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## A Case of Metastatic Gastric Signet Ring Cell Carcinoma Presenting as Tuberculous Peritonitis in an HIV/HCV Coinfected Patient

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### Abstract

Portal hypertension due to cirrhosis is the most common cause of ascites. However, in patients with multiple risk factors for ascites, determining the etiology can sometimes present a diagnostic challenge. We present a case of new onset ascites in a 59 year old African American man with concomitant HIV (CD4 54) and chronic active hepatitis C infection, and suspected tuberculosis corroborated by elevated Adenosine Deaminase (ADA). In this unusual case, despite suggestive symptoms and signs, we found the cause of his ascites was neither infection nor liver disease, but rather metastatic gastric malignancy. Ultrasound with Doppler did not show cirrhotic features or vascular thrombus in the liver, while diagnostic and therapeutic paracentesis revealed a low Serum Albumin-Ascites Albumin Gradient (SAAG) and high ascitic fluid WBC count with monocytic predominance. Repeat ascitic fluid testing confirmed low SAAG, and ADA was elevated at 26.3 IU/mL. Laparoscopy revealed innumerable peritoneal lesions resembling tuberculous seeding of the peritoneum. However, peritoneal histopathology returned as metastatic signet cell adenocarcinoma. Esophagogastroduodenoscopy (EGD) revealed a large, firm, ulcerated gastric body tumor, and pathology was consistent with diffuse signet ring cell carcinoma. In this case, cirrhosis and tuberculous infection were highly suspected, however there was no evidence of cirrhosis, and although acid-fast smear and Quantiferon testing were negative, tuberculous peritonitis was strongly considered due to history of HIV and high monocyte predominance of ascitic fluid with elevated ADA. Both findings are suggestive of tuberculous peritonitis, however, ADA can also be elevated in the setting of malignancy and the pre-test probability of tuberculosis should be considered when sending ADA levels. Confirmation of the diagnosis with peritoneal biopsy is paramount.

**Keywords:** Gastric cancer; Tuberculosis; HIV; HCV

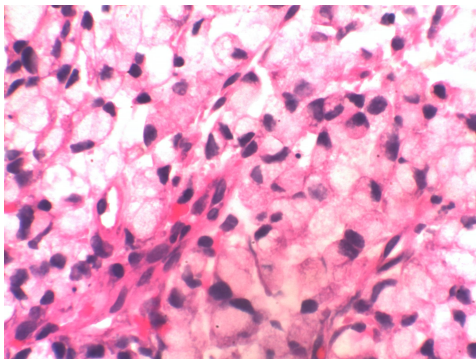
### Introduction

New onset abdominal ascites can sometimes present a diagnostic challenge. Portal hypertension due to cirrhosis is the most common cause of ascites [1], and would certainly be the most likely etiology of ascites in any patient with a prior history of chronic liver disease. Paracentesis provides both therapeutic and diagnostic benefit, allowing for fluid analysis that may identify its etiology, clarifying the source of pathology as either hepatic, cardiac, infectious or malignant. Among infectious causes, the diagnosis of tuberculous peritonitis is often delayed due to the organism's fastidious growth pattern, but should also be strongly considered in a patient with known HIV infection. The adenosine deaminase test can be helpful to confirm the diagnosis of tuberculous peritonitis [2]. Here we present a case report of new onset ascites in a man with a prior history of concomitant HIV and hepatitis C. In this rare case, despite suggestive symptoms and signs, we found the cause of his ascites was neither infection nor liver disease, but rather metastatic gastric malignancy.

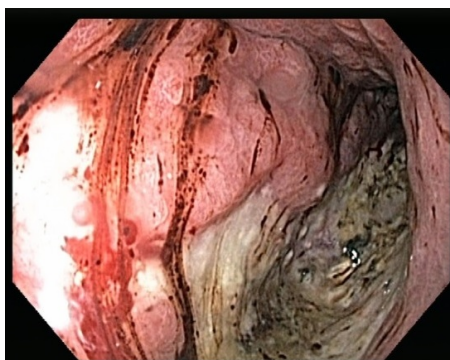
### Case

Our patient was a 59 year old African American man with HIV (CD4 54) and chronic active hepatitis C who presented to the emergency department with abdominal pain, nausea, weight loss and increasing abdominal girth for two months. Pertinent findings on physical examination included a full, distended abdomen with bulging flanks, with visible fluid wave and audible dullness to percussion. Laboratory findings were significant for hypoalbuminemia, but there was absence of transaminase elevation, thrombocytopenia, or elevated INR. Given his known history of HIV, tuberculous peritonitis was considered, but both PPD and serum Quantiferon-TB tests were negative.

