

Acute chylous ascites mimicking acute appendicitis in a patient with pancreatitis

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Abstract

We report a case of acute chylous peritonitis mimicking acute appendicitis in a man with acute on chronic pancreatitis. Pancreatitis, both acute and chronic, causing the development of acute chylous ascites and peritonitis has rarely been reported in the English literature. This is the fourth published case of acute chylous ascites mimicking acute appendicitis in the literature.

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INTRODUCTION

Chylous ascites is the accumulation of chyle in the peritoneal cavity. It develops following disruption of the lymphatic system caused by traumatic injury or by either benign or malignant processes^[1]. It is usually a chronic process. This phenomenon is rarely associated with the symptoms and signs of peritonitis. Acute abdominal pain with peritonism due to sudden extravasation of chyle into the peritoneal cavity is rare but has been described in the literature.

CASE REPORT

A 38-year-old indigenous Australian man presented to the emergency department of our institution with a 24-h history of generalised abdominal pain localizing to the right iliac fossa. Associated symptoms included anorexia and nausea but no vomiting or diarrhoea. On examination he was systemically unwell with a temperature of 38.6 and was tachycardiac with a pulse of 115 beats per minute. On examination the patient was exquisitely tender in the right iliac fossa with rebound tenderness and peritonism, consistent with a diagnosis of acute appendicitis. His medical history was significant for chronic alcohol dependence and chronic pancreatitis. The patient admitted to drinking in excess of 120 g of alcohol daily but denied alcohol consumption for the two days prior to presentation. He had no significant surgical history.

Haematology and biochemistry investigations on presentation demonstrated deranged liver function, a macrocytosis and thrombocytopenia. Gamma glutamyl transaminase was elevated at 1392 U/L, with lesser elevations of alkaline phosphatase and alanine transferase, consistent with his known history of alcohol (ETOH) abuse and probable underlying alcoholic liver disease. C reactive protein (CRP) and white cell count (WCC) were not elevated. Blood film was normal and blood cultures negative. Lipase was elevated at 153 U/L.

The patient was taken to theatre with a clinical diagnosis of acute appendicitis. Subsequently a grid-iron incision was made revealing "milky" peritoneal fluid which at the time was presumed to be pus secondary to a perforated viscus despite its atypical appearance.

The appendix was not inflamed. Specimens were taken for biochemistry and microbiology. The decision was then made to perform a laparoscopy with a view to identifying the source of the fluid. When no obvious cause could be found, a midline laparotomy incision was made. The duodenum and stomach were mobilized, the lesser sac entered to exclude a perforated peptic ulcer while the remainder of the small bowel and colon were examined with no evidence of perforation. The only significant finding was that of a malrotated pancreas with an indurated and erythematous head consistent with acute pancreatitis. At this stage a presumed diagnosis of chylous peritonitis was made. Drain tubes were inserted and the midline incision was closed. The patient was discharged on day 4 after an uneventful post-operative course. In the initial few days post-operatively, there was significant drainage of the small blood-stained milky fluid seen at laparotomy. Interestingly, after several hours of stasis there was sedimentation of the blood from chyle within the drain tube bags. Fluid was sent for lipoprotein electrophoresis to confirm the diagnosis of chylous ascites.

A CT scan was completed on day 1 post-operatively to define the anatomy of the pancreas and exclude other pathology. The CT report confirmed an annular pancreas with changes surrounding the pancreatic head representing focal pancreatitis. There were no pancreatic calcifications seen.

The peritoneal fluid was significant for cholesterol of 7.3 mmol/L and triglycerides of 26.0 mmol/L (2280.7 mg/dL), highly suggestive of chylous effusion. Fluid amylase and lipase levels were elevated at 115 U/L and 2165 U/L, respectively, suggesting pancreatitis as the aetiology in this case. Plasma triglyceride levels were not measured.

