Black ascitic fluid in a patient with history of alcohol abuse: report of an unusual case and literature review

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Abstract

The differential diagnosis for black ascites include pancreatic ascites (PA). In majority of cases described the ascitic fluid as amber or black-colored. We report the case of a 33-year-old man with a history of alcohol abuse who presented with 24 h of epigastric pain and abdominal distension. Laboratory results showed lipase of 1270 U/l, amylase of 442 U/l and albumin of 2 g/dl. A contrast computed tomography scan of the abdomen showed pancreatic necrosis with atrophy and free abdominal fluid. Paracentesis was performed, yielding opaque black ascitic fluid. An ascitic fluid analysis demonstrated amylase of 2769 U/l, albumin of 1.6 g/dl, was negative for malignant cells and tuberculosis. Serum-Ascites Albumin Gradient was resulted in 0.4. The diagnosis of chronic pancreatitis was suspected, and he received supportive care with pain medication and bowel rest. He was discharged with symptom free on Day 14 after admission.

INTRODUCTION

The gross appearance of ascitic fluid can provide useful diagnostic information [1]. The differential diagnosis for black ascites can include bowel perforation with leakage of fecal matter, fungal peritonitis, metastatic melanoma, primary ovarian carcinoma and pancreatic ascites (PA) [2]. PA is an uncommon cause of abdominal ascites, accounting for approximately 1% of all cases [1].

CASE REPORT

A 33-year-old Mexican man with a history of alcohol abuse presented to the hospital with 24 h of epigastric pain and abdominal distension. He reported daily intake of 3 l of cane alcohol over the past week. He was hospitalized in the last 2 years for six episodes of alcohol-related acute pancreatitis. He informed a history of steatorrhea, progressive ascites and 10 kg loss of weight over the last year.

His examination was notable for generalized abdominal tenderness, tense ascites and oliguria. Laboratory results showed hemoglobin of 16 g/dl, white blood cell count of 11 × 10⁹/l and a platelet count of 262 × 10⁹/l. His international normalized ratio was 1.4, with an aspartate aminotransferase of 42 IU/l, alanine aminotransferase of 19 IU/l, total bilirubin of 0.8 mg/dl, alkaline phosphatase of 111 U/l, lipase of 1270 U/l, amylase of 442 U/l, tryglycerides of 140 mg/dl, creatinine of 6 mg/dl and albumin of 2 g/dl. Hepatitis and human immunodeficiency virus serology were negative. A contrast computed tomography scan of the abdomen was notable for pancreatic necrosis with atrophy, (Fig. 1, arrow), no alteration of bile tract and free abdominal fluid (Fig. 2, arrow). Paracentesis was performed, yielding 4.4 l of opaque black ascitic fluid (Fig. 3). An ascitic fluid analysis demonstrated the following: red blood cell count of 2–3/μl, white blood cell count of 30 cells/μl, total protein of 3.3 g/dl, amylase of 2769 U/l and albumin of 1.6 g/dl.

Determination of Serum-Ascites Albumin Gradient (SAAG) resulted in 0.4. The ascitic fluid analysis was negative for
malignant cells and tuberculosis. There were no organisms on ascitic fungal and bacterial cultures. The diagnosis of chronic pancreatitis was made due to the patient's medical history as well as his ascitic fluid analysis.

The patient received supportive care with pain medication and bowel rest. His renal function enhanced (Serum Creatinine of 0.8 mg/dl and Urine Output of 1.2 l/day) over the next 48 h after the paracentesis. On Day 5 of hospitalization the patient's abdominal pain resolved and he initiated sodium-restricted diet without pain. He was discharged with symptom free on Day 14 after admission. At the 2-week follow-up, he was asymptomatic.

**DISCUSSION**

PA was first reported in the literature by Smith [5], who described two cases of chronic pancreatitis associated with ascites. PA is frequently a result of pancreatic duct leak or chronic pancreatitis secondary to alcoholism. Patients typically have a history of chronic alcoholism, pancreatic disease or liver disease. Pleural effusions may be present, and PA is more common in males [2]. The incidence of ascites in chronic pancreatitis is difficult to estimate [7]. In majority of cases described the ascitic fluid as amber or black-colored [6]. Low SAAG is found in the absence of portal hypertension and is usually due to peritoneal disease. The most common cause is peritoneal carcinomatosis. Other causes are tuberculous peritonitis, pancreatic disease, biliary ascites, nephrotic syndrome, sepsis and bowel obstruction or infarction [8]. Given the patient's SAAG of 0.4, it could be argued that portal hypertension was not the cause of ascites. PA is defined as the persistent accumulation of intraperitoneal fluid which is characterized by a high amylase level (an ascites amylase over 1000 U/l than serum amylase) and a high protein content, usually over 3 g/dl [4]. In our case, cause of black ascites was clarified by patient's history of alcohol abuse, ascitic fluid analysis (high amylase, negative cytology and cultures) and computed tomography findings. No randomized clinical trials exist to guide treatment for PA. Treatment is guided by case reports, case series and meta-analyses with no established treatment algorithm [3]. Therapeutic options for PA include conservative and interventional options. Conservative therapy includes serial large volume paracentesis, elemental diet, parenteral nutrition, diuretics and somatostatin analogues. These interventions are thought to decrease pancreatic secretions promoting serosal apposition and healing. Interventional therapy includes either...
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surgical or endoscopic options encompassing a wide variety of procedures including sphincterotomy, cystogastrotomy, cystojejunoscopy and cystoduodenostomy. These procedures facilitate closure of pancreatic ductal disruption by promoting flow of pancreatic fluids into the digestive tract [2–4]. This case adds to the limited literature reporting successful management of PA with conservative therapy. In this case, therapeutic abdominal paracentesis, pain medication and bowel rest were sufficient to improve ascites.

CONFLICT OF INTEREST STATEMENT
None declared.

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ETHICAL APPROVAL
Not applicable.

CONSENT
Written consent was obtained from the patient.

GUARANTOR STATEMENTS
José Martín Alanís Naranjo.

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