

Single Case

An Unusual Cause of Abdominal Ascites

Kimberly A. Wong^a Kristin A. Olson^b Eric W. Chak^c

^aDepartment of Internal Medicine, University of California Davis School of Medicine, Sacramento, CA, USA; ^bDepartment of Pathology, University of California Davis School of Medicine, Sacramento, CA, USA; ^cDivision of Gastroenterology and Hepatology, University of California Davis School of Medicine, Sacramento, CA, USA

Keywords

Ascites · Liver · Mesothelioma

Abstract

Abdominal ascites is most commonly caused by portal hypertension from liver cirrhosis. When present, portal hypertension is associated with an elevated serum-ascites albumin gradient (SAAG) ≥ 1.1 g/dL. In contrast, a SAAG < 1.1 g/dL suggests malignancy, tuberculosis, pancreatitis, or nephrotic syndrome. Here, we present a case of low SAAG ascites caused by epithelioid peritoneal mesothelioma in a woman with no known liver disease. The diagnosis proved elusive until diagnostic laparoscopy with biopsy was performed.

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Introduction

Approximately 10% of ascites cases in North America and Western Europe are explained by malignancy, with mesothelioma representing only 1% of such cases [1]. Peritoneal mesothelioma has a prevalence of 1 per 1,000,000 individuals per year, with approximately 250 new cases diagnosed in the United States annually [2]. The peritoneum is the second most common site of mesothelioma after the pleura, with peritoneal mesothelioma representing 10–20% of all mesotheliomas diagnosed in the United States [2]. Asbestos exposure is the most widely recognized risk factor for development of the disease [3, 4]. Presenting signs and

symptoms include abdominal pain, increased abdominal girth, weight loss, new-onset hernia, and incidental ascites [4, 5]. Diagnosis is often delayed and relies on computed tomography (CT) imaging and laparoscopy for staging and confirmatory biopsy [5]. Treatment options include cytoreductive surgery, intraperitoneal chemotherapy, and systemic chemotherapy [2]. Median survival time with treatment ranges from 30 to 92 months [5].

Case Report

A 73-year-old woman with a 5-week history of abdominal swelling was referred to hepatology clinic. She had no history of liver disease. Her medical history included hypothyroidism and a remote total abdominal hysterectomy and bilateral salpingo-oophorectomy for uterine leiomyomata. She had no history of smoking, alcohol abuse, or direct exposure to asbestos. Her most recent mammogram and colonoscopy were normal. Her mother and sister had melanoma, her brother had brain cancer, and her father had colon, prostate, and bladder cancer. Physical examination of the patient revealed a nondistended abdomen and no lower extremity edema. A recent abdominal ultrasound revealed a moderate volume of ascites but no splenomegaly or other signs of portal hypertension. Basic chemistry panel and liver chemistries were completely normal, though a complete blood count was notable for a mild thrombocytosis (platelet count of 432,000/mm³). C-reactive protein was elevated to 3.5 mg/dL, suggesting systemic inflammation. Serum Quantiferon Gold was negative.

A diagnostic paracentesis led to removal of 2 L of serous fluid characterized by serum-ascites albumin gradient (SAAG) of 0.5 g/dL, total protein of 6.4 g/dL, normal lactate dehydrogenase (103 U/L), and normal glucose of 99 mg/dL. Ascitic fluid cell count revealed 695 WBC/mm³ with 4% neutrophils and 73% lymphocytes. Gram stain of the fluid did not reveal any pathogenic bacteria, and cytology was negative for malignancy. Ascitic fluid adenosine deaminase, bilirubin, and amylase were also normal.

Imaging tests were unrevealing as to the cause of her ascites. Transthoracic echocardiogram showed a normal left ventricular ejection fraction and no evidence of right heart failure. Magnetic resonance imaging (MRI) of the abdomen with and without contrast did not reveal an intra-abdominal malignancy. A noncontrast CT of the chest (contrast was held due to shellfish allergy) revealed biapical scarring with pleural and subpleural nodularities that were stable compared to prior imaging. Subsequent positron emission tomography scan revealed no fludeoxyglucose uptake of these nodules, suggesting they were benign. A diagnostic upper endoscopy revealed no varices or obvious malignancy, and random duodenal biopsies were normal. A repeat diagnostic colonoscopy was normal. Transjugular liver biopsy revealed normal liver histology without fibrosis. Recurrent paracenteses were required to control her abdominal ascites despite use of oral diuretics.

Finally, a diagnostic laparoscopy was performed and revealed diffuse peritoneal studding with mucinous deposits, more concentrated in the upper abdomen, and a fluid-filled mass associated with the appendix. Peritoneal biopsies revealed low-grade malignancy consistent with mesothelioma. Chemotherapy and possible cytoreductive surgery are planned.

Discussion

Malignancy-induced ascites is relatively uncommon, with adenocarcinoma of the ovaries, breast, and stomach accounting for about half of such cases. Mesothelioma is an unusual cause

of malignancy-induced ascites, representing only 1% of such cases [1]. Here, we review the established diagnostic algorithm for ascites and acknowledge both the utility and limitations of ascitic fluid testing.

