Pancreatic ascites and pancreatic pleural effusion: pathogenesis and computed tomographic demonstration

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We report two cases of pancreatic ascites, with a proven pancreatic pleural effusion. Both patients were alcoholic abusers and heavy smokers who presented with upper abdominal pain and evidence of ascites on the admission physical examination. The contrast enhanced CT scan revealed massive ascites with evidence of pancreatitis. Acute pancreatitis with unusual complication of pancreatic ascites was diagnosed and proven by demonstrating high level of amylase in the ascites. The first case was delay diagnosed and resulted in death while the second case was early diagnosed, given appropriate treatment and complete recovery.

Key words: Pancreatitis, Pancreatic ascites, Exudative ascites.

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รายงานผู้ป่วย 2 รายที่ได้รับการวินิจฉัยว่ามีภาวะของน้ำในช่องท้องและน้ำในช่องทร��อก
เนื่องจากการร้วมของน้ำอย่างติดอยู่ ผู้ป่วยทั้ง 2 รายมีประวัติสุขภาพ ที่ผ่านมาไม่คุ้น
ด้วยอาการสำคัญคือ ปวดท้องด้านบนซ้าย ตรวจจราจรกายภาพ พบมีน้ำในช่องท้อง และในช่องทรวง
อกซ้าย ผู้ป่วยทั้ง 2 รายได้รับการวินิจฉัยเบื้องต้นว่าเป็นโรคตับอ่อนอักเสบเฉียบพลัน และได้รับการส่ง
ตรวจทางเอกซเรย์คอมพิวเตอร์ เพื่อหาสาเหตุของน้ำในช่องท้อง และน้ำในช่องทรวงออก ผลการตรวจ
ทางเอกซเรย์คอมพิวเตอร์พบมีน้ำในช่องท้องเป็นจำนวนมาก ร่วมกับมีน้ำในช่องทรวงออกซ้าย และมี
ภาวะตับอ่อนอักเสบเฉียบพลัน แพทย์ผู้ทำการรักษาได้ทำการรักษาผู้ป่วยในช่องท้องและในช่องทรวงออกซ้าย
โดยใช้วัสดุที่มีประสิทธิภาพสูงสุด และได้รับการตรวจวินิจฉัยทางทรวงปฏิบัติการมี เพื่อต้องการตระเวนของน้ำอย่างติดอยู่ ผลการตรวจวินิจฉัยทาง
ทรวงปฏิบัติการมีพบว่ามีระดับของน้ำอย่างติดอยู่ในช่องทรวงใบลานสูงมาก ผู้ป่วยทั้ง 2 ราย
ได้รับการวินิจฉัยว่าเป็นโรคตับอ่อนอักเสบเฉียบพลัน ร่วมกับมีการทรวงออกรัศมีโดยผู้ป่วยรายแรกมีสัด
น้ำในช่องท้องและน้ำในช่องทรวงออกซ้าย เนื่องจากการรัศมีของน้ำอย่างติดอยู่ ผู้ป่วยรายแรกมีการรัศมี
เนื่องจากเกิดภาวะติดเชื้อของน้ำในช่องท้องและทรวงออก ผู้ป่วยรายที่สองได้รับการรักษาที่ทำฟังก์
และอาการดีขึ้นจนหายเป็นปกติในที่สุด
Pancreatic ascites is well established as an uncommon complication of chronic relapsing pancreatitis with occasionally presented in severe acute pancreatitis. The pathogenesis is secondary to disruption of pancreatic duct or ruptured pancreatic pseudocyst. Although the pancreatic ascites has been often cites clinically, it has not been well documented in radiological literature.

