UNUSUAL CLINICAL AND IMAGING FINDINGS IN PANCREATIC PSEUDOCYSTS

Somrak LOWANITCHAI, MD.

ABSTRACT

Four cases of pancreatitis, with the presence of pancreatic pseudocysts were presented. All were clinically not indicative to be pancreatitis. They came to the hospital with a chief complaint of epigastric pain, left flank mass, enlarge scrotum, and massive bilateral pleural effusion. Sonography and Computed Tomography were performed and revealed pancreatic pseudocysts at the superior recess of omental bursa, subcapsular region of left kidney, in splenic hilum and at area posterior to left lobe of liver anterior to the stomach, which suggested to be in the hepatogastric ligament. Careful observation of the image findings in these cases will be helpful to make a differential diagnosis from other diseases.

INTRODUCTION

Pseudocyst is a fluid collection composing of necrotic material, proteinaceous debris, and enzymatic material that is confined by a fibrous capsule. They vary greatly in size, and are round or oval in shape which develop late, usually after repeated episodes of pancreatitis. At the time of diagnosis,most other signs of acute pancreatitis have already subsided. The incidence of pseudocysts following acute pancreatitis is 2% -4%, and in chronic pancreatitis is 10–15%.[13] Their clinical significance are related to their size and to dangerous complications, such as rupture, bleeding, or abscess formation. In patients with history of heavy alcoholic intake, pseudocyst are often detected in asymtomatic individuals. Spontaneous resolution of pseudocysts can occur simply from resorption of fluid or drainage into a loop of bowel. Internally drainage of pseudocysts may occur if it is greater than 5 cm in size.[4] With modern imaging procedures, the unusual sites of pancreatic pseudocysts seem to be increasing. Followed were 4 patients being sent for sonography and Computed Tomography without clinical symptoms suggesting to be pancreatitis. They presented with mid epigastric pain, left flank mass, enlarge scrotum and bilateral pleural effusion. The first two cases turned out to be pseudocysts at the superior recess of omental bursa, subcapsular pseudocyst of left kidney, and the last two cases, findings were fluid in scrotum from pancreatic ascites, and bilateral pleural effusion from pancreatitis. The sonographic and Computed Tomographic imaging features are described and the possiblitics how the pancreatic secretion entered into these organs are discussed.

CASE ONE

A 45-year-old man presented with a chief complaint of abdominal pain and discomfort at the epigastrium 10 hours before admission and intensified 2 hours before his coming to the hospital. Physical examination showed tenderness at the epigastrium and RUQ of abdomen. Plain abdomen showed only small bowel ileus. Sonographic study revealed low echoic to anechoic

Department of Radiology, Sapprasitthiprasong Hospital, Mueng, Ubonratchathani, 34000 THAILAND.

mass near or at the caudate lobe of liver, size 1.6 cm. (fig. 1). 2 days later, sonography was done due to clinically increasing in abdominal pain. This time, the mass suspected to be in the caudate lobe showed moderately increasing in size to be about 5.0x4.5cm^2 (fig. 2). Other findings in this study were a moderate amount of ascites and bilateral pleural effusion. The pancreas looked normal in size and echogenicity. Computed Tomography at 8th day of admission revealed fluid attenuation mass size 5.0 cm. at the superior recess of omental bursa (fig. 3.) with minimal fluid along the porta hepatis (fig. 4). Mild dilated pancreatic duct and small pseudocyst at the pancreatic body (Fig. 5). Final diagnosis was pancreatic pseudocyst at the superior recess of omental bursa and small amount of fluid tracking along the porta hepatis.

Conservative treatment was done. The followed up sonography every 1 month showed some decreasing in size of the pseudocyst at the superior recess of omental bursa, and finally after 4 months, it showed to have a complete resolution of the pseudocyst.

CASE TWO.

A 32-year-old man was admitted to the hospital from a motorcycle accident. He was in the trauma ward for observation of any abnormal neurological sign. 6th day after admission, he complained to have left side abdominal pain. Physical examination revealed a palpable mass at the left flank and an initial clinical diagnosis was perinephric hematoma. Sonographic study showed a 10 cm. encapsulated fluid collection compressing the upper pole of left kidney. Some internal echo fluid contents were evidence. (fig. 6). Other findings were thickening of the mesentery at the upper abdomen, and only part of pancreatic head was visualized which appeared to have mild prominent duct.

Sonography diagnosis was subcapsular pseudocyst of left kidney VS renal abscess with rupture outside the renal capsule. Computed Tomography showed thin and some part thick wall cystic mass at upper pole of left kidney compressing normal renal parenchyma (fig. 7). Computed Tomography well demonstrated this cystic mass extending from the pancreatic tail (fig. 8) which now can be well seen in detail. There was a mild dilatation of the pancreatic duct with others small cystic masses, size 1-2 cm. at the left psoas muscle and root of mesentery which were also evidence (fig. 9). The diagnosis was subcapsular pancreatic pseudocyst of left kidney with small pseudocysts at the mesentery, and left psoas muscle. Cystogastrostomy was done, and the fluid revealed to have elevated amylase level. The patient eventually recovered after a long post operative course.

4 months later,there is a complication by having an extrapancreatic fluid collection at the left lobe of liver (fig. 10).

CASE THREE

A 32-year-old man with a history of a progressive swelling of the right scrotum about 2 weeks ago was referred for sonography of the scrotum without any other clinical detail. Tentative clinical diagnosis was hydrocele of the right scrotum, and tumour of testis was to be excluded. Scrotal sonography revealed a bilateral hydroceles with a moderate amount of fluid in the rightside and a small amount of fluid in the left side with a normal testis in both sides. (fig. 11). Additional sonography of the abdomen showed focal bulging contour of pancreatic head, size about 3.7cm. This could be focal pancreatitis, or pancreatic head mass (fig. 12), with mild prominent of pancreatic duct and a moderate amount of ascites was seen. Sonography diagnosis was focal pancreatitis or CA head of pancreas. Computed Tomography 3 day after that revealed a focal enlargement of pancreatic head (fig. 13), small pseudocysts at the splenic hilum, mesentery (fig. 14,15) and a small amount of fluid at the left

anterior pararenal space. Computed Tomography diagnosis was focal chronic pancreatitis with small pseudocysts. Follow up sonography 2 months later, the pancreatic head was reduced in size to 1.5cm., no ascites, and still presence of fluid in the scrotal sac, however, decreasing in amount.

CASE FOUR

A-28 -year-old man with a history of dyspnea for 3 months, without fever. Chest film showed right pleural effusion. Thoracocentesis revealed dark reddish color fluid, cell 1500 mm^3 ; PMN 31 % ; LYM 69 % ; Protein 1596 mg %, Sugar 63 mg % (Bs 52 mg %) ; Gram stain and AFB were negative. Pleural biopsy showed chronic pleuritis with abortive granuloma, no definite fungus. At this time anti TB drug was given. 1 month later his symptom was not improve. Chest film still presence of massive right pleural effusion. Intercostal drainage was done. Pleural fluid showed brownish color fluid, Celll 1500 mm3 ; PMN 42% ; LYM 54 % ; Mono 4% ; Protein 3950 mg %, Sugar 83 mg % (Bs 104 mg%) ; Cytology ; bloody with normal mesothelial cell and macrophage, negative for malignancy cell. Pleural biopsy revealed no granuloma, nor malignant cell. He was still treated as TB pleuritis. 2 weeks later he came to the hospital due to progressive dyspnea. Chest film showed bilateral pleural effusion (fig. 16). Sonography of the thorax and abdomen showed bilateral pleural effusion, massive on the left side, anechoic mass size 2 cm. posterior to left lobe of liver. Computed Tomography showed well capsulated fluid collection posterior to left lobe liver anterior to stomach (fig. 17), dilated pancreatic duct (fig. 18).