SAAG ≥ 1.1 g/dL is suggestive of portal hypertension, while SAAG < 1.1 g/dL is suggestive of infection, malignancy, or other peritoneal causes. Since no evidence of spontaneous bacterial peritonitis was present, the next step suggested by the diagnostic algorithm is evaluation for peritoneal carcinomatosis (via cytology) and tuberculous peritonitis [6]. Our patient's cytology was negative for malignancy on two separate paracenteses. The sensitivity for cytologic diagnosis of malignancy in a peritoneal effusion is estimated to be as low as 62% [7]. With cytologic examination of three samples from three different paracenteses, this sensitivity may increase to 97% [8]. Need for mycobacterial culture was obviated because this patient was at low risk for tuberculosis, and previous Quantiferon Gold testing was negative. Normal ascitic fluid amylase and lack of proteinuria effectively excluded pancreatic and kidney causes. As mentioned, MRI of the abdomen and pelvis was negative for evidence of malignancy.

The diagnosis of epithelioid peritoneal mesothelioma was finally obtained after laparoscopy, which allowed for direct visualization of peritoneal thickening and nodularity (Fig. 1) and histopathologic examination of the peritoneal biopsies (Fig. 2, 3).

Statement of Ethics

Informed consent was obtained with the patient discussed in this case report.

Disclosure Statement

The authors have no conflicts of interest to report.

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Fig. 1. Diffuse peritoneal nodularity seen during diagnostic laparoscopy.

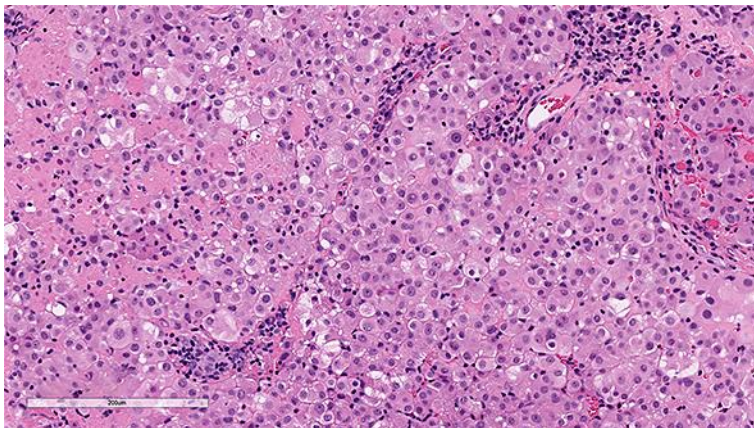


Fig. 2. Epithelioid peritoneal mesothelioma is characterized by large, epithelioid cells with plentiful and often bubbly cytoplasm, centrally located nuclei with prominent nucleoli, and crisp cell borders. HE. $\times 20$.

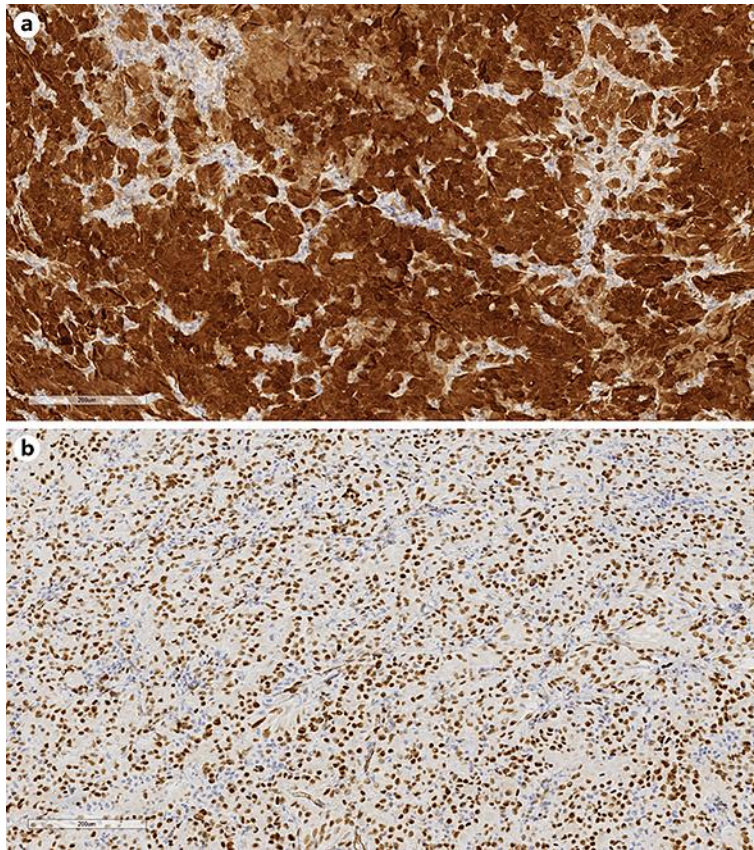


Fig. 3. Epithelial peritoneal mesothelioma can be differentiated from adenocarcinoma in part by its immunopositivity for calretinin (**a**; calretinin stain, $\times 13$) and WT-1 (**b**; WT-1 stain, $\times 13$).