

## Sustained Amylasemia in a Patient with Newly Diagnosed Alcoholic Cirrhosis; “A Clinical Picture That Is Worth a Thousand Words”.

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Pancreatic ascites; Alcoholic cirrhosis; Chronic pancreatitis; Pancreatic duct leakage; Amylasaemia

## 1. Abstract

Inflammation and distortion of the liver parenchyma leading to cirrhosis can be triggered by various causes. Alcohol is one of the commonest culprit factors among viruses, medications, toxic metals or autoimmune mechanisms. It has been observed though, that alcohol may injure not only the liver but the pancreatic tissue as well, either simultaneously or at a deferred time.

This perplexes the clinical picture if both organs are diseased and each manifestation cannot be always clearly explained. Therefore, recalling relevant pathophysiological mechanisms may help clinicians ending up to the right diagnosis and subsequently selecting the appropriate treatment. Herein, we present a 50 years-old man, with a history of gallstones disease and alcohol abuse, who suffered an episode of acute pancreatitis. Liver cirrhosis was also present and his hospitalization was complicated by pancreatic ascites. Therapeutic abdominal paracenteses, total parenteral nutrition, along with somatostatin infusions were applied. The patient was medically observed for a long-time period. The pancreatic ascites resolved spontaneously, without the need of any interventional therapeutic approach.

## 2. Introduction

Pancreatic ascites is a rare complication of chronic pancreatitis, pancreatic trauma and to a lesser extent of acute pancreatitis [1].

Pancreatic ascites is either the consequence of disruption of the pancreatic duct, or that of indirect communication through a pre-existing pseudocyst leading to ascitic fluid leakage into the peritoneal cavity. Pleural effusion is also a common accompanying finding. The analysis of the ascitic fluid reveals exudative fluid but in order to establish the diagnosis of pancreatic ascites, it should be checked the presence of amylase in it [2]. The pathophysiological “clue” is the leaking of pancreatic fluid into the peritoneum and this can be achieved by different mechanisms. Chronic pancreatitis constitutes a 78% of ductal disruption whereas pancreatic trauma a 9%. It is not the result of a malignant pancreatic disease [3]. Due to the rarity of this clinical condition the incidence is not known, while it has been reported that men are affected more than women, and the ages that are overwhelmed are between 20 and 50 years [1]. Associated clinical signs and symptoms include; abdominal distention, early satiety and weight loss [4]. Presence of pancreatic fluid in abdominal cavity is indicative of diagnosis; amylase that is greater than serum amylase and elevated protein levels (>3g/dL). If the pancreatic enzymes are secreted in an active state or become activated into the abdominal cavity, then this could lead to potential lethal complications [5]. Conservative management includes parenteral nutrition, ascitic fluid paracentesis and delivering intravenously octreotide or somatostatin [6]. Interventional strategies are also applied. Endoscopic retrograde cholangiopancreatography (ERCP) exhibits where the pancreatic duct leakage is located,

allowing thus highly skilled clinicians to insert a stent at the level of the duct's deficit, when this is appropriate [7]. Our internal medicine team, recently encountered an enigmatic case-presentation, where a 50 years-old man suffered an episode of acute pancreatitis. Synchronously, he developed ascitic fluid and he was diagnosed with non-compensated liver cirrhosis. Analysis of the fluid affirmed that pancreatic ascites had developed. The presence of a pseudocyst (less than 4 cm in diameter) was demonstrated through the Magnetic resonance imaging (MRI) of the abdomen and magnetic resonance cholangiopancreatography (MRCP). Conservative management was beneficial for our patient.

