



A RARE CASE REPORT OF PANCREATICOPLURAL FISTULA WRONGLY DIAGNOSED AS PULMONARY TUBERCULOSIS

Dr. Shaheed Arman Khondekar*, Dr. Rajbir Singh and Dr. Arjun Sharma

Junior Resident, Department of Pulmonary Medicine, Government Medical College, Amritsar – 143001.

***Corresponding Author: Dr. Shaheed Arman Khondekar**

Junior Resident, Department of Pulmonary Medicine, Government Medical College, Amritsar – 143001.

Article Received on 28/05/2023

Article Revised on 18/06/2023

Article Accepted on 08/07/2023

ABSTRACT

Pancreaticopleural fistula (PPF) is a rare complication of pancreatitis that requires a high index of clinical suspicion as patients typically present with pulmonary symptoms related to the pleural effusion rather than pancreatitis. Diagnosis is made by detection of amylase in the pleural fluid. Magnetic resonance cholangiopancreatography can aid in visualizing the fistula. This usually presents with chest symptoms due to pleural effusion, pleural pseudocyst, or mediastinal pseudocyst, abdominal complaints are usually less or present late. Diagnosis requires a high index of clinical suspicion in patients who develop alcohol-induced pancreatitis and present with pleural effusion which is recurrent or persistent. Analysis of pleural fluid for raised amylase will confirm the diagnosis and investigations like CT, Endoscopic retrograde cholangiopancreatography (ERCP) or magnetic resonance cholangiopancreatography (MRCP) may establish the fistulous communication between the pancreas and pleural cavity. The optimal treatment strategy has traditionally been medical management with exocrine suppression with octreotide and ERCP stenting of the fistulous pancreatic duct. Operative therapy considered in the event patient fails to respond to conservative management.

KEYWORDS: pancreaticopleural fistula, pleural effusion, pancreatitis.

INTRODUCTION

Pancreaticopleural fistula (PPF) is a rare complication of acute and chronic pancreatitis, that occurs approximately in 0.4% of patients with pancreatitis.^[1] Due to chronic pancreatic inflammation, an abnormal connection known as fistula is formed between the pancreatic duct and the pleural space, leading to pancreatic secretion drainage into the pleura causing pleural effusion that is high in amylase. Patients typically present with pulmonary symptoms related to the pleural effusion rather than to pancreatitis leading to delay in diagnosis.^[1] Once the diagnosis is confirmed, treatment is usually directed to obliterate the fistula between the pancreas and the pleural.

Pancreaticopleural fistula has been recognized as a clinical entity since case reports were published in late 1960's.^[2] Since that time, pancreaticopleural fistulae and pancreatic ascites have been termed as internal pancreatic fistulae which share common pathogenesis which includes the disruption of main pancreatic duct, resulting in leakage of pancreatic fluid.^[3,4,5,6,7] This rare entity may be seen in patients with acute and chronic pancreatitis or may follow traumatic and surgical disruption of the pancreatic duct.^[3,4,5,6,7] It is characterized by massive pleural fluid and has a tendency to recur following treatment. While

conservative management with pancreatic duct stenting and inhibition of pancreatic secretion with octreotide may achieve closure of fistula in 31 to 45% of cases, surgery leads to healing in 80 to 90% of cases but carries a mortality up to 10%.^[3,4,5,6,7]

CASE REPORT

A 57 year old Asian male, chronic alcoholic for 20 years presented to the pulmonary medicine emergency department with chief complaints of pain abdomen for 20 days and shortness of breath for 7 days. The shortness of breath of subacute onset and gradually progressive in nature. There was no history of Paroxysmal Nocturnal Dyspnea (PND) or orthopnoea. There was no history of seasonal or diurnal variation. Patient's shortness of breath was relieved on lying down and on left lateral position. Shortness of breath increased on exertion. Pain abdomen was in the epigastrium region for 20 days acute onset and dull aching in nature with radiation of pain to the back. Pain reduced on stooping forward. There was associated mild grade fever along with the pain. Patient gave history of multiple left sided thoracentesis. On general physical examination patient had mild pallor with no clubbing, no pedal oedema, no icterus, no cyanosis, and no lymphadenopathy. Spo2-96% room air, Respiratory rate- 22/min, B.P- 118/74 mm of Hg.

RESPIRATORY EXAMINATION

Inspection - Chest shape and size normal, bilaterally symmetrical, decreased movement on left side with mild shift of trachea on the right side, with no visible veins over the chest wall, with no pulsation seen.

Palpation – Temperature was raised, movement of chest was reduced on left side, trachea was slightly shifted to the right side. Position of apex was normal and no tenderness was present on the chest.