Sputum AFB for 3 days was negative, pleural fluid showed dark greenish color, cell 310 cm^3, PMN 63 %, LYM 37 37 %, Protein 4120 mg %, Sugar 107 (BS 110), amylase 9710 U/L, Serum amylase 578 U/L (Normal 30-110 U/L)

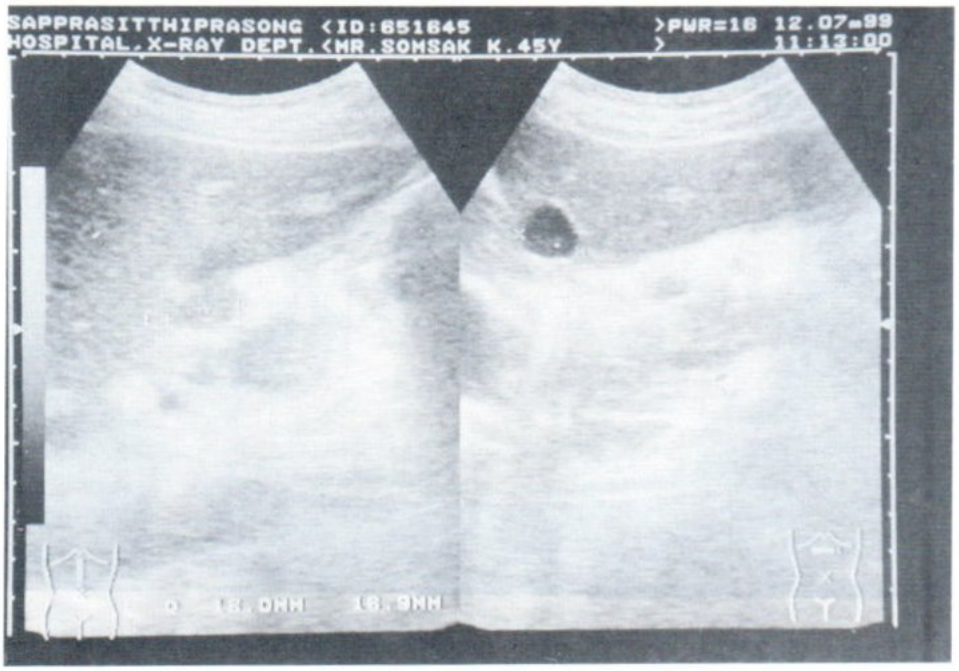

Fig. 1. Low echoic to anechoic mass, well defined border at, or near caudate lobe of liver, adjacent liver parenchyma showing mild low echoic change.

Bs = Blood Sugar

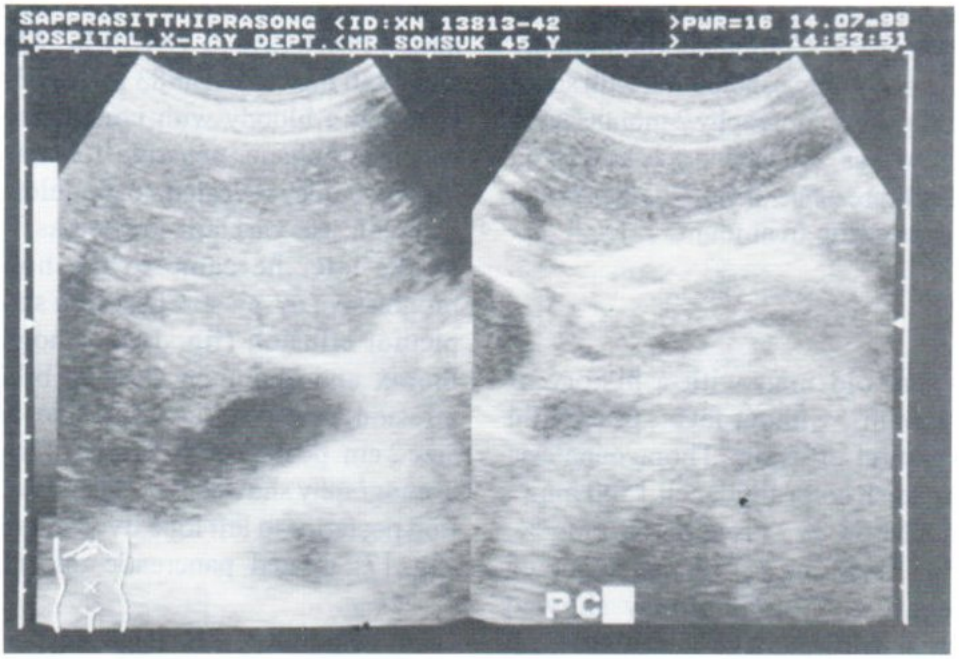

Fig. 2. The low echoic mass was increase in size, pancreas showed normal size and echo, mild prominent pancreatic duct.

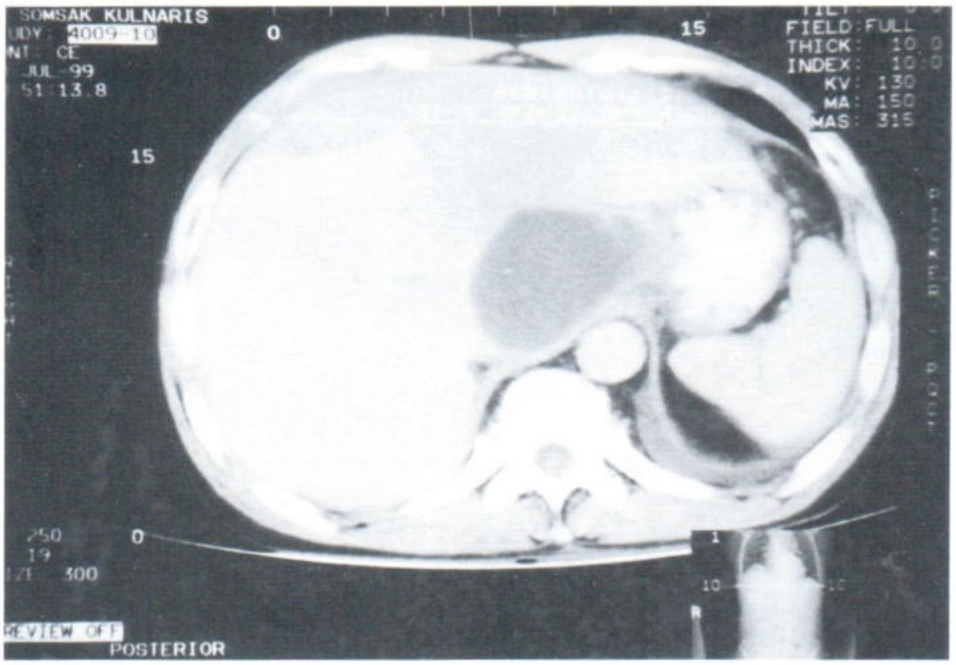

Fig. 3. CT scan with IV contrast enhancement, there is well defined border mass at superior recess of omental bursa.

