

Title:

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DOI: 10.17235/reed.2025.11279/2025

Link: [PubMed \(Epub ahead of print\)](#)

Please cite this article as:

Ojeda Remizova María, Cáceres Pieter Carmen Elisa, Diz Calderón Carlos, Pereiro Morillo Sol, Cerrella Cano Carolina, Junquera Alonso Eduardo, Parapar Álvarez Leire, Duque Alcorta Jose María. Spontaneous bacterial peritonitis (SBP) in ascites due to advanced pancreatic adenocarcinoma in the absence of chronic liver disease. Rev Esp Enferm Dig 2025. doi: 10.17235/reed.2025.11279/2025.

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Spontaneous bacterial peritonitis (SBP) in ascites due to advanced pancreatic adenocarcinoma in the absence of chronic liver disease

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Keywords: Spontaneous bacterial peritonitis. Ascites. Pancreatic adenocarcinoma.

Dear Editor,

We present the case of an 82-year-old woman diagnosed with pancreatic adenocarcinoma with hepatic metastases and no history of liver disease. She was admitted to the hospital for fever (38 °C) and abdominal distension with no pain. A therapeutic paracentesis was performed, draining 3,500 mL of cloudy whitish ascitic fluid. The analytical findings were consistent with spontaneous bacterial peritonitis (SBP) and portal hypertension (PH) (Table 1). Blood tests showed leukocytosis (21,300 leukocytes/ μ L), C-reactive protein 13 mg/dL, and procalcitonin 0.62 ng/mL. She also had known chronic cholestasis of neoplastic origin. *Escherichia coli* was isolated in both blood cultures and ascitic fluid culture. Abdominal ultrasound showed a neoplasm in the pancreatic head with a metallic biliary stent, numerous hepatic metastases, and moderate ascites. The portal and hepatic veins were patent and of preserved caliber. There was no splenomegaly or collateral circulation. No additional indirect or invasive studies were performed for assessment of PH.

She was treated with intravenous ceftriaxone 2 g daily for 7 days with clinical and analytical improvement. In the follow-up paracentesis, less turbid yellowish fluid was drained with analytical remission (Table 1). The patient was discharged for continued follow-up by home palliative care and died three weeks later.

DISCUSSION

Spontaneous bacterial peritonitis (SBP) is defined as a monomicrobial infection of ascitic fluid due to bacterial translocation from the gut, without evidence of an intra-abdominal origin. The 2021 AASLD guidelines establish the diagnosis of SBP based on the presence of >250 polymorphonuclear cells/ μL and low protein content with a serum-ascites albumin gradient (SAAG) >1.1 (1). In contrast, secondary peritonitis (SP) is characterized by polymicrobial dissemination from an intra-abdominal origin.

SBP typically occurs in ascites due to PH in the setting of chronic liver disease; however, its occurrence in non-cirrhotic patients with malignant ascites, as in our case, is extremely rare (2–5). Our patient developed SBP with bacteraemia in the context of ascites due to PH secondary to metastatic hepatic infiltration, similar to the case reported by Miyashita H et al., where portal vein thrombosis was also present (5). The most plausible pathophysiological mechanism of SBP associated with malignant ascites, is the development of post-sinusoidal PH due to embolization of the hepatic veins or sinusoidal PH due to massive metastatic infiltration. Unlike peritoneal carcinomatosis, where peritoneal cells secrete a leukocyte- and protein-rich fluid that induces ascites extravasation, in ascites due to PH the protein and complement (C3 and C4) concentrations are low, reducing opsonizing and bactericidal activity and increasing the risk of SBP (2). Another contributing mechanism is the dysfunction of the reticuloendothelial system barrier due to hepatic metastatic infiltration, impairing its role as a bacterial filter of the venous flow. Most cases described in the literature occur in gastrointestinal tumors (3).

In a multicentre retrospective study analysing 208 patients with malignant ascites, 29 patients presented with bacterial peritonitis (14% prevalence); pancreatic adenocarcinoma was the most frequent primary tumor (55%), and radiologically the most frequent findings were PH ($n=14$) and hepatic metastases ($n=16$) (3). Therefore, the mechanism of ascites should be considered in patients with disseminated tumors (e.g., massive hepatic metastases, Budd-Chiari syndrome), and one should remain alert to the possibility of SBP due to PH—even in the absence of radiological signs—since it is potentially fatal but treatable.

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Table 1. Biochemical Analysis of Initial Paracentesis and Subsequent Follow-Ups

Ascitic Fluid	At admission 27/06/23	Within 48 hours 29/06/23	After 7 días 6/07/23
Leukocytes (cells/ μ L)	8750/ μ L	2030/ μ L	564/ μ L
Polymorphonuclear Cells (%) and cells/ μ L)	88% 7700/ μ L	89% 1806/ μ	25% 141/ μ L
Lymphocytes (%)	12%	11%	75%
Albumin (g/dL)	1.2	0.6	0.8
Serum–Ascites Albumin	1, 07	1.6	1,4



Gradient (SAAG)			
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Serum–Ascites Albumin Gradient (SAAG)			
Total Proteins (g/dL)	2.08	1.12	1.36
LDH (U/L)	--	99	62
Glucose (mg/dL)	49	410*	232*
Culture	<i>E.Coli</i>	negative	negative

*Glucose elevation is explained by poor glycemic control in a patient with type 2 diabetes mellitus (DM2), with marked fluctuations in serum glucose due to discontinuation of oral antidiabetics and the necessity for continuous insulin therapy adjustments.