Case Report

Pancreatic ascites with 'inflammatory transudate'

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Abstract

Pancreatic ascites is not an uncommon condition but requires a high degree of suspicion for prompt diagnosis. Since both hepatic cirrhosis and pancreatitis are related to ethanol abuse, routine investigation of patients with cirrhotic ascites for the rise of ascitic fluid amylase levels will facilitate early diagnosis. The combination of inflammation of pancreas and portal hypertension due to hepatic cirrhosis could give a picture of 'inflammatory transudate'.

Keywords: Ascites, inflammatory transudate, pancreatic ascites

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INTRODUCTION

Ascites is excessive and abnormal accumulation of fluid in peritoneal cavity. When this occurs during the course of acute or chronic pancreatitis, it is called 'pancreatic ascites'.[1] It could usually be due to alcohol abuse. Awareness of pancreatic ascites has increased since the early 1950s when it was said to be first reported.[2]

CASE REPORT

A 33-year-old man presented to medicine outpatient service in a teaching hospital with complaints of painful abdominal distension, and breathlessness and left-sided chest pain of 1-month duration. At the age of 8 years, he was treated for pulmonary tuberculosis. There is a history of ethanol and nicotine abuse. Physical examination details are as follows: pulse 82/min, blood pressure 120/70 mmHg in the right arm in the supine position, respirations 28/min and temperature 98.5°F.

There was mild distension of abdomen with tenderness over all the regions; shifting dullness was present. Examination of respiratory system revealed flat note over the left mammary, left infra-axillary and infrascapular regions and reduced to absent breath sounds in the same regions suggesting pleural effusion.

In view of the chronic ethanol abuse, pain abdomen and unilateral shifting dullness, the possibility of chronic liver disease complicated by tuberculosis peritonitis was considered. Ultrasonography of abdomen showed features of cirrhosis of liver, gross ascites, left-sided pleural effusion and mild splenomegaly with splenic collaterals. Contrast-enhanced computed tomography (CECT) of the abdomen showed features of acute pancreatitis with multiple pseudocysts. Upper gastrointestinal endoscopy showed fundic varices with duodenitis. Based on CECT abdomen and other imaging findings, elevated levels of ascitic fluid amylase and the presence of exudative

Access this article online	
Quick Response Code:	Website: www.jcsr.co.in
	DOI: 10.4103/JCSR.JCSR_26_19

How to cite this article: Prasad MR, Gopalakrishna P, Madhuri MS, Sridhar MS. Pancreatic ascites with 'inflammatory transudate'. J Clin Sci Res 2019;8:29-31.

left-sided pleural effusion [Tables 1 and 2], the diagnosis of pancreatic ascites was established. The patient was managed conservatively by medical therapy which is the initial treatment recommended; he was discharged at request for treatment at another centre of his choice.

Table 1: Laboratory investigations

Blood/serum	Liver function tests
Haemoglobin 13 g/dL	Total bilirubin 0.9 mg/dL
PCV 39%	
RBC 4.44 million/cu mm	SGOT 24 IU/L
MCV 88 fL	SGPT 17 IU/L
MCH 30 pg	
MCHC 34%	
Platelets 178,000/μL	ALP 103 IU/L
ESR 15 mm after 1st hour	
Blood urea 16 mg/dL	Hepatitis viral serology - negative
Serum creatinine 0.7 mg/dL	

MCH=Mean corpuscular hemoglobin; MCHC=Mean corpuscular haemoglobin concentration; MCV=Mean corpuscular volume; PCV=Packed cell volume; RBC=Red blood cell; ALP=Alkaline phosphatase; ESR=Erythrocyte sedimentation rate; SGOT=Serum glutamate oxaloacetate transaminase; SGPT=Serum glutamate pyruvate transaminase