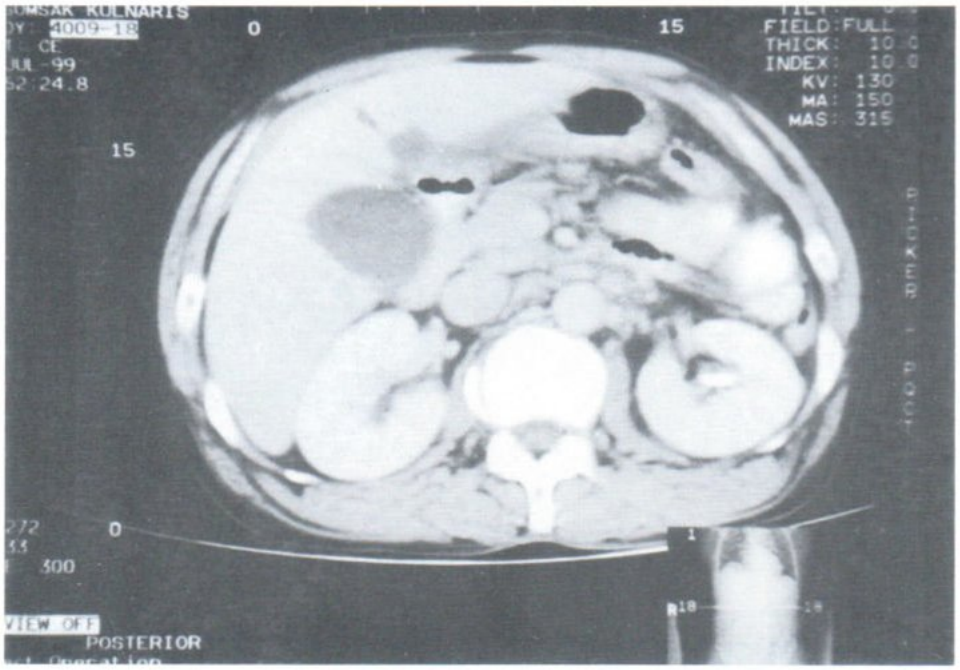

Fig. 4. Inferiorly to slice in fig. 3, also fluid is noted along the porta hepatis and posterior border of left lobe of liver.

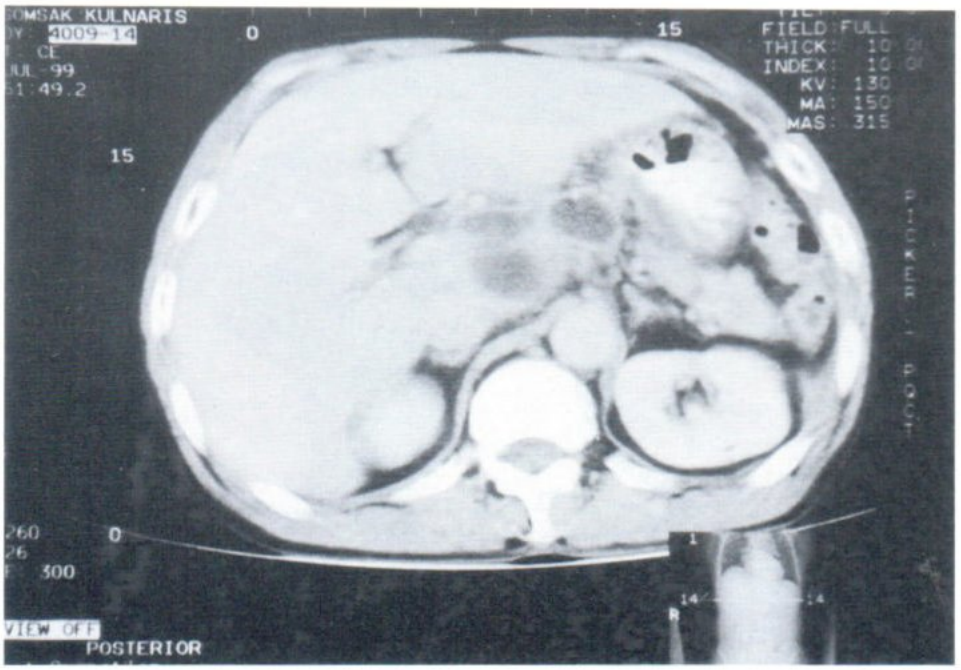

Fig. 5. Small pseudocysts at pancreatic body, the inferior portion of pseudocyst at the superior recess of omental bursa is also seen in this CT slice.

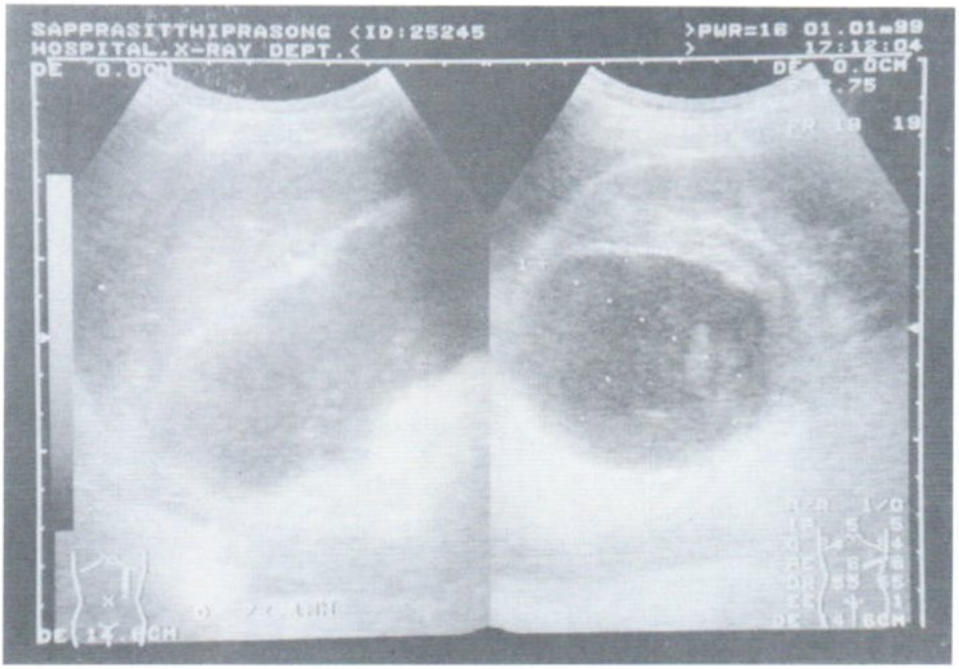

Fig. 6. Encapsulated fluid echoic mass at the upper pole of left kidney.

