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Beyond cirrhosis: A case series of non-cirrhotic causes of ascites in women

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Abstract

Ascites is most commonly a manifestation of cirrhosis and portal hypertension, yet a wide range of non-cirrhotic conditions can underlie its development, particularly in women where overlapping clinical presentations often complicate diagnosis. This case series highlights three uncommon etiologies of ascites: primary peritoneal carcinomatosis, Budd-Chiari syndrome, and abdominal tuberculosis, each posing unique diagnostic and therapeutic challenges. Despite shared features such as abdominal distension and nonspecific laboratory findings, careful integration of ascitic fluid analysis, imaging, and histopathology allowed accurate distinction between these entities. Primary peritoneal carcinomatosis was confirmed on omental biopsy and managed with systemic chemotherapy; Budd-Chiari syndrome, linked to oral contraceptive use, responded to anticoagulation; and peritoneal tuberculosis was established through biopsy-proven granulomatous inflammation and treated effectively with anti-tubercular therapy. These cases emphasize that while cirrhosis accounts for the majority of ascites worldwide, clinicians must remain alert to alternative causes. A structured diagnostic approach combining biochemical, radiologic, and histopathological evaluation is essential to ensure timely recognition and tailored treatment, thereby improving outcomes in patients presenting with non-cirrhotic ascites.

Introduction

Ascites, defined as the pathological accumulation of fluid within the peritoneal cavity, is most frequently encountered in the setting of advanced liver cirrhosis and portal hypertension. It is estimated that approximately 50% of patients with cirrhosis will develop ascites within ten years, and its occurrence usually denotes hepatic decompensation and a poor prognosis [1,2]. Since cirrhosis accounts for the majority of ascites cases, it is commonly presumed to be the underlying cause when ascites is identified.

Nevertheless, ascites may also result from a wide spectrum of non-hepatic conditions, which are particularly important to recognize due to their distinct management strategies and prognostic implica-

tions. Malignant ascites, often related to peritoneal carcinomatosis, accounts for nearly 10% of cases in clinical practice and may mimic ovarian or gastrointestinal malignancies [3]. Similarly, Budd–Chiari syndrome, characterized by hepatic venous outflow obstruction, is an uncommon but well-documented cause of ascites, with risk factors such as myeloproliferative disorders and oral contraceptive use [4,5]. Peritoneal tuberculosis represents another diagnostic challenge, especially in endemic regions, as its clinical and radiological presentation can closely resemble peritoneal carcinomatosis or advanced ovarian cancer [6,7].

In women, these non-cirrhotic causes of ascites often present a diagnostic dilemma due to overlapping clinical features and nonspecific laboratory results [8]. Although conventional tests such as the Serum–Ascites Albumin Gradient (SAAG), cytology, and imaging can offer valuable diagnostic information, confirmation often necessitates histopathological evaluation [9]. A systematic and comprehensive approach is therefore essential to avoid misdiagnosis and treatment delays.

The present case series describes three women who developed ascites due to non-cirrhotic aetiologies primary peritoneal carcinomatosis, Budd–Chiari syndrome, and abdominal tuberculosis. By highlighting the diagnostic process and clinical course of these patients, we aim to underscore the importance of maintaining a broad differential diagnosis when evaluating ascites, particularly in populations where both infectious and malignant diseases remain prevalent.

Case Presentations

Case 1: Primary peritoneal carcinomatosis

A 55-year-old woman presented with progressive abdominal distension of one month's duration, associated with loss of appetite and significant weight loss over the preceding three months. There was no history of abdominal pain, fever, vomiting, cough, dyspnoea, or altered sensorium. She had no previous similar complaints and no known comorbidities.

On examination, the patient was conscious, oriented, and afebrile. She was moderately built but appeared pale. There were no icterus, pedal oedema, lymphadenopathy, or other stigmata of chronic liver disease. Vital signs were stable with a pulse of 98/min, blood pressure of 110/80 mmHg, and oxygen saturation of 98% on room air.

Laboratory investigations showed haemoglobin of 9 g/dl, total leukocyte count of 11,000/mm^3, and platelet count of $1.4\times10^5/mm^3$. Liver and renal function tests were within normal limits, with serum albumin 3.6 g/dl, PT/INR 1.2, and negative viral serologies. Ultrasonography revealed moderate ascites without hepatosplenomegaly.

Ascitic fluid analysis showed a haemorrhagic exudate with low Serum–Ascites Albumin Gradient (SAAG) and high protein content. Cytology was positive for malignant cells, and ascites recurred despite repeated therapeutic paracenteses. Contrast-enhanced CT of the abdomen demonstrated multiple peritoneal nodules, omental thickening with haziness, and ascites. Omental biopsy revealed sheets of poorly differentiated malignant cells with prominent nucleoli, consistent with primary peritoneal carcinomatosis.

The patient was started on chemotherapy with cisplatin and pemetrexed and continues to be followed up in the oncology clinic.