### 3. Case Presentation

A 50-years-old man was transferred at the department of our internal medicine clinic from an urban hospital due to complicated acute pancreatitis by ascites and lower extremity edema. The patient initially was admitted to the former hospital due to continuous epigastric pain and the laboratory results displayed high serum and urine amylase; 1.100 U/L and 25.360 U/L, respectively. Abdominal computed tomography with intravenous administration of iodine contrast was performed, where the presence of gallstones within the gallbladder, large volume of ascitic fluid and a dilation of pancreatic duct (0.8cm) close to the head of the pancreas were displayed. He had no significant past medical history, except for a known biliary sludge, and he was not taking any medication. Besides, his social history was significant for alcohol use disorder and smoking (45p/y). He was subsequently transferred to our clinic for further investigation and treatment. His vital signs, upon his arrival at the emergency department, were the following; blood pressure 105mmHg over 80mmHg, heart rate 100bpm, oxygen saturation 98% on room air and temperature of 36.7C. The patient was alert and oriented to time and place. Coarse neurological and musculoskeletal exam revealed no pathological findings. On cardiac auscultation, the heart sounds were audible, rhythmic and no murmurs or gallops were noted. There was no jugular venous distention, while bilateral lower extremity edema was present. Reduced respiratory sounds at the bases of the lungs were noted during lung auscultation, while abdominal examination revealed ascites and hepatomegaly. The abdomen was diffusely tender to palpation and bowel sounds were present. Palmar erythema and spider angiomas were not present. A 12-lead electrocardiogram showed sinus rhythm. On the chest X-ray no cardiomegaly was noted and the lung parenchyma was normal; however, pleural effusions at the bases bilaterally, were present. The abnormal laboratory findings were; WBCs 13.890, Hct 38.6, Hb 12 mg/dl, (MCV=100.6 fL), PLTs 575.000, Creatinine 0.6mg/dL, Na 131 mmol/L, gGT (CK) 135 U/L, Amylase 2542 U/L, C-reactive protein (CRP) 90.10 mg/L. Therapeutic paracentesis of the ascitic fluid was ordered and analysis of it showed exudate (protein of ascitic fluid=3.6mg/dL) and amylase up to 12.000 U/L. Spontaneous bacterial peritonitis

was excluded, while cytology did not display any malignant cells. The patient was admitted to the floor and a repeated computed tomography was ordered. The results were consistent with cirrhotic liver, swelling and heterogeneity of the head of the pancreas and ascites. Upper endoscopy of the gastrointestinal tract showed small esophageal varices and portal gastropathy. Due to lower extremity edema, cardiac ultrasound was commanded where ejection fraction was preserved, however diastolic dysfunction and mild mitral regurgitation were recorded. The patient was started on diuretics; furosemide 40mg and spironolactone 100mg orally, complexes of vitamin B, folic acid and low-dose propranolol. He was initially fed orally and while his clinical course was improving, he re-developed another episode of acute pancreatitis, a week later, with spikes in serum and urine amylase, as well as ascitic amylase. MRI and MRCP was immediately ordered and the findings were the following: liver cirrhosis, cholelithiasis, normal diameter of the common bile duct with no gallstones, ascitic fluid and an enclosed fluid collection adjacent to the lesser sac (40mm to 45mm in diameter) that exerts pressing phenomena to the stomach. Thus, it was concluded that the pancreatic duct was in direct communication with a pseudocyst and through this cyst, the fluid was diverted into the abdominal cavity. At that time, there was a dialogue with the gastroenterologists and it was concluded that a patient is suffering a rare complication of acute pancreatitis, named pancreatic ascites. There was a lot of conversation regarding treatment management; conservative versus invasive through stenting with endoscopic retrograde cholangiopancreatography (ERCP). It was decided to follow the conservative approach first. Hence, the patient continued the pre-existing treatment, with the exception of withholding the oral feeding. Total parenteral nutrition was started, rich in proteins and low in fat content. Intravenous somatostatin was also administered in order to decrease the exocrine function of the pancreas. The ascitic and serum amylase levels were monitored daily. The ascitic fluid remarkably subsided after repetitive paracenteses. The patient remained afebrile, the amylase levels (serum and ascitic) along with inflammatory markers returned to normal and a repeated computed tomography showed minimum accumulation of fluid within the abdominal cavity, confined to the perihepatic space. At that time the parenteral nutrition switched to oral with an excellent tolerance by the patient. He never developed abdominal pain or discomfort and he eventually discharged after almost a month of hospitalization.