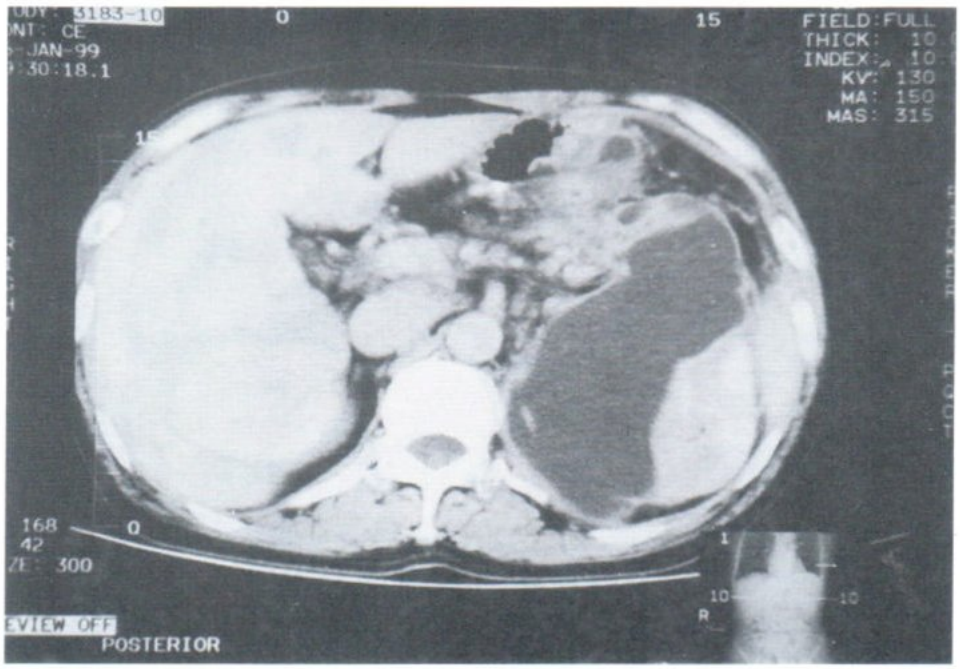

Fig. 7. CT scan showed subcapsular pseudocyst at the upper pole of left kidney.

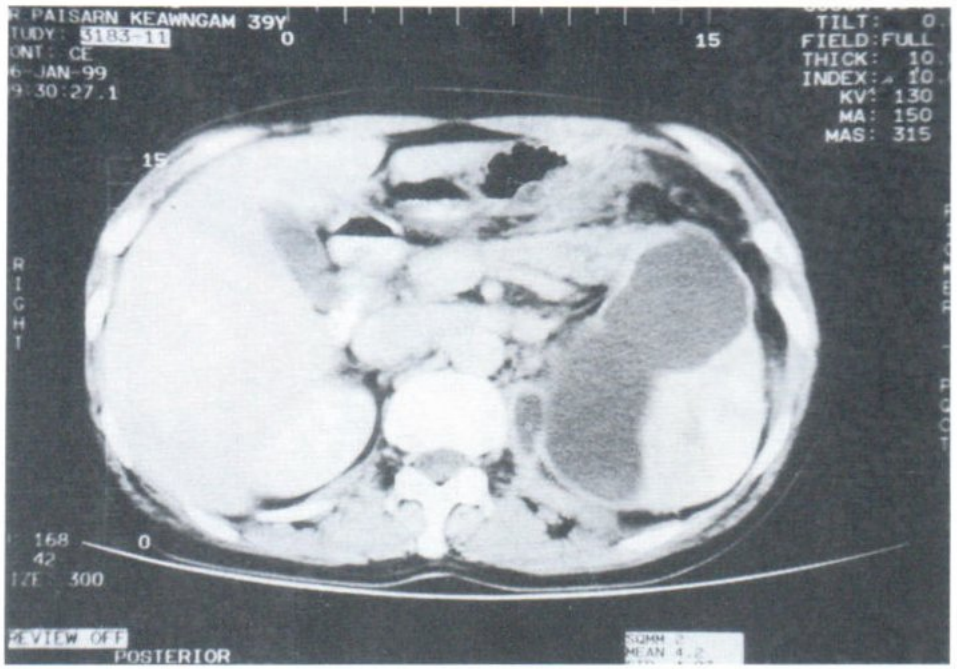

Fig. 8. At pancreatic tail level, there was well demonstrable continuity between subcapsular pseudocyst of left kidney and pancreatic tail.

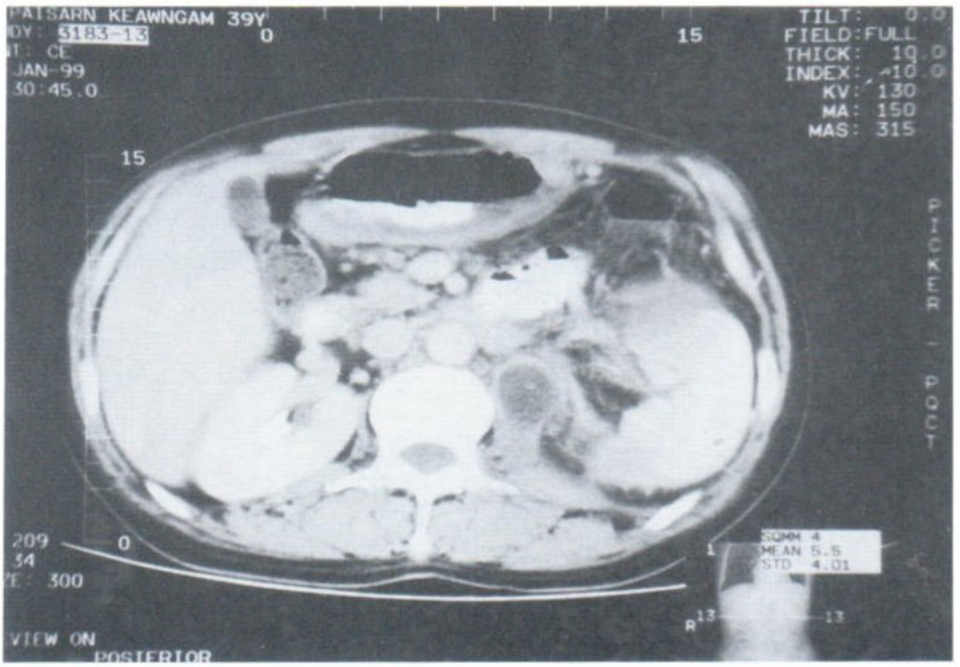

Fig. 9. Small pseudocyst at area of left psoas muscle.

