Spontaneous Bacterial Peritonitis in the Western Region of Saudi Arabia

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We studied 14 patients with spontaneous bacterial peritonitis among 147 patients with chronic liver disease (liver cirrhosis, liver carcinoma) admitted during a 3-year period. The common clinical presentations in these patients were abdominal pain, hepatic encephalopathy, fever and decreased bowel sounds. The commonest organism in ascitic fluid culture was Escherichia coli. Two patients had recurrent peritonitis. Despite antibiotic therapy, 11 of these patients died in hospital due to advanced liver disease, progressive renal failure, septicemia and gastrointestinal bleeding.

Spontaneous bacterial peritonitis (SBP) is a clinical condition in which ascitic fluid is infected without any apparent evidence of sepsis in the abdominal cavity. Conn & Fessel\(^1\) first described this condition in ascitic patients with liver cirrhosis. The prevalence of SBP is between 10 and 25% in hospitalized patients\(^2\) in Western studies. In Saudi Arabia chronic liver disease is due predominantly to hepatitis B and C infection.\(^3\)\(^-\)\(^5\) We did not come across any published study of SBP in Saudi Arabia. We studied liver cirrhotic patients who were admitted to the National Guard King Khalid Hospital, Jeddah, over a 3-year period (between February 1989 and February 1992) for SBP. The aim of the study was to assess the prevalence of SBP in hospitalized patients, the clinical pattern and outcome.

Materials and Methods

The National Guard King Khalid Hospital is a tertiary care hospital in the city of Jeddah which has a population of about 2 million (estimated) and this hospital caters for the general public and Saudi National Guardsmen and their dependants.

All patients with liver cirrhosis and liver carcinoma who were admitted between February 1989 and February 1992 were studied retrospectively. We reviewed the records of those patients who deteriorated clinically e.g. refractory ascites, progressive renal failure and gastrointestinal bleeding, mental confusion, and unexplained febrile episodes. In patients who had clinical evidence of ascites, 20 ml fluid was aspirated and the following were recorded on admission:

1. Colour of ascitic fluid
2. White cell count (polymorphonuclear leucocytes) in ascitic fluid
3. Microbiological assessment e.g. Gram stain, Ziehl-Neelsen's stain, was performed. Also 5 ml of ascitic fluid was inoculated into a blood culture bottle at the bedside
4. Ascitic fluid protein estimation (in 11 patients)
5. Blood and urine cultures were taken
6. Peripheral blood count and biochemical profile including liver function tests.

We included in our study only those patients who fulfilled the following criteria:

1. Clinical features of liver cirrhosis and ascites
2. Positive ascitic fluid culture
3. Absence of obvious source of infection such as perforated viscus, abdominal surgery within the previous 4 weeks, intra-abdominal abscess.

Results

During the study period of 3 years we reviewed 147 patients with chronic liver disease (liver cirrhosis, liver carcinoma) with ascites and identified 14 patients with SBP. Among the 14 SBP patients there were nine male patients and five female patients. Twelve patients were Saudi nationals and the other two were expatriates (one Egyptian, one Sudanese). The age range was between 24 and 64 years (mean 45 years). Eleven patients had liver cirrhosis, two patients had primary liver carcinoma and one patient had a Budd-Chiari syndrome due to polycythaemia.

Among these 14 patients, nine patients’ sera were positive for HbsAg antigen, two patients had a past exposure to schistosomiasis and the remaining three patients’ serology for hepatitis B were negative. In these patients, hepatitis C antibody tests were not available. Eleven patients were classified in Child ‘C’ Category, two were in Category ‘B’ and one was in Category ‘A’.

Ascitic fluid in SBP patients was noted to be cloudy in six patients and straw coloured in eight patients. A Gram stain of ascitic fluid showed positive results in two patients only.

Ascitic fluid polymorphonuclear leucocyte count (PMN) between 250 and 1600 cells/cm³ were observed in 11 patients and in the other three patients white cell counts were between 150 and 250 cells/cm³ (PMN). Classically SBP is recognized if ascitic fluid white cell count is over 250 cells/cm³ with a positive ascitic fluid culture and absence of a primary source of infection. The variant of SBP, i.e. bacteriascites, is a well recognized phenomenon when ascitic fluid leucocyte count is less than 250 cells/cm³ but ascitic fluid culture is shown to be positive. Escherichia coli was grown in ten patients, the other four patients each had Pneumococcus, Serratia, Aeromonas hydrophila, and Streptococcus sanguis in the ascitic fluid.

We observed that in two patients where ascitic fluid leucocyte counts were >250 cells/cm³, but failed to grow any organism and these patients were excluded from our study.

The leucocyte count in peripheral blood, serum bilirubin, alkaline phosphatase, and serum aspartate transaminase were raised except in one patient with schistosomal liver disease in whom serum bilirubin was normal. Serum albumin and prothrombin time were low in all patients. In three patients blood cultures were positive. Mid-stream urine culture was negative in all 14 patients.