Case report

Case 1

A 35-year-old man, a heavy drinker and smoker who presented with left upper abdominal pain, relived by forward bending of the body. The vital sign and physical examination revealed low-grade fever, slightly increased pulse rate, mild tachypnea, moderately abdominal distension with generalized abdominal tenderness and evidence of ascites. The biochemical laboratory test revealed high level of serum amylase (2,032 U/L, normal 0-220 U/L) and the serum lipase (1,408 U/L, normal 0-190 U/L). Peritoneal tapping was performed which showed straw-colored ascitic fluid. The ascitic fluid was later confirmed to be exudative (specific gravity 1.030, protein 3.7g %, albumin 0.97g %). However the bacteriologic culture and cytologic study of the ascitic fluid was negative. The contrast enhanced CT scan was performed in order to verify the possible etiology of the ascites. It showed massive ascites with multiple fluid collections locating within the lesser sac and along the gastrocolic ligament (Fig.1A). The pancreas itself was small with multiple small intrapancreatic fluid collections in its head and tail (Fig.1B). The small bowel loops were retracted and conglomerated towards midline, consistent with exudative nature of ascitic fluid (Fig.1C). Bilateral pleural effusion and atelectasis of both lung bases were also observed (Fig.1D). Based upon the clinical information and CT findings, acute

Figure 1. 35-year-old man with alcoholic pancreatitis and pancreatic ascites.
A. Multiple fluid collections within the lesser sac and along the gastrocolic ligament (arrow). Intraperitoneal free fluid is noted within the right perihepatic and hepatorenal fossa (Morrison's pouch).

Figure 1B. The pancreas is rather small size with small intrapancreatic fluid collections in its head and tail (arrow). Peripancreatic fat stranding is noted.
Figure 1C. Massive ascites with conglomerated bowel loops toward the midline. The peritoneal tapping was performed with demonstrating high level of the ascitic amylase. 

Pancreatitis with unusual complication of pancreatic ascites was diagnosed. To confirm diagnosis, repeat peritoneal tapping was performed. The ascitic fluid was found to have a very high amylase level (26,170 U/L) which was diagnostic for pancreatic ascites. The fluid was negative for malignancy or other infectious etiologies. The pleural effusion was not tapped for amylase level. The endoscopic retrograde cholangiopancreatography (ERCP) was performed which demonstrated leakage of distal pancreatic duct. The patient was treated conservatively but developed clinically sepsis and infected intra-abdominal fluid collection was considered. Therefore, the laparotomy was performed for peritoneal lavage and debridement. After operation, the patient developed renal failure and eventually expired.

Case 2

A 49-year-old man, with history of acute pancreatitis 3 months previously. He is also a heavy drinker and smoker. This admission, he presented with severe epigastric and left upper abdominal pain.

Figure 1D. CT scan section of the lower chest level. Bilateral pleural effusion with bilateral basal lungs atelectasis. The pleural effusion was not tapped for amylase level, therefore pleural effusion was not proved. 

Figure 2. 49-year-old-man with alcoholic pancreatitis, accompanying with pancreatic ascites and pancreatic pleural effusion.

A. The pancreas is of small size with focal peripancreatic fluid around its body (arrow). Noted is ascites in the right perihepatic region.
Figure 2B,C. A 0.5 cm proximal pancreatic duct stone (white arrow), causing dilatation of the distal pancreatic duct (black arrow).

Figure 2D. Moderately intraperitoneal free fluid in the right perihepatic region (RH) and fluid collection within the lesser sac (LS). Peritoneal fluid contained high level of amylase.

Figure 2E. Minimal left pleural effusion is observed (white arrow). The pleural fluid was later confirmed to have high level of the amylase enzyme.

The vital sign showed low-grade fever with slightly increased pulse rate and mild dyspnea. The physical examination revealed decreased breath sound and vocal resonance at left lower lung, mild abdominal distension, markedly left upper abdominal pain with evidence of ascites. The biochemical laboratory test revealed high level of the serum amylase (4,830 U/L, normal 0-220 U/L) and serum lipase (11,790 U/L, normal 0-190 U/L). The contrast enhanced CT scan showed atrophic pancreas with peripancreatic fluid around its body (Fig. 2A). There is a 0.5 cm stone in the proximal pancreatic duct causing dilatation of distal pancreatic duct (Fig.2B,C). Moderately intra-abdominal free fluid with fluid collections within the lesser sac is also observed (Fig.2D). Moderately left pleural effusion is noted (Fig.2E). The peritoneal
tapping was performed to establish the etiology of ascites. It showed serosanguinous fluid which was confirmed to be exudative (specific gravity 1.031, protein 5.5 g%). The ascitic amylase level was high (86,730 U/L) which was compatible with pancreatic ascites. The left thoracentesis was also performed which showed turbid yellow fluid with high level of pleural fluid amylase (12,690 U/L), consistent with left pancreatic pleural effusion. The endoscopic retrograde cholangiopancreatography (ERCP) showed complete obstruction of proximal pancreatic duct, secondary to the proximal pancreatic duct stone. The endoscopic sphincterotomy with stone extraction was attempted but it failed. Therefore, laparotomy was performed, and the stone was successfully removed. The patient showed good recovery and finally discharged home.