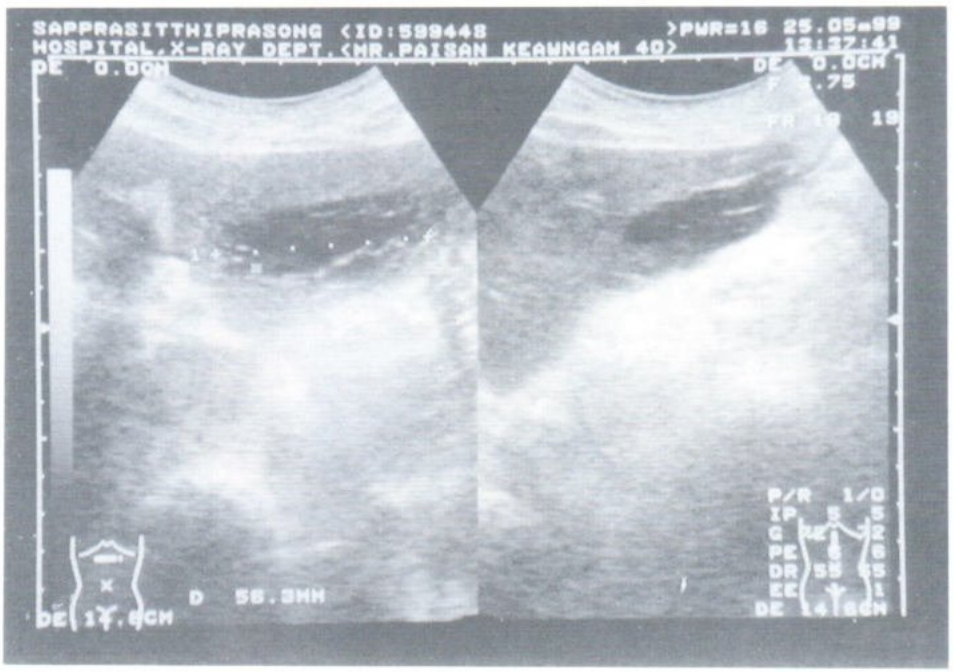

Fig. 10. Extrapancreatic fluid collection at the left lobe of liver.

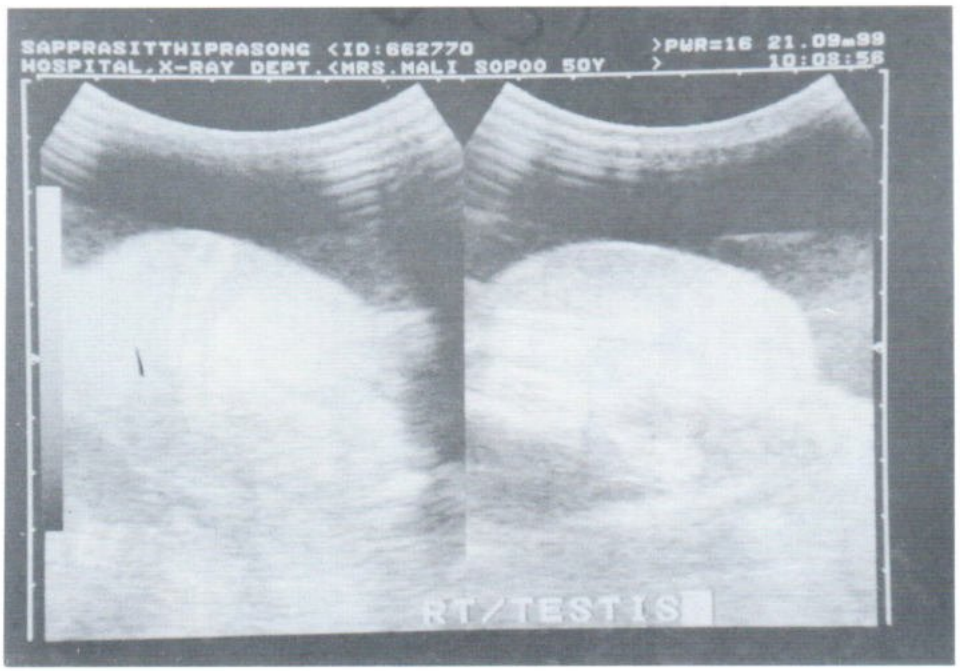

Fig. 11. Hydrocele at right scrotal sac.

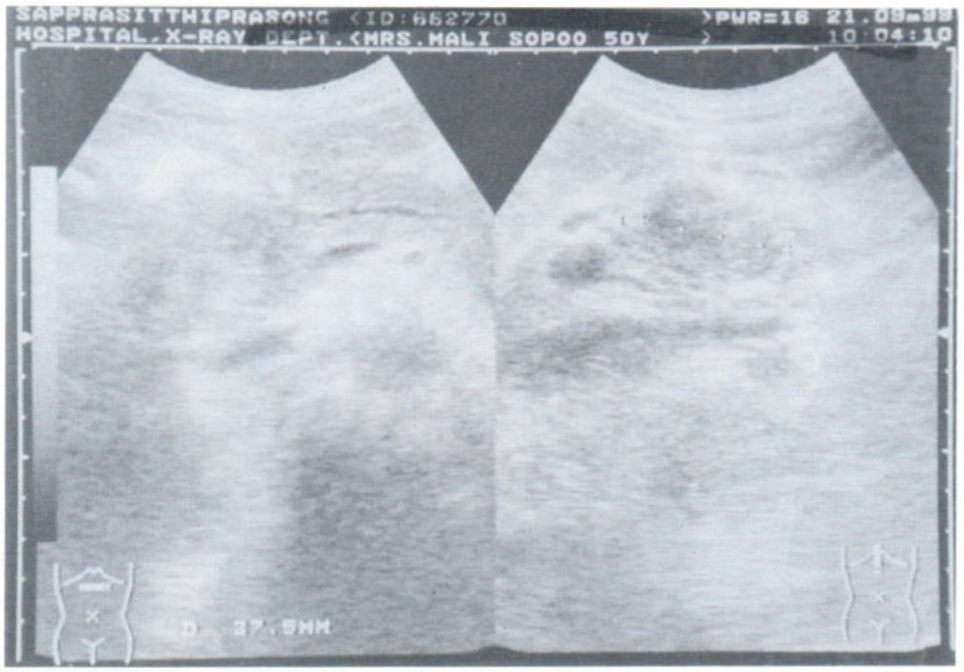

Fig. 12. Low echoic mass at pancreatic head, and mild dilated pancreatic duct.