A possible precipitating factor for SBP was noted in half of our patients. Gastrointestinal bleeding was observed in two patients, bacteraemia in two patients and variceal sclerotherapy in two patients and pleural tap was performed in one patient prior to development of SBP.

Eleven patients were treated with cefuroxime and metronidazole parenterally for 10–14 days. Three patients were treated with parenteral ampicillin and gentamicin.

Despite antibiotic therapy, 11 of our patients died in hospital. All of these patients had advanced liver disease and half of them also had progressive renal failure. The three surviving patients were subsequently lost to follow-up.

Discussion

Hepatitis B-related chronic liver disease is common in Saudi Arabia, in contrast to alcoholic liver disease in the Western world. The prevalence of the carrier rate of hepatitis B is between 8 and 26% in Saudi Arabia. Two recent studies have shown that liver cirrhosis due to chronic B hepatitis varies between 17 and 21% in this part of the world. In our series of SBP patients, nine patients’ sera were positive for HbsAg (64%).

In our study the prevalence of SBP was 9.5% and this is similar to other studies. We feel that the prevalence may well be higher than our observations indicate. We suspect that some cases remain undiagnosed as neither the primary care physician nor the hospital based physician are aware of this condition. The clinical deterioration is perhaps regarded as a natural course of events of progressive liver failure with the background of chronic liver disease.

In our series, abdominal pain and hepatic encephalopathy were the prominent clinical features in contrast to 246 patients in seven large series in whom fever (67%) and abdominal pain (60%) were the initial presentations in SBP. This anomaly may be due to our small sample size.

The aetiology of SBP has been discussed by Crossley & Williams. We found that three patients in our series had undergone invasive procedures prior to developing SBP and two patients had had gastrointestinal bleeding which may cause increased permeability of gut thereby allowing bacteria to pass into ascitic fluid.

We note that only in two patients were Gram stains of ascitic fluid positive. This might have been due to the fact that a greater volume of ascitic fluid was not centrifuged in the laboratory. It has been observed when 50 ml of ascitic fluid is centrifuged, then the yield of Gram stain positivity increases from 19% to 55%.
Ascitic fluid of all of our patients with SBP grew organisms and this is attributed to bedside inoculation of ascitic fluid into blood culture bottle, rather than using the loop technique where the yield is poor. With the loop technique the number of microorganisms is far less than with direct inoculation of infected ascitic fluid into the blood culture bottle. Runyon compared the conventional culture method versus blood culture bottle results in patients with neutrocytic ascites and concluded that the yield was 42 versus 91%.

*Escherichia coli* was the commonest organism in our series and this is similar to other studies. However, it is interesting to note that one of our patients had an initial episode of SBP with *aeromonas hydrophila* and a second episode of SBP with *E. coli*. In another patient, there were two episodes of SBP with *E. coli* within a period of 3 months. Tito noted that 51% of their patients developed one or more episodes of SBP and this is related to more severe liver disease. It is usual to find a single organism in SBP. When one observes more than one organism, the suspicion of perforation of gut or contamination should be borne in mind.

The mechanism of SBP has been postulated to be due to spontaneous bacteremia which results in seeding of 'susceptible ascites fluid'. In chronic liver disease, the phagocytic activity of reticuloendothelial cells is reduced. In addition to this, in the presence of portal hypertension, intrahepatic and portosystemic shunting of blood leads to bacteremia and SBP.

In this series, total ascitic fluid protein varied between 0.45 g and 24 g/litre in 11 of our patients. In three patients, ascitic fluid protein was not documented. It has been observed that low ascitic fluid protein parallels low complement and opsonic levels which predispose to SBP. Half of our patients died due to progressive renal failure and the remainder died due to a combination of factors such as liver failure, infection and gastrointestinal bleeding.

Despite active treatment, the mortality in our series was high, (78%), the same as in other series. These patients who died had severe jaundice, peripheral leucocytosis and elevated serum creatinine indicating a poor prognosis in hospitalized patients. There is a greater tendency in cirrhotics to develop renal failure and 30% of patients with cirrhosis who were commenced on gentamicin, developed renal failure despite non-toxic blood levels. Currently aztreonam, co-amoxiclav and third generation cephalosporins are the antibiotics of choice, as these antibacterial agents penetrate the ascitic fluid rapidly and achieve anti-bacterial concentrations.

Hoefs & Runyon categorized the mode of death in SBP into two groups—fast and slow rates of death. In our series eight patients died between 10 days and 35 days and three died within 10 days of admission. Hoefs indicated the patients with alcoholic hepatitis with SBP tend to succumb rapidly in contrast to slow and quiescent death in advanced liver disease. We would like to stress that none of the 'early deaths' in our series were due to alcoholic liver disease, as it is well known that, due to religious and social reasons, alcohol consumption is at a low level in Saudi Arabia. Those patients who 'died early' had severe liver disease with renal failure i.e. hepatorenal syndrome.

We conclude that SBP exists in the Saudi population. The prevalence is similar to other reported series although there may be considerable underdiagnosis of the condition. Abdominal pain and hepatic encephalopathy remain the prominent clinical features in a Saudi population. Mortality remains high despite active treatment.

References