e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2023; 15(12); 475-478

Case Report

A Patient with Tuberculous Pleural Effusion Later Diagnosed with Massive Pancreatic Pleural Effusion Due to Pancreaticopleural Fistula - A Case Report

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Received: 10-09-2023 Revised: 23-10-2023 / Accepted: 26-11-2023
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Conflict of interest: Nil

Abstract

Background

Massive pleural effusion due to Pancreatitis can be the missed cause when hemorrhagic effusion is being explored for just tuberculosis or Malignancy, specially when effusion is on right side.

Though rare, hemorrhagic pleural effusion can be the sole manifestation of Pancreatitis, especially on the right side of thorax.[1-6]. Here a case is presented where a middle age male patient came primarily with the complain of dyspnea which was gradual in onset. He was cachexic and had h/o pain abdomen 10 days back which was uneventful. But later he developed massive pleural effusion . The investigations further revealed the pleural effusion was due to the complication of chronic pancreatitis.

Keywords: pancreatitis, hemorrhagic pleural effusion.

Introduction

There are several admission in the hospital due to complications of Pancreatitis complications in which pleuropulmonary cause is upto 94% [7]. Pulmonary complications includes arterial hypoxia, atelectasis, pneumonia and acute respiratory distress syndrome (ARDS).

Other intrathoracic complications includes pleural effusion, empyema ,pericardial effusion, cardiac arrhythmias, mediastinal collections or pseudocyst [6,8].

Pulmonary complications includes

1. ALI/ARDS: diffuse bilateral coalescent opacities on diuretics with increased density posteriorly.
2. Pulmonary edema: Diffuse bilateral alveolar opacities with peripheral sparing that resolves on administration of diuretics.
3. Infections: Lobar or broncho lobar consolidations with air bronchograms, GGOs.

Pleural complications includes

1. Empyema
2. Pancreatic pleural fistula

Mediastinal complications includes

1. Pseudocyst

Cardiac complications includes Pericardial effusion, CCF. Vascular complications includes Pulmonary thromboembolism, thoracic aortic aneurysm.

The incidence of pleural effusion in acute pancreatitis is around 50% based on the detected by CT [9]. Pleural effusions are usually left sided which are chemically induced or sympathetic in nature, with normal fluid amylase levels.

In chronic Pancreatitis , pleural effusion is rare and is usually due to the formation of pancreatic pleural fistulas [10,11].

In acute pancreatitis, the pleural effusion usually resolves as the inflammation subsides. If it persists for longer than 2 weeks or if there is right sided massive pleural effusion, the possibility of pancreatic pseudocyst or pancreatic pleural fistula should be considered [11,12]. Pancreticopleural fistula are abnormal communications between the pancreatic duct and viscera or third space cavities of the thorax or the abdomen. They can be due to direct or indirect through the pancreatic fluid collections, seen in 0.4% cases and commonly in alcoholics [13]. Recurrent effusions and rapidly accumulating effusions raises suspicions of pancreaticopleuralfistula[13].

Case Report

A patient 39 year old, male came to the emergency for the c/o of pain in epigastric area with radiation to back since 20 days which relieves slightly in sitting position. Pain increases in food intake. There was fever, high grade since 20 days. There was no H/O loose motions or vomiting. He had also c/o shortness of breath, mMRC grading III since the same duration which was more of discomfort. There was right sided chest pain which was of pricking type and increased on deep breathing, syncope or generalised anasarca or pedal edema.

He was started outside Antituberculous treatment.

Patient was chronic alcoholic with daily intake of more than 250 ml of binged alcohol since more than 20 years. No h/o diabetes, Hypertension, Coronary heart disease.

On examination, he was conscious, oriented and responding to commands. On per abdomen examination, tenderness was present on epigastrium. On respiratory examinations, respiratory movements were reduced on the right side and trachea was shifted towards left side. On percussion there was presence of stony dull note over right hemithorax. On auscultation, respiratory sound was absent and vocal resonance were decreased over the right hemithorax. Cardiac examination was normal.

BP was 114/82 mm Hg, PR 112/min, spo2 98%, RBS 99 mg%. On general examination, he was severely cachexic, thin built, with temporal hollowness, mild pallor, no icterus, no lymphadenopathy, Respiratory rate 20/minute, no intercostal recession.

ICD inserted on right side. Drainage was >300 ml/ day, slowly decreasing to >100 ml /day in 1 week. Colour was dark brown, turbid and thick.

Chest X-ray showed right sided massive pleural effusion with mediastinal shift to opposite side.

Following investigations done:

Hb- 7.2 gm/dl, TLC 13,400 /cmm, ⟹20,000 ⟹ 22,000

platelet count 6.85 $\times 10^3$ µl, Serum protein 5.38gm%, corrected reticulocyte count 1.2 %

GBP- Reduced red cell density, mild anisocytosis, predominantly normocytic normochromic with microcytic hypochromiccells, neutroplhilic leucocytosis, thrombocytosis.

Serum uric acid 2.59mg/dl

Serum Albumin: 2.34 g/dl, S Amylase 275 U/L, S. Lipase 160 U/L, S. potassium 3.7 mmol/l, S. sodium 132 mmol/l

PT/INR 18/1.42 sec
KFT normal
LFT normal
Troponin I 7.0 ng/L
HIV -ve, HBsAg -ve, HCV -ve
HsCRP 112.42 mg/l
Serum LDH 259.50 U/L
Iron profile- serum Iron 13.87 µg/dl
Serum UIBC 109 µg/dl
Serum TIBC 123.60
Serum Ferritin 1337 ng/ml
Serum folic acid 15.69 ng/ml
Vitamin B12 1655 pg/ml
Free T3 2.32 Pg/ml, T4 1.00 ng/dl, TSH 1.45 uIU/ml

Pleural fluid examination

Colour: pus like turbid pleural fluid culture -ve, ZN stain for AFB -ve, genexpert for MTB -ve, glucose 59.70 , protein 2.45 gm%, Albumin- 1.12 gm%, TLC > 10,000 cells/cmm DLC Lymphocytes 40%, Neutrophils 60%.