Table 2: Blood/serum, ascitic fluid, pleural fluid analysis

Component	Blood/serum	Ascitic fluid	Pleural fluid
Total leucocyte count - cells/mm^3	1200	500	1356
Differential count (%)	PMN=78 Lymphocytes=16 Eosinophils=3 Monocytes=3	Neutrophil=10 Lymphocyte=90	Neutrophil=7 Lymphocyte=93
Glucose (mg/dL)	104	109	128
Protein (g/dL)	6.3	2.9	3.9
Alb (g/dL)	3.1	1.8	2.8
Amylase (IU/L)	308	984	486
LDH IU	158	-	133
Pl fl LDH/S LDH	NA	NA	0.84
(S-Fl) TP	NA	3.4	2.4
Fl TP/STP	NA	0.46	0.619
(S-Fl) Alb gradient	NA	1.3	0.3
Fl Alb/S Alb	NA	0.58	0.9
ADA (U/L)	-	10.1	7.3

NA=Not available; LDH=Lactate dehydrogenase; PMN=Polymorphonuclear leukocytes; Alb=Albumin; ADA=Adenosine deaminase; FL: Fuid; STP=Serum total protein; TP=Total pro fein

DISCUSSION

Pancreatic ascites is said to more common among males between ages of 20 and 50. While the abdominal pain is described as 'variable' and abdominal tenderness is notably lacking,[1] both were present in the case of the patient reported. Pancreatic ascites occurs generally against the background of heavy alcohol consumption. Several possible mechanisms of pathogenesis of pancreatic ascites were described. Transdiaphragmatic lymphatic transfer of pancreatic enzymes, intrapleural rupture of mediastinal extensions of pseudocysts and diaphragmatic perforation are some of them. The diagnostic value of pleural fluid amylase levels was recognized. The predilection for left-sided pleural effusion is explained on the anatomical basis of intimate relation of pancreas to the left hemidiaphragm.[3]

The mechanism of ascites and pleural effusion is succinctly described by Cameron[2] as due to disruption of the main pancreatic duct, resulting in an internal fistula between the duct and peritoneal cavity, producing massive ascites and if the duct disruption is posterior, pancreatic secretions can track up into the mediastinum and then escape into one or both pleural spaces, resulting in an internal fistula between the pancreatic duct and pleural space, producing a massive effusion.

The amylase in the ascitic fluid of a patient with pancreatic ascites is always elevated even if the serum levels are normal or slightly elevated as clearance from serum is faster in comparison to that of ascites. In addition, the ascitic fluid protein is usually elevated to the range of 3 g/dL or above. The combination of ascitic fluid protein level in 'exudative' range and serum albumin ascitic fluid albumin gradient (SAAG) in the portal hypertensive range or 'transudative' range gives a picture of 'inflammatory transudate' similar to the observation by Witte *et al*[4] that peritoneal transudate could be a diagnostic clue to portal system obstruction in patient with intra-abdominal neoplasms or peritonitis. In the present case, though exudative peritoneal fluid is expected, the level of SAAG of 1.3 g/dL suggests the existence of portal hypertension for which imaging and endoscopic findings lend credence. Hence, apart from the traditional concept of transudate and exudates, an additional term inflammatory transudate is also useful so that a wider range of conditions can be considered in the differential diagnosis. Thus, in the evaluation of ascites, the terms, transudate, exudates, serum-to-ascites total protein gradient[5] and SAAG[6] which gives clue whether portal hypertension is present or not and 'inflammatory transudate' form a battery of investigations in the diagnosis of ascites.

In patients with chronic alcoholic liver disease and pancreatitis, the ascitic fluid picture could be misleading. A 'gestalt' of ascitic fluid easily settles the issue. Since the ascitic fluid protein parameters had mixed features, ascitic fluid of the patient reported can be termed as 'inflammatory transudate'.

All patients with ethanol abuse and cirrhotic ascites should be routinely investigated for possible pancreatic ascites. A high degree of suspicion of pancreatic ascites in all patients with alcoholic hepatic cirrhosis without reference

to pain abdomen would allow recognition of more number of patients with pancreatic ascites, and proper medical and surgical management can be planned.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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