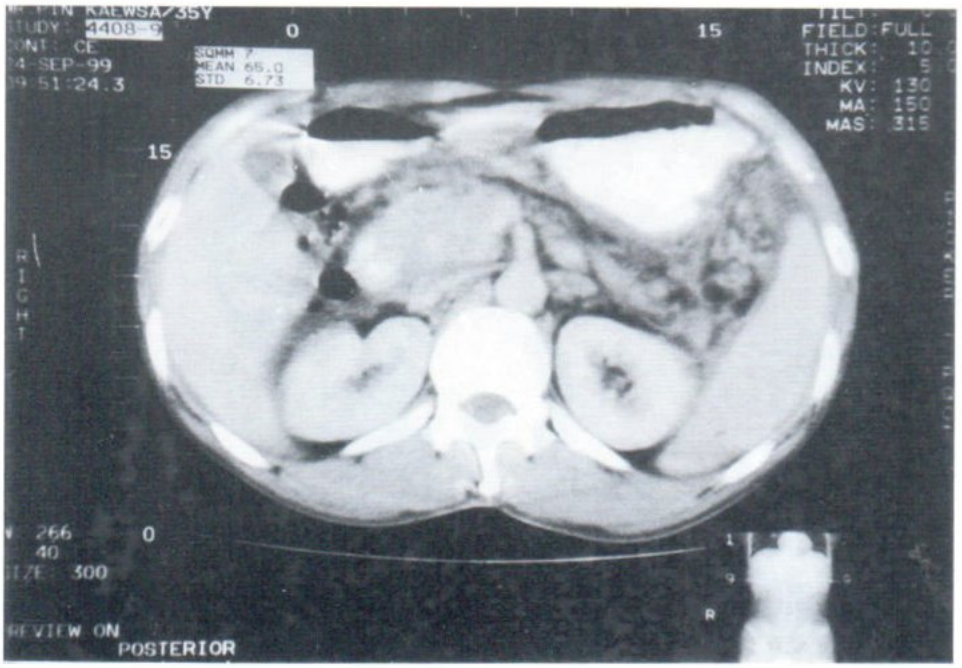

Fig. 13. Focal enlargement of pancreatic head, small pseudocyst at mesentery.

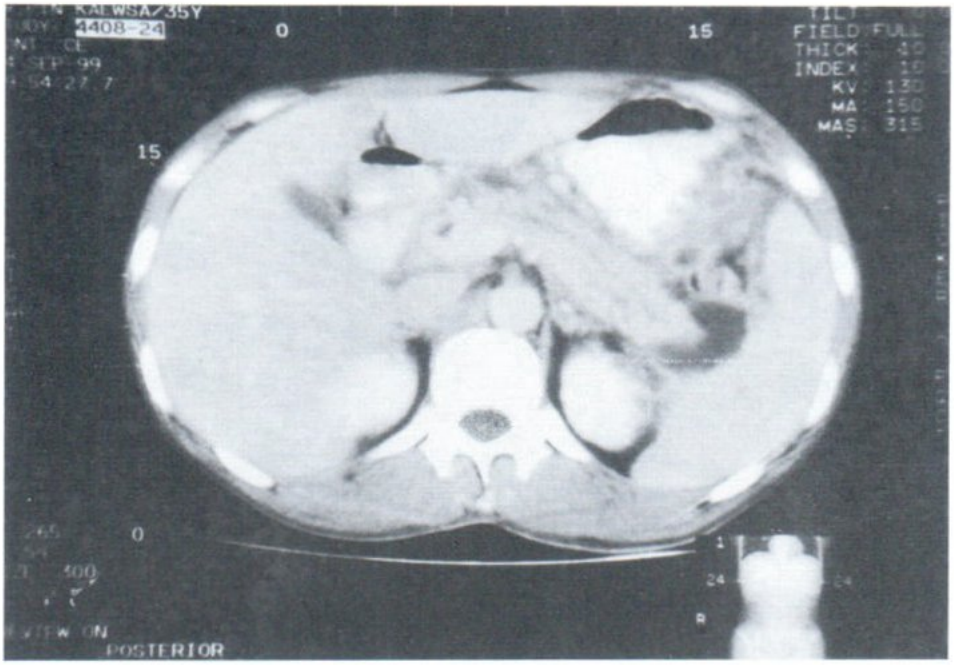

Fig. 14. Fluid collection, or pseudocyst at splenic hilum, mild dilated pancreatic duct.

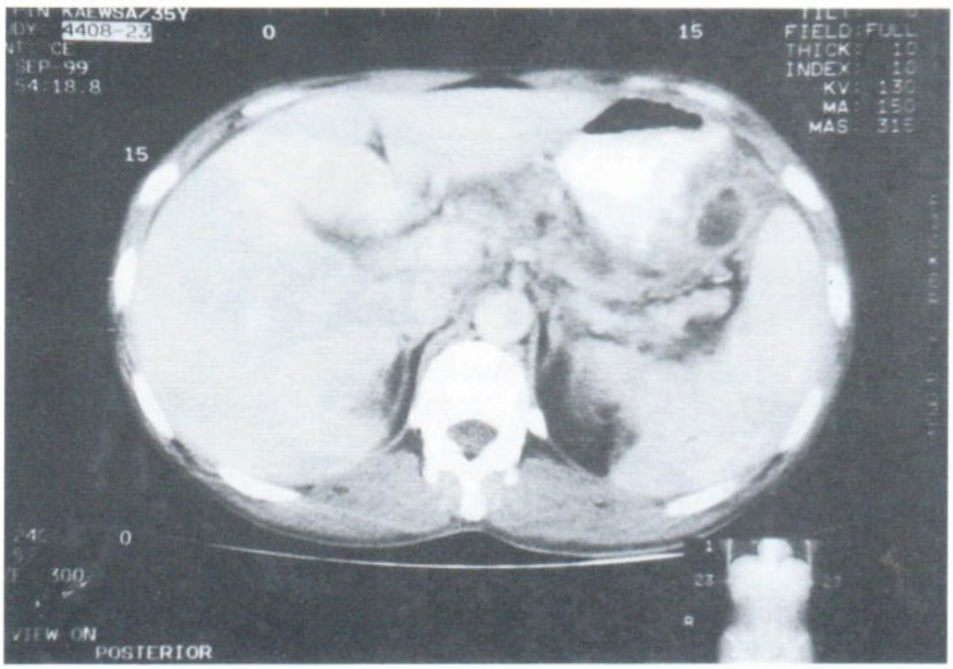

Fig. 15. Small pseudocyst at lesser sac.

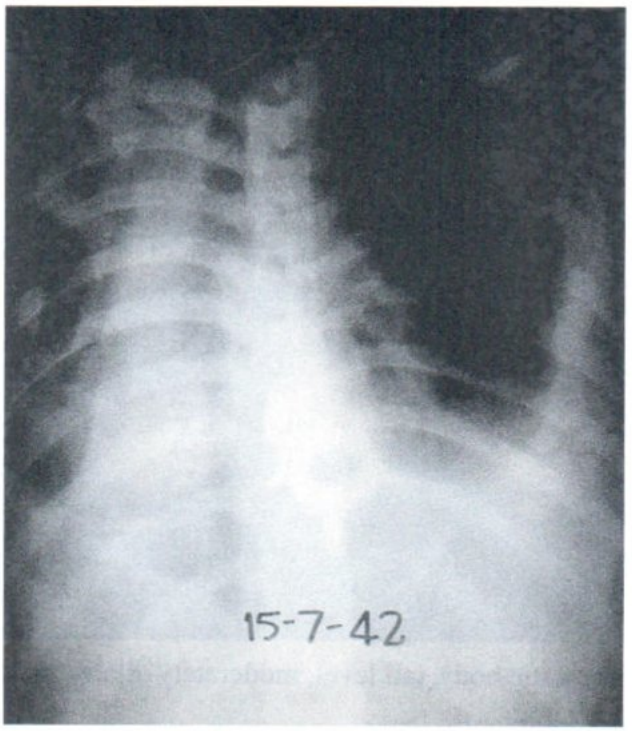

Fig. 16. Bilateral pleural effusion, some loculated component on the right side.

