revealed ALT - 43 IU/L, AST - 45 IU/L, alkaline phosphatase - 493, total bilirubin 6.5 mg/dL, amylase-547 IU/L, lipase-776 IU/L. Complete hemogram revealed hemoglobin 15.7 g/dL and TLC 16,400/cumm.

US showed complex cystic lesions in the right liver lobe with daughter cysts, the mass communicating with the CBD; echogenic contents within suggested hydatid cyst. Other findings were an enlarged pancreas, cholelithiasis, splenomegaly and ascites (**Figure 1**) Plain CT abdomen confirmed the US findings (**Figure 2**). MRCP demonstrated the communication of cyst to biliary tree with dilated CBD till ampulla (**Figure 3**).

ERCP and biliary sphincterotomy was performed and all hydatid membranes, muck and debris removed from the CBD. A biliary stent was deployed. Post-procedure hospital course was uneventful and the patient showed dramatic improvement both in clinical and laboratory parameters. The pancreatitis rapidly resolved. He was discharged on albendazole therapy.

Discussion

Hydatid acute pancreatitis, as a result of hydatid material that enters the bile duct, is a rare complication of hydatid liver disease. Only a limited number of cases have been reported in the literature.[5]

The mechanism of acute pancreatitis caused by hydatid disease of liver open to biliary tree resembles that of gall stone pancreatitis. Hydrostatic pressure inside the hydatid cyst greatly exceeds that in the bile and thus rupture of the cyst into the bile ducts. Overt communication can lead to expulsion of hydatid cyst material (membranes, scolices, daughter cyst) into the biliary tree causing cholestatic jaundice and recurrent cholangitis. The passage of this material through the papilla of Vater can cause occlusion of the pancreatic duct and / or bile reflux into pancreatic duct leading to acute pancreatitis. A local allergic reaction to ecchinococcal antigen inside the ampulla may also play a part in the initiation of pancreatitis.[6,7]

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Development of huge pancreatic pseudocyst following organophosphorus poisoning – a case report and review of Literature

Introduction

There are a number of case reports of acute pancreatitis associated with organophosphorus poisoning (1-5). A cause and effect relationship has been demonstrated in animal studies (6). However this association is perhaps still not widely recognized. Neither do commonly used surgical and medical textbooks describe acute pancreatitis as a presenting feature of organophosphorus poisoning nor is organophosphorus poisoning listed as one of the aetiological factors of acute pancreatitis and its complications. In adults the frequency of acute pancreatitis related to organophosphorus poisoning is 12.7% (7). Organophosphorus poisoning is not uncommon in our society; given the widespread availability and use of organophosphorus insecticides. Poisoning may be ccupational, accidental or suicidal.

Case Report

A male patient aged about 18 years was admitted in the surgical emergency of Nehru Hospital, B.R.D. Medical College, Gorakhpur with a two-day history of severe abdominal pain, abdominal distension, vomiting, palpitation and fever. Ten days prior to this admission, he was admitted in the medical emergency of the same hospital following ingestion of two teaspoons of organophosphate insecticide (Feridon) in a suicide attempt. He was managed conservatively and was discharged after improvement. There was no past history of trauma, alcoholism or pancreaticobiliary disease, nor significant medication history.

On examination, he was dehydrated, anemic, febrile, tachycardic, tachypneic, hypotensive, and had upper abdominal distension and rigidity all over the abdomen. Intravenous medications were started and investigations revealed hemoglobin 9 g/dL, total leucocytes count 16000/cumm, differential count- polymorphs 80% lymphocytes 10% eosinophils 6% and monocytes 4%, serum alkaline phosphatase 412 IU/L, serum amylase 800 IU/L, lactic dehydrogenase 1200 IU/L, serum lipase 560 IU/L, serum calcium 7 mg/dL, blood urea 54 mg/dL and serum creatinine 1.6 mg/dL. Other laboratory findings were near normal range. Electrocardiogram was normal excepting the tachycardia. Radiological investigations revealed obliterated left costophrenic angle in X-ray chest, features of peritonitis (ground-glass appearance) in X-ray erect abdomen and bulky pancreas with peripancreatic cyst of size 12 cm x 8 cm x12 cm with mild ascites on ultrasonography (USG).