Pleural fluid Amylase

27786 U/L, Lipase – 16167 U/L

USG Abdomen showed Liver normal size (142 mm), normal echotexture, no evidence of any lesions. Gall bladder , CBD, Portal vein normal.

Pancreas mildly heterogenous in echotexture.

Spleen enlarged (133 mm).

Mild ascites present, Massive echogenic pleural effusion on right side.

Mild left sided pleural effusion.

CECT Abdomen: Liver enlarged in size (150mm), Gall bladder partially distended, CBD normal, Pancreas appears bulky and heterogenous with few non-enhancing hypodense areas noted predominantly in pancreatic body and tall region (involving >30% of pancreatic parenchyma) with pancreatic and peripancreatic free fluid.

Spleen is enlarged in size (133 mm). No focal lesions . no collaterals at splenic hila. Splenic vein is not well visualised and is replaced by multiple dilated tortous collaterals channels at splenic hilum, peripancreatic and perigastric region.

Kidney, urinary bladder and prostate normal Mild ascites present, few subcentimetric, mesenteric and retroperitoneal lymph nodes seen. Bilateral pleural effusion with segmental collapse with ICD catheter in situ in right side. Minimal effusion was seen.

Impression: Features are suggestive of Acute Necrotizing Pancreatitis with Acute necrotic collection (Modified CT Severity index 10/10).

CECT Thorax: Right sided multiple loculated effusion (Largest AP DEPTH=62mm) noted in right pleural cavity with enhancing passive collapse of right middle and lower lobe.

Left sided pleural effusion (AP DEPTH 22mm) with fissural extension, Ground glass opacities with fibrotic bands noted in right upper lobe. Few enlarged mediastinal lymph nodes are noted.

MRCP with MRI abdomen was advised as CECT abdomen didn't revealed any pancreatic pleural fistula. The report showed a fistula tract between the left pleural cavity and pancreas.

Treatment of PPF includes conservative management with stent placement and/or octreotide or surgically closing the fistula. ERCP is needed for diagnosis and management conservatively. As patient denied invasive procedure and also patient's drain was removed after 1 week of conservative treatment.

The patient was discharged but readmitted for the complaint of repeat right sided pleural effusion. He was managed conservatively and referred to the gastrosurgery department for surgical closure of Pancreatic pleural fistula.

Discussion

The Atlanta Symposium 1992 , has divided acute pancreatitis into mild and severe types, on the basis of clinical and biochemical findings [14]. Later in 2008, classification was revised as "Acute Pancreatitis Classification Working Group" and a new morphological classification was defined on the basis of imaging findings as "interstitial edematous pancreatitis" and necrotizing pancreatitis". This revised classification identifies two phases of the disease- early and late- and severity of the disease as mild, moderate or severe depending on the presence or absence of organ failure, fluid collections and comorbid conditions. [15,16,17].

The complications of the acute pancreatitis may be local or distant and immediate or delayed. Several factors increases these complications like age, gall stones, the presence of organ failure and pancreatic parenchymal necrosis.[18].

The modified CT severity index describes the severity of pancreatic inflammation. This score is based on the percentage of necrosis, the extent of fluid collections and extra pancreatic involvement. It has been shown to have prognostic accuracy for the development of complications. [19,20,21].

Intrathoracic complications can involve the pleura, lung, heart, pericardium or the mediastinum including the mediastinal vasculature[22,23,24]

This patients developed Pleural effusion. One of the mechanisms is the transdiaphragmatic lymphatic blockage. There may be disruption of pancreatic duct, leading to the leakage of pancreatic enzymes and the formation of pancreaticopleuralfistula. Exudation of fluid into the pleural cavity from the subpleural diaphragmatic vessels may also cause pleural effusion [24].

The confirmation of the fistula is by- high pleural fluid amylase levels though other causes must be excluded like parapneumonic effusions: pulmonary tuberculosis; esophageal perforation; liver cirrhosis; hydronephrosis; leukrmia/lymphoma and malignancy of the lung, pancreas, rectum, and the gynecological system.

Levels above 50,000 u/L favors fistula. [25]. MRI with MRCP is the modality of choice to visualise the fistula as it is superior to both CT and ERCP.

Therefore History and physical examination are non-specific for PPF and further evaluation with imaging and laboratory work up is required. Sensitivity for PPF is USG<CT<MRCP/MRI.

Though ERCP is invasive but it is the treatment of choice. CT and MRCP are useful in evaluating additional pancreatic parenchyma and ductal structural abnormalities such as pancreatic mass, pseudocysts or collections.[26,27]. Which is valuable for surgical or interventional planning.[28]

Management and Outcome: The majority of patient's recovered (85%) in the various studies, who received medical therapy before any intervention, consistent with previous reports.[28] The management included octreotide, somatostatin, total parentralnitrition and antibiotics.

Conclusion

Imaging modalities, particularly CT and MRCP, play essential role in prompt procedural diagnosis of PPF. If ERCP done early, this will be a safe option to avoid surgical therapy.

Author's Contribution: Jyoti Verma did the plan of investigations and management, Ankit singh have contribution in interdepartmental references and timely as required inward management and Tabish abbasi have contribution in sampling and collection of reports.

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