryonic antigen and alkaline phosphatase levels as a diagnostic tool for secondary peritonitis in cirrhotic patients (Wu et al, 2001). Thirty-eight patients with secondary peritonitis were studied and 34 patients with spontaneous bacterial peritonitis were recruited as controls. These criteria were found to have 92% sensitivity and 88% specificity. Further studies are required to validate these criteria.

CONCLUSION

A high index of suspicion of secondary bacterial peritonitis among cirrhotic patients with ascites is important. The symptoms and physical signs are often non-specific. Early diagnostic paracentesis, appropriate radiological examination and revision of response to antibiotics are the keys to the management of this condition. HM

CASE REPORT

The patient was a 64-year-old woman suffering from diabetes mellitus and Child's grade B hepatitis B virus-related liver cirrhosis. In November 2001, she complained of persistent dull right upper quadrant pain and fever for 2 days. She did not notice any jaundice, tea-coloured urine or pale stool. There was no increase in abdominal distension.

On admission, her oral temperature was 37.4° C. She had moderate ascites with no sign of peritonism. Otherwise she was haemodynamically stable and had no hepatic encephalopathy.

Liver biochemistry revealed albumin 29 g/litre, total bilirubin 25 mmol/litre, alkaline phosphatase 190 IU/litre and alanine aminotransferase 34 IU/litre. Haemoglobin level was 9.7 g/dl, platelet count was 26 x 10^9/litre, white cell count was 7.9 x 10^9/litre and prothrombin time was 18.6 seconds. Chest X-ray was clear.

She was given intravenous ampicillin, cefuroxime and metronidazole. Urgent abdominal ultrasound showed mildly cirrhotic liver and small amount of ascites. The gallbladder wall was thickened and distended, but there was no biliary dilatation, pericholecystic fluid or ultrasonic Murphy's sign. Endoscopic retrograde cholangiopancreatogram did not reveal any ductal dilatation or intraductal lesion.

Her fever subsided after 6 days of antibiotic treatment. Nevertheless, she still had right upper quadrant pain. Prothrombin time was prolonged to 20.3 seconds, and her platelet count dropped to 10 x 10^9/litre. Diagnostic paracentesis showed ascitic fluid polymorph count 34/ml, total protein less than 10 g/litre and negative culture.

A follow-up ultrasound 2 days later showed 7 cm subhepatic collection filled with sludge. The diagnosis was acute cholecystitis with perforation. Percutaneous drainage of the collection was performed. She remained afebrile and was discharged after 1 week. An elective laparoscopic cholecystectomy was performed 2 months later.

Akriviadis EA, Runyon BA (1990) Utility of an algorithm in differentiating spontaneous from secondary bacterial peritonitis. *Gastroenterology* **98:** 127–33

Fernandez J, Navasa M, Gomez J, Colmenero J, Vila J, Arroyo V, Rodes J (2002) Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. *Hepatology* **35:** 140–8

Runyon BA, Hoefs JC (1984) Ascitic fluid analysis in the differentiation of spontaneous bacterial peritonitis from gastrointestinal tract perforation into ascitic fluid. *Hepatology* **4:** 447–50

Wu SS, Lin OS, Chen YY, Hwang KL, Soon MS, Keeffe EB (2001) Ascitic fluid carcinoembryonic antigen and alkaline phosphatase levels for the differentiation of primary from secondary bacterial peritonitis with intestinal perforation. *J Hepatol* **34:** 215–21

Dr Vincent Wai-Sun Wong is Medical Offier and **Dr Henry Lik-Yuen Chan** is Associate Professor in the Department of Medicine and Therapeutics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong

Correspondence to: Dr HLY Chan