Patient was managed conservatively and became asymptomatic except upper abdominal distension but oral feeding was well tolerated. He was discharged from the hospital

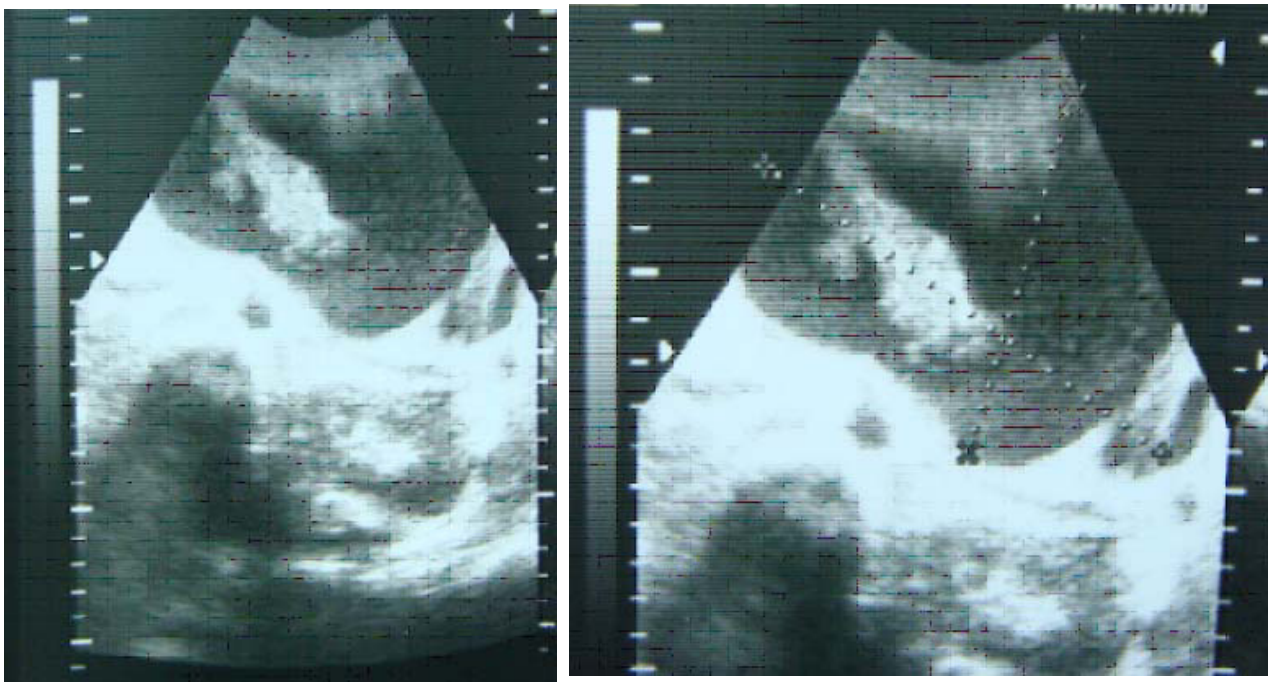

Fig.ure 1: USG of the abdomen showing pancreatic pseudocyst

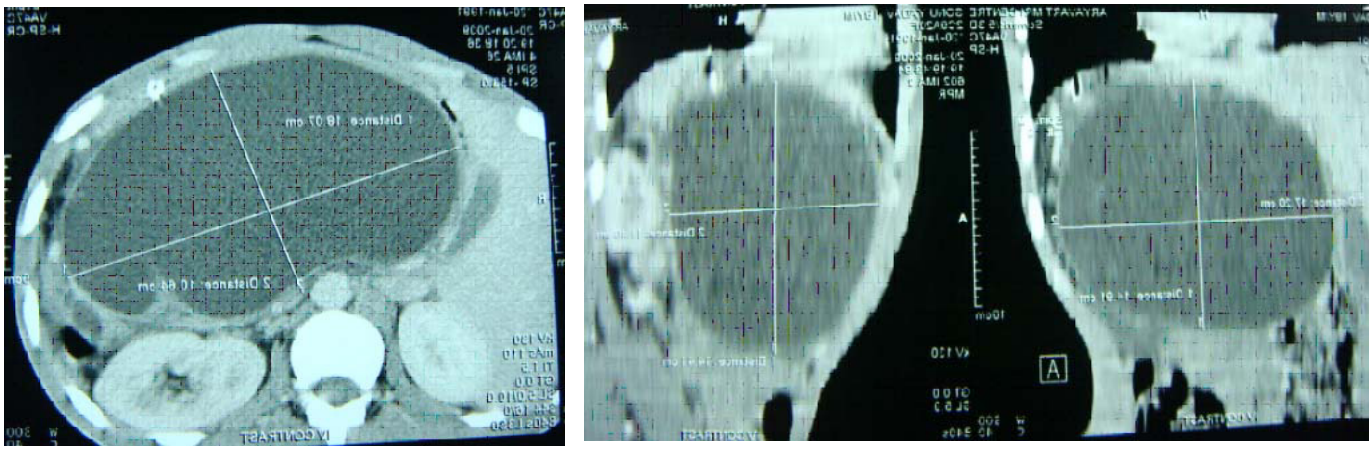

Fig.ure 2: CT scan of the abdomen showing huge pancreatic pseudocyst

with advice to follow up in the OPD with serial hemoglobin and ultrasonography (**Figure 1**). At six-week follow up his other radiological findings became normal but the size of the cyst gradually progressed. He was readmitted and a dynamic computerized tomography (CT) scan was performed which showed diffusely bulky pancreas measuring 2.1 cm, 3.4 cm, and 2 cm in head, body and tail regions, respectively with huge pancreatic pseudocyst of size 18 cm x 10 cm x 15 cm in transverse x AP x cranio-caudal diameter with minimal ascites (**Figure 2**). After correcting anemia by blood transfusion and further preoperative assessment and treatment, he underwent exploratory laparotomy and internal drainage of the cyst in the form of cystogastrostomy. A postoperative ultrasonography was performed to ensure complete drainage of the cyst. His postoperative recovery was uneventful. The patient is under regular follow up for the last nine months and has no complaints till date.

Discussion

Organophosphorus compounds are commonly used worldwide as agricultural insecticides and in this region to eliminate household insects (mosquitos and cockroaches). Acute pancreatitis following organophosphorus poisoning generally shows a subclinical course (8). Organophosphate insecticides are potent inhibitors of enzyme acetylcholinesterase which leads to increased acetylcholine activity responsible for systemic symptomatology seen in organophosphorus poisoning such as hypersialorrhoea, pinpoint pupils, cramping abdominal pain and vomiting. They directly stimulate secretion of pancreatic juice and trypsin. The pancreatic duct and bile duct pressure increases because of over secretion of pancreatic juice and ampullary spasm, pancreatic duct spasm and edema. This hyper-stimulation has been hypothesized as the mechanism of acute pancreatitis following organophosphorus poisoning (9-11). In 1979, Dressel (12) described the first case report of pancreatitis following organophosphorus poisoning.

A pancreatic pseudocyst develops within a period of 1-4 weeks following the onset of acute pancreatitis as a result of pancreatic enzymes, debris, fluid, tissues and blood collection. The incidence of pancreatic pseudocyst development in acute pancreatitis is 15% (13).

