Spontaneous staphylococcal peritonitis: A case report

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INTRODUCTION

Ascites is a common problem in South Sudan but published data regarding the incidence are sparse. Much of that is related to nutritional deficiencies, the high incidence of Hepatitis B and C, and congestive heart failure. Although most ascites is transudative in nature, exudative ascites does occur and requires different management strategies.

CASE REPORT

A 41-year-old female presented with a two-week history of increasing abdominal girth. She had abdominal pain, but no vomiting or diarrhoea. She was negative for Hepatitis B and C. On examination, she had a clear chest and normal heart sounds, no murmurs or signs of heart failure. She had no significant adenopathy, was not jaundiced, and had no distension of superficial abdominal veins. Examination revealed a markedly distended abdomen that was moderately tender without localization. She had moderate tympany to percussion of the abdomen. An ultrasound showed +3 ascites. She denied any history of instrumentation of her abdomen. A paracentesis was performed for both diagnostic and therapeutic reasons, removing 1.8 litres of fluid.

The paracentesis showed slightly cloudy fluid. Gram stain showed an excess of WBC’s and Gram (+) cocci in clusters. There were no Gram (-) organisms. The laboratory, on the basis of the microscopical appearances, identified the organisms as a Staphylococcus. Facilities for culture and sensitivities were not available. She received IV cloxacillin, gentamicin, and oral metronidazole. Her symptoms improved, but after five days, she had re-accumulation of cloudy fluid, though ultrasound assessment of ascites was judged to be only +2. [There is no universally agreed scoring system for recording the degree of ascites as found with ultrasound. However, we have used the following: +1 is fluid in the pouch of Douglas and gutters, +2 is in the lower abdomen and pelvis, +3 is around the liver and spleen, and +4 the liver is floating].

Repeat Gram stain again showed moderate WBC’s and Gram (+) cocci in clusters. In the presence of bacterial infection the fluid was judged most likely to be an exudate, so we drained 4 litres, attempting to drain all.

The patient was discharged on cloxacillin. She returned one week later with a slight re-accumulation of ascites. The patient was also diagnosed with malaria and treated with Coartem (artemether and lumefantrine). A week later there was no abdominal pain or distension and, no ascites was noted by ultrasound.

DISCUSSION

Ascites is a common problem in South Sudan, primarily from patients with liver cirrhosis, and portal hypertension (often secondary to Hepatitis B or C) or congestive heart failure. Spontaneous bacterial peritonitis (SBP) most typically occurs in the setting of previous ascites. Bacterial peritonitis is typically from ruptured viscus, usually small bowel or colon. In that setting, the usual organisms are Gram (-) rods, such as Escherichia coli, and Klebsiella. Staphylococcal peritonitis is commonly found in the setting of peritoneal dialysis, and may be encountered following instrumentation of the abdomen, such as paracentesis,
but this patient denied having any previous abdominal punctures. The ascites was present and cloudy at the time of the initial paracentesis; the Gram stain was positive at that time, thus making it unlikely that the infection could have been introduced at the time of the paracentesis.

As with pleural fluid, ascites can be either a transudate (from increased pressure, as caused by congestive heart failure or portal hypertension) or an exudate from an inflamed membrane. The definitive distinction between a transudate and exudate is by protein content (exudate > 3.5 g/dl, transudate < 2.5 g/dl) but our hospital does not have that capacity at present. Transudates tend to respond well to diuretics, whereas an exudative peritonitis responds poorly. Bacterial infections, including TB, generally cause an exudative ascites.

If the likely aetiology of the ascites is unclear, paracentesis is indicated. As with pleural fluid, ascites can be either a transudate (from increased pressure, as caused by congestive heart failure or portal hypertension) or an exudate from an inflamed membrane. The definitive distinction between a transudate and exudate is by protein content (exudate > 3.5 g/dl, transudate < 2.5 g/dl) but our hospital does not have that capacity at present. Transudates tend to respond well to diuretics, whereas an exudative peritonitis responds poorly. Bacterial infections, including TB, generally cause an exudative ascites.

If the likely aetiology of the ascites is unclear, paracentesis is indicated. If the ascites is a transudate, 4-5 litres is usually the accepted upper limit of safe drainage to prevent large shifts of intravascular volume. If the ascites is exudative, such as in our case or in TB peritonitis, all the fluid should be removed if possible.

This is the second case of spontaneous Staphylococcal peritonitis to be reported. There is, of course, the possibility that the patient failed to report some instrumentation of the peritoneum, either at home or with the tribal doctors, but there were no wounds on the abdomen to suggest such manipulations. The response to parenteral antibiotics and paracentesis was favourable.

**CONCLUSION**

In a patient with rapid accumulation of ascites, particularly in the absence of congestive heart failure or Hepatitis B or C infection, paracentesis should be considered. The instruments required are typically available in most hospitals in South Sudan. If the fluid is clear or straw coloured and free flowing, it is most likely a transudate, so no more than 5 L should be removed at a time. If the fluid is cloudy, slow flowing, bloody, milky in appearance, further diagnostic studies are indicated and an attempt to drain all the ascites is appropriate.

**References**