### 4. Discussion

Acute pancreatitis is a common disease, especially in patients with gallstone disease or alcohol abuse. Nonetheless, pancreatic ascites is an exceptionally rare complication of acute pancreatitis. Due to its rarity, the exact incidence of pancreatic ascites has not been well recorded. The linked risk factors are those alike in acute pancreatitis; gallstones and alcohol [8]. It has been delineated mostly

in men [1]. Peripancreatic fluid collection is the most typical imaging finding that results during an episode of acute pancreatitis. If they do not resolve spontaneously, pancreatic pseudocysts are organized. Fluid collections do mainly locate in the lesser sac of the abdomen. They do possess a well-circumscribed wall and contain pancreatic enzymes, blood and necrotic tissue. Pseudocysts are usually encountered in cases of chronic pancreatitis [9]. Nonetheless, our patient had no clinical signs and symptoms of chronic pancreatitis; pancreatic calcifications, steatorrhea, and diabetes mellitus [10] and indeed the development of the pseudocyst in this patient, after the first reportable episode of acute pancreatitis is undoubtedly scarce. There are two main pathophysiological mechanisms that predispose to pancreatic ascites development; pancreatic duct injury due to inflammation along with fistula formation and pancreatic pseudocyst formation. Both processes result in leakage of pancreatic chyme into the peritoneum [4]. In the case of pseudocyst formation, the scenario is that pseudocysts tend to have a weak and breakable wall, especially in the case of chronic pancreatitis. Pancreatic secretions are directed from the disrupted duct into the pseudocyst and then through pseudocyst's fragile wall into the peritoneum [11]. In the absence of pseudocyst, a communication between disrupted pancreatic duct and peritoneum is created through a fistulous pattern. Depending on the location of the fistula tract there will be different clinical manifestations; for instance, ascites will be the result of emptying pancreatic secretions into the abdominal cavity through a fistula that is arising from an anterior pancreatic duct disruption, whereas fistulas from a posterior pancreatic duct opening can cause pleural effusions through the aortic or esophageal hiatus or even through the dome of the diaphragm [12]. As we have already mentioned, pancreatitis is a significant cause of pancreatic duct injury, with chronic pancreatitis being more common than acute. Other etiologies include abdominal trauma, lithiasis, ampullary stenosis and iatrogenic, e.g. ERCP. A patient of middle age with a history of gallstone disease or alcohol use disorder who is presenting with increased abdominal girth and weight loss after an episode of acute pancreatitis should raise suspicion of pancreatic ascites. The medical history is most of the times negative for a chronic inflammatory illness [13]. Elevated levels of amylase ( $>1000\text{IU/L}$ ) and protein ( $>3\text{g/dL}$ ) of the ascitic fluid portray this particular disease. Moreover, the calculated serum-ascites albumin gradient (SAAG) is less than  $1.1\text{g/dL}$  [14]. The site of pancreatic duct, concerning where the leakage is coming from, may be visualized through ERCP and/or secretin augmented MRCP. The management of pancreatic ascites splits into medical, endoscopic and surgical therapy. At a 30% to 50% of cases, complete resolution of symptoms has been achieved through conservative measures; nothing per mouth, total parenteral nutrition, somatostatin or octreotide infusions, repeated paracentesis. If these measures fail, then diagnostic and therapeutic intervention

with ERCP may be the next step [15]. Stenting of the pancreatic duct decreases the pressure in the ductal part and redirects pancreatic secretions into the small bowel, allowing the traumatic region to heal. However, this is a challenging process, since it is an interventional approach that carries many risks. If both aforementioned measures fail or the injury occupies almost the whole duct, then surgery may be the last therapeutic step; partial pancreatectomy or pancreaticojejunostomy [16]. Our patient responded well to medical therapy alone and hopefully there was no need for endoscopic intervention, which by itself poses the patient at an increased risk for acute post-procedural pancreatitis.

## 5. Conclusion

The case-report that was just presented is very instructional and educative. The patient was diagnosed with acute pancreatitis as well as liver cirrhosis. Fluid into the abdominal cavity was developed, hence the cirrhosis was already non-compensated. However, the sustained mild abdominal pain, the intolerance to oral feeding, the prolonged amylasemia along with the presence of amylase within ascitic fluid according to its analysis, were very indicative of pancreatic ascites. MRCP denoted pseudocyst formation. Through pancreatic duct and subsequently communicating pseudocyst, the pancreatic fluid containing enzymes were drained into the abdominal cavity. The concluding diagnosis was pancreatic ascites. A paradoxical part of this puzzle-vignette however, was that our patient developed pancreatic pseudocyst in the absence of chronic pancreatitis, which is usually the prerequisite circumstance. This is why it is argued that in medicine not all the clinical cases do follow the rules by the book.

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