DISCUSSION

Pathophysiology and aetiology

Chylous ascites is the accumulation of a milk-like peritoneal fluid rich in triglycerides, due to the presence of thoracic or intestinal lymph in the abdominal cavity^[1]. Causes of chylous ascites (Table 1) relate to disruption of the lymphatic system due to traumatic injury or obstruction.

Chronic chylous ascites is usually asymptomatic, however, abdominal pain from chylous peritonitis, with sudden outpouring of chyle into the peritoneum, has been described in the literature. A review of 140 cases of all forms of chylous ascites showed that 21% of adults and 14% of children with chylous ascites have the acute form^[2].

Pancreatitis is a recognised but rare cause of chylous effusion. In most published cases pancreatitis, usually chronic, results in the development of chronic chylous ascites without acute abdominal pain. Until 1984 only two cases of pancreatitis, both chronic, had been reported as causes of chylous effusions^[3]. Goldfarb^[4] described the first case of acute pancreatitis associated with acute chylous ascites, abdominal pain and

Table 1 Aetiology of chylous ascites^[3]

Congenital (most common in the paediatric population)
Congenital idiopathic
Intestinal lymphangiectasia (mega lymphatics)
Primary lymphatic hypoplasia
Chyle cysts
Lymphangiomas
Acquired
Neoplastic (most common in adult population)
Malignant
Lymphoma
Kaposi's sarcoma
Lymphangiomyomatosis
Carcinoid tumours
Other cancers (breast, pancreatic, colon, renal, testicular, ovarian, prostate)
Benign
Postoperative
Resection of the abdominal aorta
Retroperitoneal lymphadenectomy
Pancreaticoduodenectomy
Vagotomy
Radical nephrectomy
Warren shunt
Nissen fundoplication
Placement of peritoneal dialysis catheter
IVC resection
Inflammatory
Radiation therapy
Tuberculosis
Pancreatitis
Filariasis/ascariasis
Peritoneal dialysis
Sarcoidosis
Constrictive pericarditis
Retroperitoneal fibrosis
Coeliac spurae
Whipple's disease
Retractile mesenteritis
Traumatic
Blunt (including Battered Child Syndrome)
Shear force to the root of the mesentery
Penetrating
Obstructive
Adhesions
Volvulus
Intussusception
Aortic aneurysm
Haemodynamic
Cirrhosis
Right heart failure
Dilated cardiomyopathy
Jugular, innominate, left subclavian, or portal vein thrombosis
Nephrotic syndrome

peritonism in 1984.

In his review of the literature, Goldfarb discussed three other cases of chylous effusion which were likely secondary to pancreatitis. Acute abdominal pain and peritonism were not features of these presentations. Since 1984 few cases of pancreatitis associated chylous ascites have been described. In 1999, Ben-Ami *et al*^[5] described acute chylous ascites secondary to acute pancreatitis. This was diagnosed during elective cholecystectomy and did not present with symptoms of peritonitis. In 2006, Chuang *et al*^[6] described a case of hypertriglyceridemia-associated acute pancreatitis with chylous ascites in pregnancy. In this

Table 2 Characteristics of ascitic fluid in chylous ascites^[1]

Colour	Milky and cloudy
Triglyceride level	Above 200 mg/dL (2.28 mmol/L)
Cell count	Above 500 (predominance of lymphocytes)
SAAG	Below 1.1 g/dL
Cholesterol	Low (ascites:serum < 1)
LDH	Between 110-200 IU/L
Culture	Positive in some cases of tuberculosis
Amylase	Elevated in cases of pancreatitis
Glucose	Under 100 mg/dL
Cytology	Positive in some cases of malignancy

review there appeared to be a lack of correlation between the formation of chylous ascites and the severity of the pancreatitis.

Aalami proposed two mechanisms believed to play a role in the development of acute chylous ascites in the setting of pancreatitis. These are: the compression of lymphatic channels by an inflamed pancreas and the direct damage of channels by pancreatic enzymes^[3].

