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Case Report

CASE REPORT

Concomitant fungal peritonitis and high ascitic amylase as a rare manifestation of gastric perforation

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Abstract

High ascitic amylase concentration has been reported to be a characteristic of pancreatic ascites. However, values greater than 2000 U/l can also be seen in intestinal perforation. Fungal peritonitis is a serious entity that could also be caused by hollow viscous perforation. Herein we report a 22-year-old woman with epigastric pain, imitating an acute pancreatitis, and abdominal distention. Laboratory and radiological investigations revealed a high ascitic Amylase level with secondary fungal peritonitis due to gastric ulcer perforation. This case highlights the importance of careful clinical evaluation and a multi-disciplines approach in patients with high ascitic Amylase levels especially in limited-resources areas in order not to miss a diagnosis in which a surgical approach can be lifesaving. To the best of our knowledge, this is the first reported case of concomitant very high ascetic Amylase level and fungal peritonitis as a manifestation of gastric perforation.

INTRODUCTION

Markedly elevated ascitic amylase values have been reported to be a characteristic of pancreatic ascites. However, values greater than 2000 U/l can also be seen in intestinal perforation [1, 2].

Fungal peritonitis (FP) represents approximately 12% of all cases of peritonitis, with Candida being the most common cause. Patients with hollow viscus perforation are at high risk of developing Candida peritonitis (CP); which has very serious consequences and a mortality rate estimated between 20 and 70% [3]. Establishing the diagnosis of CP is a challenge and the treatment is still controversial [4, 5].

CASE REPORT

A 22-year-old female was admitted to the emergency room for epigastric pain that radiated to the right and left hypochondriac regions and to the back. It started 5 days before admission and was accompanied with bilious vomiting, fever, chills and gradual abdominal distention.

The patient had a history of a normal vaginal delivery to a healthy baby ten days before admission. Patient's medications included mineral supplementary for iron deficiency anemia and intramuscular Diclofenac Sodium for analgesia after delivery. No history of smoking or Alcohol consumption was reported.

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By the time of admission, the patient's vital signs were as follows: Blood Pressure 110/70 mmHg, Heart Rate 116 beats/min, Respiratory Rate 22/min, and Temperature 39.5°C. On a scale from 0 to 10 the patient scaled her pain at (10). Abdominal examination revealed epigastric tenderness and shifting dullness. The rest of the physical examination was normal.

Laboratory tests showed elevated serum amylase level (370 U/l) so a primary diagnosis of acute pancreatitis (AP) was made and the patient was admitted to the gastroenterology department.

The abdominal ultrasound revealed an intermediate amount of ascites within the abdominal cavity. Sterile ascites paracentesis was performed, the gross appearance was turbid and the chemical laboratory tests showed: glucose: 8 mg/dl, albumin: 0.5 mg/dl, LDH: 1846 UI/dl, total protein: 0.7 mg/dl, amylase: 2700 U/L. The direct cytology examination showed budding yeasts. The initial ascitic cell count couldn't be performed because the Neubauer Counter was covered with budding yeasts, but later analysis revealed a white cell count of (7000 cell/µl). Repeated analysis to determine the exact neutrophil count reported widely variable results due to dense budding yeasts in the examined samples. However, all the reported results had a neutrophil count of (>250 cell/µl). Empirical therapy with Cefotaxime and Metronidazole was started.

Culture was performed on Blood Agar, EMB Agar and Sabouraud Dextrose Agar. Direct examination of the growth and Germ Tube Test revealed the growth of Candida. Fluconazole was added to the empirical therapy.

Because our patient was young with a normal medical history, there was a high suspicion of visceral perforation as a reason for FP, so a non-enhanced abdominal CT scan due to renal impairment was performed. It revealed an abundant ascitic fluid within the peritoneal cavity and pelvis with pneumoperitoneum around the right hepatic lobe and around the spleen. (Fig. 1a and b).

An emergent laparotomy was performed. A perforated gastric ulcer in the anterior aspect of the pre-pyloric region measuring (2.5 cm) was discovered (Fig. 2). The perforation closure was undertaken with a Graham omental patch. Biopsies from the ulcer margins were negative for *Helicobacter pylori* (*H. pylori*). Bacterial culture of the abdominal fluid that was aspirated during surgery (after 36 hours of treatment with Fluconazole) revealed the growth of *E. coli* and *Klebsiella* with no residual sign of yeasts.

After four days of surgery, abdominal drainage fluid Amylase was 95 U/l, significantly lower than the value recorded on the day of surgery (4717 U/l).

The patient was treated with Meropenem and Linezolid along with Fluconazole for 14 days and was discharged after full recovery with a given prescription of oral Omeprazole at 20 mg twice daily and *H. pylori* eradication therapy.

In the follow up visits after 1–2 weeks of discharge, Clinical and radiological examination were within normal limits with well healing abdominal surgical wound.