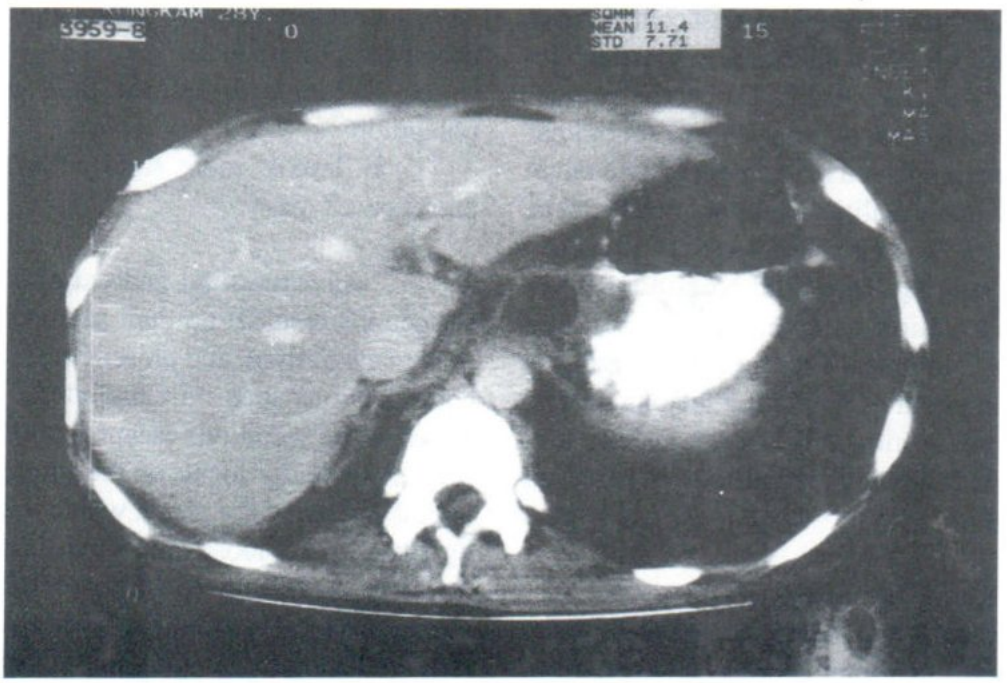

Fig. 17. Well defined pseudocyst, possibly in the hepatogastric ligament.

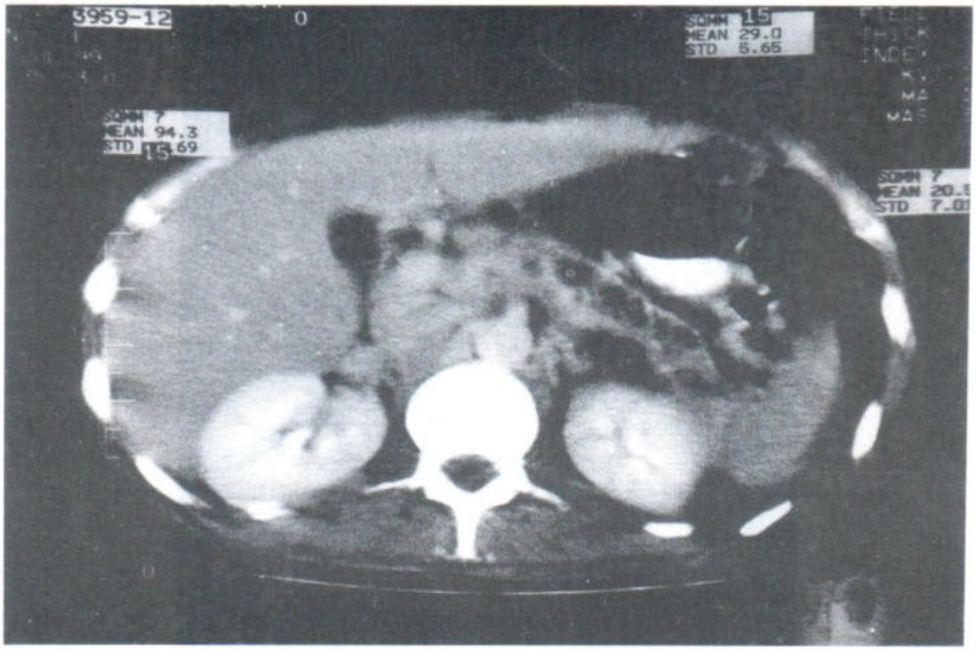

Fig. 18. At pancreatic body, tail level, moderately dilated pancreatic duct is evidence.

DISCUSSION

Pancreatitis is one of the most complex and challenging of all acute abdominal disorders. Its clinical manifestations are as numerous and diverse as its etiologies. No other acute abdominal disorders is accompanied by such a profound range of metabolic abnormalities. A broad spectrum of imflammatory changes may occur in acute pancreatitis ranging from mild edematous interstitial inflammation to fulminant necrotizing pancreatitis. Computed Tomography imaging is helpful as a method of predicting the outcome or severity of pancreatitis at the onset. Balthazar et al. devised the following grading system based on CT finding :

A. Normal pancreas.

B. Focal or diffuse enlargement of the gland (includes nonhomogeneous attenuation of the gland, dilatation of duct, and foci of fluid within the gland, as long as, there is no extrapancreatic edema).

C. Peripancreatic edema and intrinsic abnormalities of grade B.

D. Single ill-defined fluid collection or phlegmon.

E. Two or more fluid collections or the presence of gas.

Pancreatic fluid and extrapancreatic fluid collections result from duct rupture with escape of pancreatic fluid, and present the most common complications seen on Computed Tomography. When these secretions escape from the pancreas, they are typically locate on the anterior or anterolateral surface of the gland, which is covered only by a thin layer of loose connective tissue. The fluid initially leaks into the immediately adjacent spaces: the lesser sac and the left anterior pararenal space. The lesser sac is a potential space anterior to the pancreas and is separated from the pancreas by only a thin layer of connective tissue and the parietal peritoneum, explaining its common

involvement in pancreatitis. Fluid from the lesser sac rarely enters the greater peritoneal cavity due to closure of the foramen of Winslow.

The rare escape of fluid into the greater peritoneal space produces pancreatic ascites. Less frequent involve sites of extrapancreatic inflammatory process include

a) The right anterior pararenal space.

b) The perirenal space after penetrating Gerota's fascia.

c) The posterior pararenal space with spread to the pelvis and upper thigh.

d) The left lobe of liver via the lesser sac and fissure of the ligamentum venosum.

e) The spleen.

f) Between the crura of the diaphragm into the mediastinum.