Discussion

Pancreatic ascites with or without accompanying pancreatic pleural effusion is well established as the complication of chronic relapsing pancreatitis.\(^{1}\) Occasionally it is a complication of severe acute pancreatitis.\(^{2}\) It is an uncommon condition and constitutes less than 5% of the cases of ascites.\(^{3}\) Although it is often cited clinically in the medical literature, this complication is barely described in the radiological literature.

Pathologically, pancreatic ascites is well documented to be secondary to the disruption of main pancreatic duct or rupture of pancreatic pseudocyst causing leakage of the protein-rich pancreatic fluid into the peritoneal cavity.\(^{4-5}\) As a result of this protein leakage, change of the systemic capillary and peritoneal surface onotic pressure as well as activation of renin-angiotensin cascade are postulated to produce the exudative ascites.\(^{6}\) Pancreatic duct disruption tends to occur in underlying chronic pancreatitis because of small, stenotic ductal lumen which is a subject to high internal pressure, once there is superimposed acute inflammation or obstruction of pancreatic duct.\(^{1,7}\) Most cases of pancreatic duct disruption as a complication of pancreatitis are secondary to alcoholic abuse. The mean age at presentation is 43 years old with a wide range, from young children to elderly adults. The majority of patient is male, usually with a 3:1, male: female ratio.\(^{8,9}\) Similar to others, both of our patients are alcoholic abusers. One case (case 1), pancreatic duct disruption is presumed to be secondary to superimposed acute inflammation and the other case (case 2) is secondary to proximal pancreatic duct stone.

Pancreatic pleural effusion, accompanying pancreatic ascites is postulated to be secondary to transdiaphragmatic pressure gradient between abdominal and pleural cavity. The pancreatic fluid usually moves upward, directly through the dome of the diaphragm into the pleural cavity. The pancreatic pleural effusion is more common in the left sided than the right sided because of the anatomic location of pancreas as the left upper abdominal organ.\(^{10}\) Both of our cases illustrated pleural effusion, accompanying pancreatic ascites. However the pleural effusion was confirmed to have a high level of amylase in only one case (case 2). The other patient, pleural effusion may be a real pancreatic pleural effusion or merely a reactionary fluid from intra-abdominal inflammation.

To our knowledge, computed tomographic findings of pancreatic ascites or pancreatic pleural effusion have not been well documented in the
radiological literature, in spite of many case reported clinically. Part of the reason is probably unawareness of this unusual complication of pancreaticitis by radiologists. Because of the exudative nature of pancreatic ascites, many cases of pancreatic ascites are often attributed to other causes such as carcinomatosis peritonei or tuberculous peritonitis, which are more common than pancreatic ascites.

Eventhough, computed tomography is not specific for pancreatic ascites, awareness of this entity is important, differential diagnosis of pancreatic ascites along with carcinomatosis peritonei and tuberculous peritonitis should be concerned, particularly in patients who have underlying chronic pancreatitis. Once the pancreatic ascites is considered, proven of diagnosis is simply by demonstrating high level of the amylase within the ascites or pleural effusion.\(^{(12)}\)

Delay in diagnosis will usually increase risk for the major complication as well as mortality and morbidity. The major complication is superimposed infection of the ascites or pleural effusion.\(^{(11)}\) This complication is presented in our first case, who results in death. Learning from the first case, the second case was early diagnosed, given appropriate treatment and complete recovery.

In conclusion, radiologists should be aware of pancreatic ascites as a potential cause of intra-abdominal free fluid, particularly in patients with history of alcoholic abuse and chronic relapsing pancreatitis.

References