Clinical features

Chronic chylous ascites frequently presents with progressive and painless abdominal distension. As with other types of ascites, respiratory embarrassment is a common feature secondary to diaphragmatic splinting. Constitutional symptoms such as anorexia, weakness and malaise are very common, but non-specific^[1]. Other features include abdominal pain, weight loss, diarrhoea and steatorrhoea, malnutrition, oedema, enlarged lymph nodes, early satiety, fevers and night sweats^[1].

In cases of acute chylous ascites symptoms of anorexia, nausea, vomiting and severe abdominal pain are reported. A high fat meal has been reported in the literature as a common precipitant in the development of symptoms. Examination findings of peritonism have been described in the literature. Interestingly, symptoms are often maximal in the right iliac fossa^[7] and most likely a result of pooling of chylous fluid in the right paracolic gutter, mimicking acute appendicitis. Three cases have been reported in which acute chylous peritonitis presented clinically with acute appendicitis. In two cases, the patient underwent open appendectomy, while, in the third patient a midline laparotomy was performed for what was suspected to be appendicitis complicated by appendiceal perforation. In all three cases a white milky fluid was found in the peritoneal cavity, biochemical assessment of this fluid confirmed chylous ascites. The appendix was normal and exploration of the abdomen could not find any cause for the acute chylous effusion in all of the three cases described^[7-9].

Diagnosis

Laboratory testing is rarely useful. The white cell count may be elevated, but other findings are non-specific^[10]. Radiological investigations are of limited benefit although CT of the abdomen has been reported as being useful in identifying pathological lymph nodes and masses and in determining the extent and localisation of the fluid.

Paracentesis is the most useful diagnostic test. Typically chyle has a cloudy and turbid appearance. Table 2 shows the characteristics of ascitic fluid in chylous ascites.

Blood tests including a complete blood count, electrolytes, liver function tests, total protein, albumin, lactate dehydrogenase (LDH), triglycerides, cholesterol, amylase and lipase should be preformed but are by no means diagnostic.

Management

The underlying cause should be addressed whenever feasible. In patients with an acute abdomen, immediate exploration should be performed^[9]. Laparotomy usually allows a definitive diagnosis and provides an opportunity to address the underlying cause.

In chronic chylous ascites which cannot be managed surgically, the goals of treatment are (1) the maintenance of adequate nutrition, (2) decreasing the rate of chyle formation and (3) correcting the underlying disorder^[3]. Considerable controversy exists regarding the effectiveness of a high-protein, low-fat diet with medium-chain triglycerides and diuretics, or total parenteral nutrition (TPN) alone as a means to reducing chyle formation. Guidelines for management published by Aalami in 2000 recommend the commencement of TPN only if no improvement is observed after three weeks on a low-fat, medium-chain triglyceride diet. Multiple case reports describe the use of octreotide in the management of chylous ascites. Somatostatin receptors have been described in the lymphatic vessels of the intestine and it may be that octreotide is effective in managing chylous ascites because it helps to decrease lymph flow through these vessels. The use of octreotide with TPN for the treatment of chylous ascites has been described with clinical improvement in the ascites and reduction in TPN requirements and paracentesis frequency^[11].

In patients with a large amount of ascites a large volume paracentesis to relieve discomfort and dyspnoea can be performed and repeated as needed, however, the risk of infection and fat emboli should be noted^[9]. Peritoneo-venous shunting may be an option, although this is controversial. Although there is some evidence of high initial success rates after insertion, as high as 75%^[3], these shunts carry a high rate of complications which include fever, sepsis and DIC. The high viscosity of chyle has rendered shunt patency disappointing with an eventual occlusion rate approaching 100%.

In conclusion, acute chylous ascites can present with symptoms and signs of peritonism and can mimic acute appendicitis. Chylous effusions, both acute and chronic, are a complication of pancreatitis and may confuse clinical assessment and diagnosis.

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