DISCUSSION

An ascites/serum amylase ratio of >5 was reported to be a characteristic of pancreatitis. However, elevated non-pancreatic ascitic Amylase concentration can be seen in small bowel perforation, ischemia and mesenteric thrombosis. Thus, to the best of our knowledge, it is not widely used as a confirmed diagnostic criteria [1]. Our patient's ascites/serum amylase ratio was 7.29 at the time of admission which correlated with AP. The diagnosis of Gastric Perforation in our patient was a challenge in the absence of abrupt onset of abdominal pain and peritoneal irritation signs. However, with the diagnosis of FP and previous usage of Diclofenac Sodium suspicion of intestinal perforation as a differential diagnosis of AP was raised and confirmed later with abdominal CT scan. To the best of our knowledge, only three cases with markedly elevated ascitic Amylase (2803.4, 4688.4, and 3352 U/L) due to perforated gastroduodenal ulcer were reported by Amerson *et al.* [6] Serum Amylase values were 92.4, 179.2 and 221.7 U/l, respectively. None of these cases had a concomitant FP.

While neither Lipase nor MRI were available in our hospital due to wartime limited resources in order to complete an extensive pancreatic evaluation, remarkable clinical and laboratory improvement after the surgery made a concomitant AP less likely.

CP is relatively common in patients with upper gastrointestinal (UGI) perforation. Although the treatment is still controversial, antifungal therapy was found to minimize the overall morbidity and mortality [5, 7]. Our patient was treated with Fluconazole as soon as FP was established and fungal culture was negative after 72 hours of treatment. Surgery is an important mean of source control in patients with secondary peritonitis [3]. A simple closure of the perforation with Graham Omental Patch was the procedure of choice for our patient followed by *H. pylori* eradication therapy which was found to significantly reduce ulcer recurrence and to improve the healing rate of peptic ulcers [8, 9].

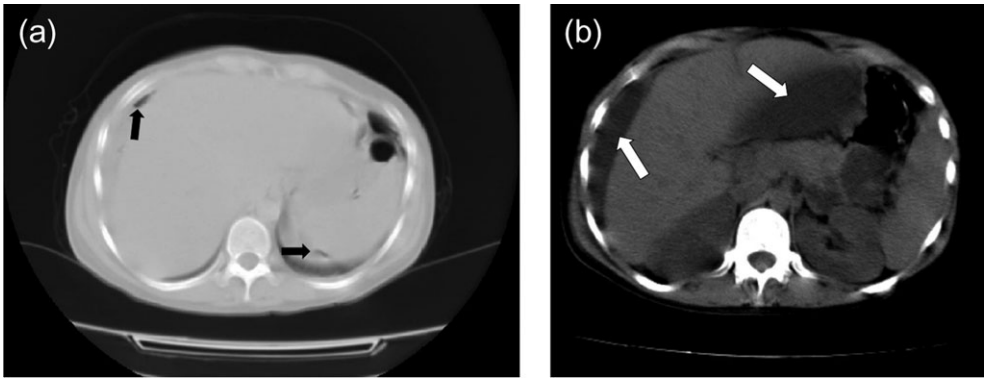

Figure 1: **(a)** Abdominal CT scan showing pneumoperitoneum around the right hepatic lobe and around the spleen (black arrows). **(b)** Abdominal CT scan showing ascitic fluid within the peritoneal cavity (white arrows).

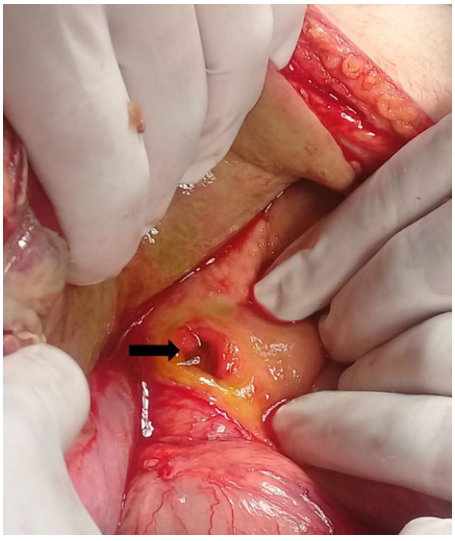

Figure 2: Photo of patient's 2.5 cm perforated gastric ulcer in the anterior aspect of the pre-pyloric region (black arrow).

The most important risk factors for peptic ulcer disease are *H. pylori* infection and usage of nonsteroidal anti-inflammatory drugs (NSAIDs). Despite histological examination negativity, *H. pylori* infection couldn't be totally ruled out because biopsies were not obtained during elective endoscopy [8]. Chronic NSAIDs usage can lead to peptic ulcer perforation, but short-term usage has also been described as a risk factor for UGI complications [8–10], which in this case might be the cause. In conclusion, gastric perforation may mimic an AP, and markedly elevated ascitic Amylase may be found in both conditions. That's why a careful and multi-disciplines approach is crucial in order not to miss a diagnosis in which a surgical approach can be lifesaving. Additionally, this case highlights the importance of medical history and close clinical evaluation in wartime and limited-resources areas.

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CONFLICT OF INTEREST STATEMENT

None declared.

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No approval was required.

CONSENT

The patient has given her informed consent to the participation and publication of this article and the figures related to it.

GUARANTOR

All authors nominated L. Alasadi as the guarantor for the Article.

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