Case 2: Budd–chiari syndrome

A 35-year-old woman presented with abdominal distension of two weeks' duration and diffuse abdominal pain for one week. The pain was dull, non-radiating, and without diurnal variation. There was no associated fever, vomiting, jaundice, melena, weight loss, or respiratory complaints. She had no significant medical history, but reported oral contraceptive use for the past three months.

On examination, the patient was conscious, oriented, and afebrile. Vitals were stable. Abdominal examination revealed distension with tenderness localized to the right hypochondrium; other systemic findings were unremarkable.

Routine blood counts, renal function, and liver function tests were within normal limits. Ascitic fluid analysis showed a high-SAAG, high-protein exudate, with negative staining for acid-fast bacilli. Portal vein Doppler demonstrated absent flow in the left hepatic vein and hepato-fugal portal flow without collateralization. Viral hepatitis markers were negative. Contrast-enhanced CT of the abdomen confirmed thrombosis of the portal and hepatic veins, diagnostic of Budd–Chiari syndrome. Echocardiography was normal, and upper gastrointestinal endoscopy revealed erosive gastritis.

The patient was diagnosed with Budd–Chiari syndrome secondary to oral contraceptive–induced hepatic venous thrombosis. She was started on intravenous heparin and subsequently bridged to long-term anticoagulation with acenocoumarol. She is currently under regular follow-up with clinical improvement and stable anticoagulation control.

Case 3: Abdominal tuberculosis

A 36-year-old woman presented with gradually progressive abdominal distension of one month's duration, accompanied by significant weight loss. There was no history of fever, gastrointestinal bleeding, respiratory symptoms, or prior tuberculosis. She had no significant comorbidities.

On examination, she was conscious and oriented, with stable vital parameters. Pallor was noted, but there was no icterus or peripheral oedema. Abdominal examination revealed a distended, non-tender abdomen with ascites, and no palpable hepatosplenomegaly.

Haematological, renal, and liver function tests were within normal limits. Ascitic fluid analysis showed a low-SAAG, high-protein exudate with lymphocytic predominance (75%) and an Adenosine Deaminase (ADA) level of 64 IU/L. Staining for acid-fast bacilli and CBNAAT were negative. Contrast-enhanced CT of the abdomen revealed features suggestive of wet-type peritoneal tuberculosis. A diagnostic peritoneal biopsy demonstrated granulomatous inflammation with caseous necrosis, confirming the diagnosis of peritoneal tuberculosis.

The patient was started on first-line anti-tubercular therapy with isoniazid, rifampicin, pyrazinamide, and ethambutol. She remains on therapy and has shown steady symptomatic improvement on follow-up.

Ascitic fluid analysis summary

Parameter	Case 1: PPC	Case 2: BCS	Case 3: TB
SAAG	<1.1 (Low)	>1.1 (High)	<1.1 (Low)
Protein	High	High (early)	High
Glucose	Low	Normal	Low
LDH	Elevated	Normal	Elevated
Cell count	Lymphocyte-predominant	Haemorrhagic	Lymphocyte-predominant
AFB/CBNAAT	Negative	Negative	Negative (biopsy +)
Tumor markers	↑ CEA, ↑ CA-125	Not elevated	Not elevated

Discussion

This series highlights three uncommon but clinically significant causes of ascites in women. Primary peritoneal carcinomatosis, although histologically similar to epithelial ovarian carcinoma, can present with minimal ovarian involvement and is often diagnosed only after cytology and biopsy confirmation. CA-125 is a useful diagnostic and monitoring marker, but prognosis remains guarded, with most series reporting survival of less than two years despite systemic therapy [10].

Budd–Chiari syndrome results from hepatic venous outflow obstruction. Oral contraceptives are recognized as an independent risk factor, particularly in women without other thrombophilic states. Anticoagulation remains the mainstay of treatment and has been shown to improve both survival and quality of life, with interventions such as Transjugular Intrahepatic Portosystemic Shunt (TIPS) or liver transplantation reserved for refractory cases [11].

Peritoneal tuberculosis is a common mimic of malignancy in endemic regions. While ascitic ADA is a sensitive marker, microbiological confirmation remains difficult due to low sensitivity of smear and culture methods. Histopathology therefore plays a vital role, as in the present case. Prompt initiation of anti-tubercular therapy is associated with excellent outcomes in the majority of patients [12]. The role of ascitic fluid analysis in distinguishing portal hypertensive from peritoneal causes cannot be overstated. The serum–ascites albumin gradient remains a simple yet reliable tool in guiding the diagnostic pathway. Our three cases illustrate how integrating fluid analysis with imaging and histopathology can narrow the differential diagnosis and ensure appropriate treatment.

Conclusion

While cirrhosis accounts for the majority of ascites worldwide, clinicians must remain vigilant for alternative causes in women presenting with atypical features. Primary peritoneal carcinomatosis, Budd–Chiari syndrome, and abdominal tuberculosis represent important differentials that require a high index of suspicion. Comprehensive evaluation through fluid analysis, imaging, and histopathological confirmation is essential for accurate diagnosis and timely therapy.

Informed consent: Written informed consent was obtained from all patients for publication of their clinical information in this report.

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