Radiologic evaluation included a right upper quadrant ultrasound examination with vascular Doppler studies, which did not show cirrhotic features including nodular liver contour or hepatofugal flow, nor vascular thrombus in the liver. Abdominal CT was also negative for intra-abdominal organ abnormality. Diagnostic and therapeutic paracentesis was performed, and revealed a low Serum Albumin-Ascites Albumin Gradient (SAAG) and high ascitic fluid WBC count with monocytic predominance. The peritoneal fluid was tested for cytology, acid-fast bacilli smear, culture, and gram stain which were all negative. A follow-up repeats diagnostic and therapeutic paracentesis again revealed a low SAAG, and Adenosine Deaminase (ADA) testing was elevated at 26.3 IU/mL. Given the disparate findings that suggested but could not confirm tuberculous peritonitis, the patient underwent a diagnostic laparoscopy with peritoneal biopsy. On laparoscopy, innumerable peritoneal lesions were noted, grossly resembling tuberculous seeding of the peritoneum. Putting together the monocytic leukocyte predominance on ascitic fluid analysis, elevated ADA level of the ascitic fluid with these laparoscopic findings, the presumed diagnosis of tuberculous peritonitis was made, and anti-tuberculin therapy was initiated. The following day, pathology from the peritoneal biopsy specimen returned as metastatic signet cell adenocarcinoma (**Figure 1**). An esophagogastroduodenoscopy was subsequently performed revealing a large, firm ulcerated tumor in the greater curvature of the gastric body (**Figure 2**). Pathology was consistent with diffuse signet ring cell carcinoma.



**Figure 1** Diffuse signet ring cell gastric adenocarcinoma.



**Figure 2** Gastric adenocarcinoma.

## Discussion

It is essential to develop a broad differential when evaluating a patient with new onset abdominal ascites. The Serum Ascites-Albumin Gradient (SAAG) can help narrow the differential diagnosis, distinguishing between exudative and transudative etiologies, and the total ascitic fluid protein further clarifies the diagnosis as either hepatic or cardiac within the transudative category. In this case, either cirrhosis or tuberculous infection were highly suspected given his past medical history. However, there was no evidence to support the diagnosis of cirrhosis on imaging including no splenomegaly, and there was no thrombocytopenia or coagulopathy on laboratory evaluation as is often seen in cirrhosis. The low SAAG also pointed toward an exudative process such as infection or malignancy, which further discounted the likelihood of cirrhosis. Tuberculous peritonitis was strongly considered due to his history of HIV, as well as the ascitic fluid testing that revealed a high monocyte predominance and elevated ADA. Both of these findings have been described as suggestive of the diagnosis of tuberculous peritonitis [3,4]. In addition, the ADA test was demonstrated by a meta-analysis to have a high sensitivity and high specificity for revealing tuberculous peritonitis in ascitic fluid [1]. However, it should be noted that the ADA can also be elevated in the setting of malignancy [5]. While HIV and AIDS are linked with certain malignancies, currently there is no evidence in the literature to suggest that HIV predisposes individuals with an increased risk of gastric cancer compared to the general population [6]. Despite the evidence to suggest the diagnosis of tuberculous peritonitis, it should also be considered, however, that his acid-fast smear and Quantiferon testing were both negative, modifying the pre-test probability of the ADA test. This case emphasizes the importance of considering the ADA pre-test probability in the diagnostic workup of new onset ascites when tuberculous peritonitis is strongly considered. Confirmation of the diagnosis with peritoneal biopsy is paramount to avoid missing a possible malignancy, as this may also present with high ADA levels.

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