More frequently in chronic than in acute pancreatitis, this fluid collection tends to be surrounded by a capsule of dense fibrous tissue assuming a cyst-like appearance (Pseudocyst). Most pseudocysts are located in the pancreas and peripancreatic region. The omental bursa is the second most common location of pseudocyst, according to Siegelman et al.[12] The inflammation causes closure of Winslow 's foramen, and the fluid will fill the entire omental bursa or be confined to a local region of the bursa. Uncommonly, the fluid will go to the superior recess of the omental bursa and produce a well defined fluid collection adjacent to the caudate lobe of liver as in the case reported. If a pseudocyst is large enough, it may split the leaves of the omentum producing a mass that project below the surface of the stomach in an anterior position. The inflammatory fluid may also digest through the hepatogastric ligament, splenorenal ligament and even beneath the capsule of solid organ such as liver, spleen,kidney. In case II, the pseudocysts involved the left kidney, left lobe of liver and left psoas muscle. The fluid collection originating from the pancreatic tail focally disrupted gerota's fascia and subsequently the renal capsule to reach the renal parenchyma. Sonography examination of the abdomen was not able to demonstrate the continuity between the pseudocyst and the pancreatic tail, however, primer sonography study was suspected to be pancreatic pseudocyst due to prominent size of pancreatic duct, even though normal size and echo appearance of the pancreas, and the adjacent mesentery appeared to be thickening. Later Computed Tomography study, other signs of pancreatitis was well demonstrable. According to Folco Scappaticci and Stuart K. Markowit,[5] since 1980, 11 cases of intrahepatic pseudocysts arising as complications of pancreatitis have been reported. They also reported one case of intrahepatic pseudocyst complicating pancreatitis in the year 1995. The mechanism have been described for a pseudocyst progression into the hepatic parenchyma. When only the left lobe is involved, it is likely that the fluid initially present in the lesser sac, spreading along the hepatogastric ligament and into the liver. When pancreatitis involves the head of the gland, the potential exists for the spread of disease into the hepatoduodenal ligament. As the common bile duct and the porta vein course within this structure into the porta hepatis, this underlying anatomy provides a mechanism by which intrahepatic pseudocyst formation can develop. In the autopsy case described by Quevedo, Achilles and de Franco,[10] pseudocyts in the liver were found to have developed as pancreatic fluid drained into the liver along the porta vein and it's major branches. In case two, fluid collection along the left lobe liver was seen, and small amount of fluid was seen, crepting along the porta hepatis in case one.

Scrotal swelling may result form a pathologic condition of intrascrotal contents, the scrotal wall or a more generalized disease process. In case three, hydrocele in the scrotum is likely descending from fluid in the abdomen, pancreatic ascites. A sonography of the abdomen

revealed the pancreatic head "mass", mild dilated pancreatic duct, however, sonography failed to demonstrate small pseudocysts 1-2 cm. in the omentum and in the splenic hilum which was well depict on Computed Tomography study. Small pseudocyst at the splenic hilum can be explained that the distal portion of the splenic artery and vein enters the splenic hilum contained within the splenorenal ligament. Because of these anatomic relationships, the spleen and splenic vessels may be involved by pancreatitis. Although rare (frequency 1%-5%), splenic involvement by pancreatitis includes intrasplenic cyst, abscess, hemorrhage, infarction, splenic rupture, and vascular injury. The case of penoscrotal edema due to acute necrotizing pancreatitis have been reported by Kevin K.L Choong.[7] The etiology of his case remains uncertain, but he considered to be an allergic cause.

Chest finding in pancreatitis, includes pleural effusion, usually on the left side with elevated amylase level, empyema, pericardial effusion, mediastinal abscess, pseudocyst, ARDS, and pulmonary edema.

Others complication can occur with pseudocyts, these include a) rupture into the peritoneum cavity, with these rupture the pseudocyst may be colonized and cause an infection b) abscess, fistula c) vascular problems as occlusion, pseudoaneurysm, or spontaneous hemorrhage. Pseudoaneurysm can occur in any vessels in the peripancreatic area, but the most common vessel is the splenic, followed by the gastroduodenal and branches of the pancreaticoduodenal arteries.

The natural history of pancreatic pseudocyts[11] ; 50 % resolve spontaneously and are not clinically significant, 20% are stable and 30% cause complications. In the patients with pseudocysts lasting 7 to 12 weeks, the number of spontaneous resolution decrease. If a pseudocyst is well defined and had a thick fibrous wall, internal drainage is indicated. Internal drainage by cystotomy is the greatest success rate and the lowest complication and recurrence rate. Bradley, Clements, and Gonzalez have recommended that surgical intervention should not be performed in the first 6 weeks because of the high incidence of spontaneous resolution. Between 6 and 13 weeks, the pseudocyst wall becomes well defined , thus internal drainage is possible. Waiting longer than 13 weeks creates a greater risk of spontaneous complications.

In conclusion, despite the rarity of such an entity, clinical, sonography and Computed Tomography of pancreatic pseudocyst, extrapancreatic fluid collection in an uncommon area, should not be mistaken for abscess, mass, or other cause of fluid collections, and correct diagnosis is not difficult by careful imaging. Other signs of pancreatitis, elevated amylase serum level or drainage fluid should arouse suspicious of this condition.

REFERENCES

1. A. Blandino, E. Scribano, G. Aloisi, et al. Subcapsular renal spread of a pancreatic pseudocyst. Abdominal Imaging 1996;21: 73-74.
2. Bradley, EL, Clements, JL, and Gonzalez, AC. The natural history of pancreatic pseudocyst. A unified concept of management. Am J Surg 1979;137:135.
3. Elliot K. Fishman, Philippe Soyer, Donald F. Bliss, et al. Splenic Involvement in Pancreatitis: Spcectrum of CT Findings. AJR 1995;164:631-635.
4. Emil J. Balthazar. CT Diagnosis and Staging of Acute Pancreatitis. Radiol Clin North Am 1989;27:19-37.

5. Folco Scappaticci, and Stuart K. Markowitz. Intrahepatic pseudocyst complicating Acute Pancreatitis: Imaging finding. AJR 1995;873-874.
6. John R. Haaga. The pancreas. In: John R. Haaga, Ralph J. Alfidi, eds. Computed Tomgraphy of the Whole Body Volume 2. St. Louis. Washington, D.C. : The C.V. Mosby Company, 1988:905-945.
7. Kevin K. L. Choong. Acute Penoscrotal Edema Due to Acute Necrotizing Pancreatitis. J Ultrasound Med 1996; 16 : 247-248.
8. Kunio Okuda, Shujiro Sugita, Etsuo Tsukada, et al. Pancreatic pseudocyst in the Left Hepatic Lobe: A Report of Two Cases. Hepatology 1991;13:359-363.
9. Matthew A.Mauro and Robert J. Stanley. In : Joseph K. T. Lee, Stuart S. Sagel, Robert J. Stanley, eds. Computed Body Tomography. New York: Raven press, 1983:213-241.
10. Quevedo FC, Achilles P, de Franco Mf. Propagacao de. Pseudocistos do pancreas pava O figado ebaco : relato de dois casos. Rev Hosp Clin Fac Med S Paulo 1975;30: 371-374.
11. Ralp Weisslerder, Mark J. Rieumont, Jack Wittenberg. Gastrointestinal imaging: Primer of Diagnostic imaging. Mosby : A Times Mirror Company, 1997;223-224.
12. Siegelman, SS, et al. CT of fluid collections associate with pancreatitis. AJR 1980;134:1121.
13. Wolfgang Dahnert. Differential Diagnosis of Hepatic, Billiary, Pancreas, and Spleen disorders: Radiology Review Manual. Williams & Wilkins: Awaverly Company, 1996;487-488.