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LETTER TO THE EDITOR

SPONTANEOUS BACTERIAL PERITONITIS COMPLICATING MALIGNANCY-RELATED ASCITES

To The Editor:

During a five-year period (1987–1992), we analyzed all cases with a diagnosis of spontaneous bacterial peritonitis (1). In two of the 60 identified cases, spontaneous bacterial peritonitis occurred in malignancy-related ascites. Both patients developed spontaneous bacterial peritonitis (SBP) in preexisting ascites. The first patient had diffuse liver metastases of an adenocarcinoma and ascites and developed an episode of spontaneous peritonitis, with *Escherichia coli* cultured from blood and ascitic fluid. The second patient had peritoneal adenocarcinomatosis with a portal vein thrombosis and developed peritonitis due to *Klebsiella pneumoniae* cultured from blood and ascitic fluid. Both patients were critically ill and had a poor nutritional status at the time of the infection. Ascitic fluid parameters are given in Table 1. The patients died eight days and eight weeks, respectively, after the onset of the infection.

A review of the literature confirms that SBP complicating malignancy-related ascites has only been described in patients who had evidence of portal hypertension. Isner et al (2) described one patient with a gastric adenocarcinoma who developed SBP after chemotherapy. At autopsy 75% of this patient's liver parenchyma had been replaced by tumor. Kurtz and Bronzo (3) reviewed more than 100 patients with cytology-positive ascitic fluid and found only three patients with bacterial peritonitis. The authors stated that these cases do not represent true SBP because invasive procedures in two patients and a nadir sepsis in one patient clearly predisposed them to bacterial peritonitis. Runyon (4) described one patient with SBP and cardiac ascites, but also in this patient there was evidence of portal hypertension with splenomegaly and esophageal varices due to liver fibrosis.

TABLE 1. ASCITIC FLUID PARAMETERS

	Patient	
	1	*2*
Leukocytes ($\times 10^9$/liter)	2.8	29.8
Polymorphonuclears (%)	88	90
Lymphocytes (%)	12	10
Lactate dehydrogenase (units/liter)	257	246
Amylase (units/liter)	45	220
Total protein (g/liter)	19	20
Albumin (g/liter)	6	12
Serum–ascites albumin gradient*	22	18

*Serum albumin minus ascitic fluid albumin.

Our cases and those reported above do suggest that the presence of portal hypertension, either due to massive hepatic metastases or portal vein thrombosis is a prerequisite for malignancy-related ascites to become infected. Intact antimicrobial activity of the peritoneal fluid and functioning hepatic tissue are important determinants in clearing microorganisms from the ascitic fluid (5). Comparing cirrhotic ascites with malignant ascites (without portal hypertension), complement concentrations C3 and C4 are about four times higher in the latter group, with better opsonic and chemoattractant activity (6).

The hepatic venous pressure gradient in patients with massive liver metastases has been found to be similar to that quantified in cirrhotic patients (7). This is confirmed by the presence of a wide (>11 g/liter) serum–ascites albumin gradient (as a manifestation of oncotic–hydrostatic balance), which has been found in patients with malignancy-related ascites and massive hepatic metastases (8, 9). The presence of portal hypertension gives rise to a significantly lower total protein concentration in the ascitic fluid, compared to patients with peritoneal carcinomatosis without portal hypertension (6, 8, 9). A low protein concentration in the ascitic fluid reflects decreased complement concentrations and decreased opsonic and bactericidal activity (6, 10, 11). This would explain the susceptibility of malignant ascites to the development of SBP in the presence of portal hypertension. In conclusion, there are only sporadic case reports of SBP not related to cirrhosis of the liver. The presence of portal hypertension due to massive liver metastases or to portal vein thrombosis appears to predispose patients with malignancy-related ascites to the development of SBP.

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