Abdominal pain that may or may not radiate to the back is a common symptom in patients. Serum pancreatic enzymes and suitable imaging techniques are used more often in determining the patient's clinical condition. The vast majority of pancreatic pseudocysts resolve spontaneously, however a cyst that does not resolve spontaneously (10% of cases) may lead to serious complications such as pain, pseudocyst rupture and abscess formation due to the expanding size of the lesion and compression over viscera (14). In our patient acute pancreatitis was suspected due to history of admission a week previous, in the medical emergency with abnormal pancreatic enzymes following organophosphorus poisoning. Pancreatic pseudocyst was diagnosed by ultrasonography. Possible etiological factors for acute pancreatitis (alcoholism, biliary diseases, medication and others) were excluded. Due to the history of ingestion of organophosphate insecticide, we diagnosed pancreatic pseudocyst as a complication of organophosphate induced pancreatitis. We initially managed the case conservatively with the help of serial ultrasonography. However, because of the progressive abdominal distension we did dynamic CT scan of the abdomen. Subsequently the patient underwent exploratory laparotomy and internal drainage (cystogastrostomy), which was followed by complete recovery.

In conclusion one should keep in mind that this complication can arise during the course of organophosphorus poisoning management and appropriate surgical treatment is necessary to reduce the morbidity and mortality.

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PET positive lesions with increased SUV in post-surgical patients: where do we stand?

Introduction

PET (Positron emission tomography) Scan using 18F – fluorodeoxyglucose (FDG), a non invasive imaging technique, has been shown to be the most accurate investigation for the detection of local recurrences and distant metastases in patients with gastrointestinal malignancies. FDG is transported in to cells by glucose transporters (primarily GLUT 1) and phosphorylated and metabolically trapped by hexokinase. Cancer cells have been shown to have increased uptake of glucose and glycolytic enzymes and therefore preferentially uptake FDG, which then decays by position emission (1).This article is designed to report on the false – positivity of FDG-PET scan in evaluation of recurrent or metastatic GI malignancies and SUVs (standardized uptake values) are examined to determine their significance in determining PET accuracy.

Case Reports

From June 2008 to August 2009 five cases of false positive PET lesions were identified in post operative patients who underwent PET- CT as part of follow-up. The details of five patients are:

Case 1

19 years old female diagnosed to have hepatoma right lobe of liver underwent right hepatectomy. After 15 months of follow-up, PET scan revealed PET positive lesion (SUV-5.2) in remnant liver along resected margin. Patient underwent non anatomical liver resection on January 2009. Histopathological examination revealed suture granuloma associated with foreign body giant cell reaction.

Case 2

52 years old female diagnosed to have carcinoma gall bladder underwent radical cholecystectomy on July 2008. After 11 months of follow up PET scan revealed soft tissue thickening in gall bladder fossa and infiltrating liver parenchyma with SUV of 4.9. Patient underwent non anatomical liver resection on June 2009. Biopsy revealed chronic inflammation with suture granuloma and fibrosis.

Case 3

64 years old female diagnosed to have carcinoma recto sigmoid underwent anterior resection. After 23 months of follow-up PET scan revealed a positive lesion in the tail of the pancreas (**Figure 1 a,b**). Patient underwent distal pancreatectomy with

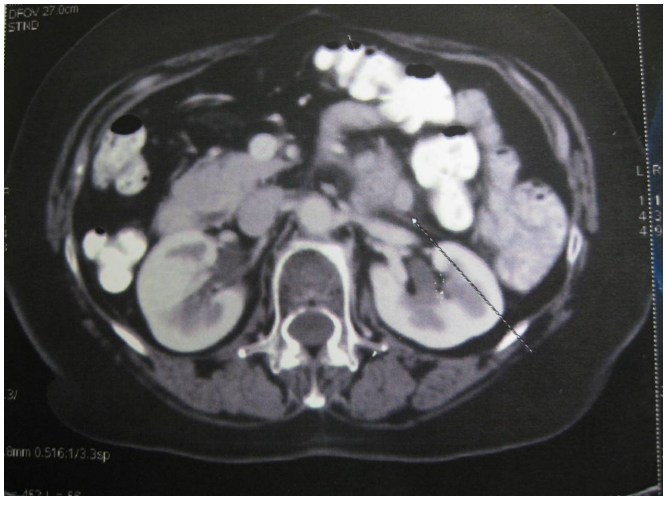

Figure 1a: In case 3, CECT abdomen Showing SOL in tail of pancreas