Percussion – Stony dullness on left side of chest.

Auscultation – Bilateral air entry was present, reduced intensity on left side with no added sounds.

Gastrointestinal examination – No abnormal findings were found on inspection, palpation and auscultation.

Procedure done – Thoracentesis was done from left 6th intercostal space. 1.2 litres of cola coloured fluid aspirated and sent for investigation. After 2 days recurrent filling of pleural fluid was seen in chest X-ray and again thoracentesis was done.

LABORATORY REPORTS

Blood Reports: Hb- 8.6gm%, TLC – 9700, DLC – 80.3/11.8/7.9, RBC – 3.87×10^6 , Platelets – 3.67×10^3 ,

ESR – 140, CRP – 40, Absolute eosinophil count – 510, RBS – 471, Blood urea nitrogen – 20, Serum creatinine – 0.9, Serum bilirubin – 0.6, SGOT – 56, SGPT – 70, Total Serum Protein – 6.6, Differential serum protein – 4.6, Serum alkaline phosphatase – 70, Serum amylase – 522, Serum lipase – 975. Pleural fluid analysis – ADA – 34, protein 4.72, Glucose 107, Pleural fluid amylase – 183, Pleural fluid for cytology - occasional inflammatory cells with formed elements and some lymphocytes with few degenerated cells and some reactive mesothelial cells with no malignant cells seen.

Pleural fluid for CBNAAT: MTB not detected.

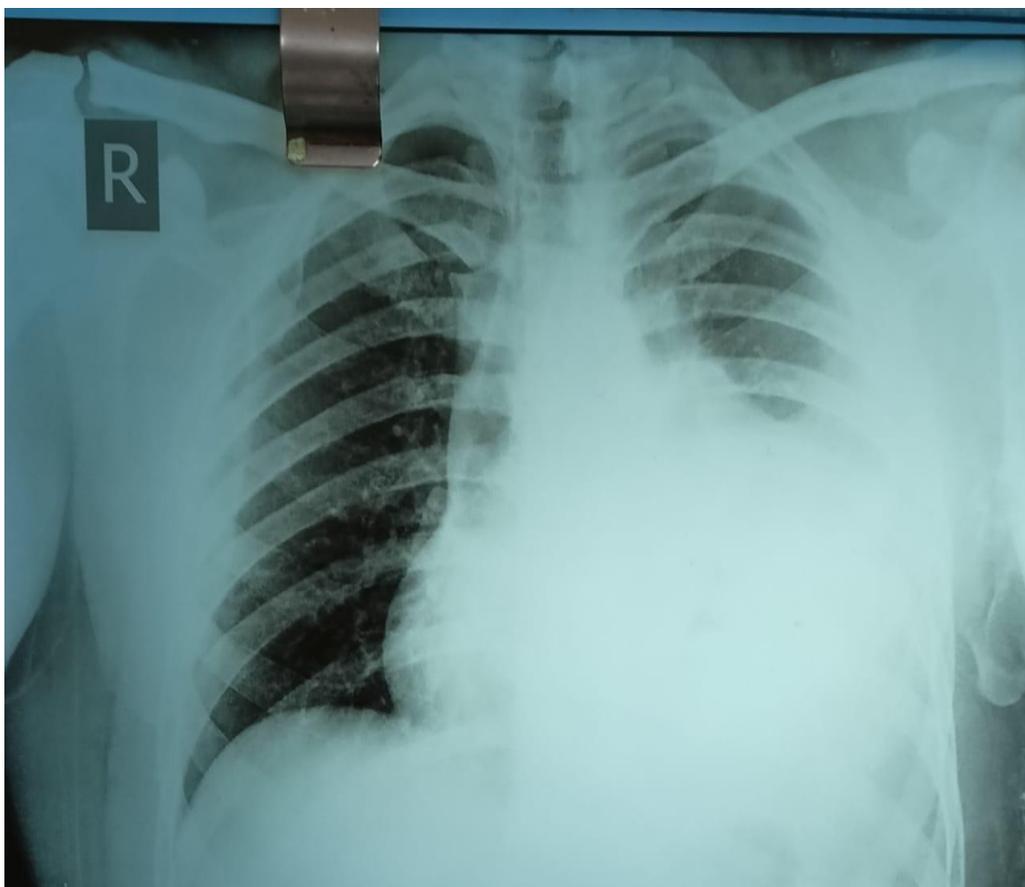
Sputum examination: Sputum for AFB negative.

Sputum for CBNAAT: MTB not detected.

Radiological investigation: HRCT thorax – gross left sided pleural effusion with near complete collapse of left lung resulting shift to right side.

CECT Abdomen: Finding suggestive of chronic pancreatitis.

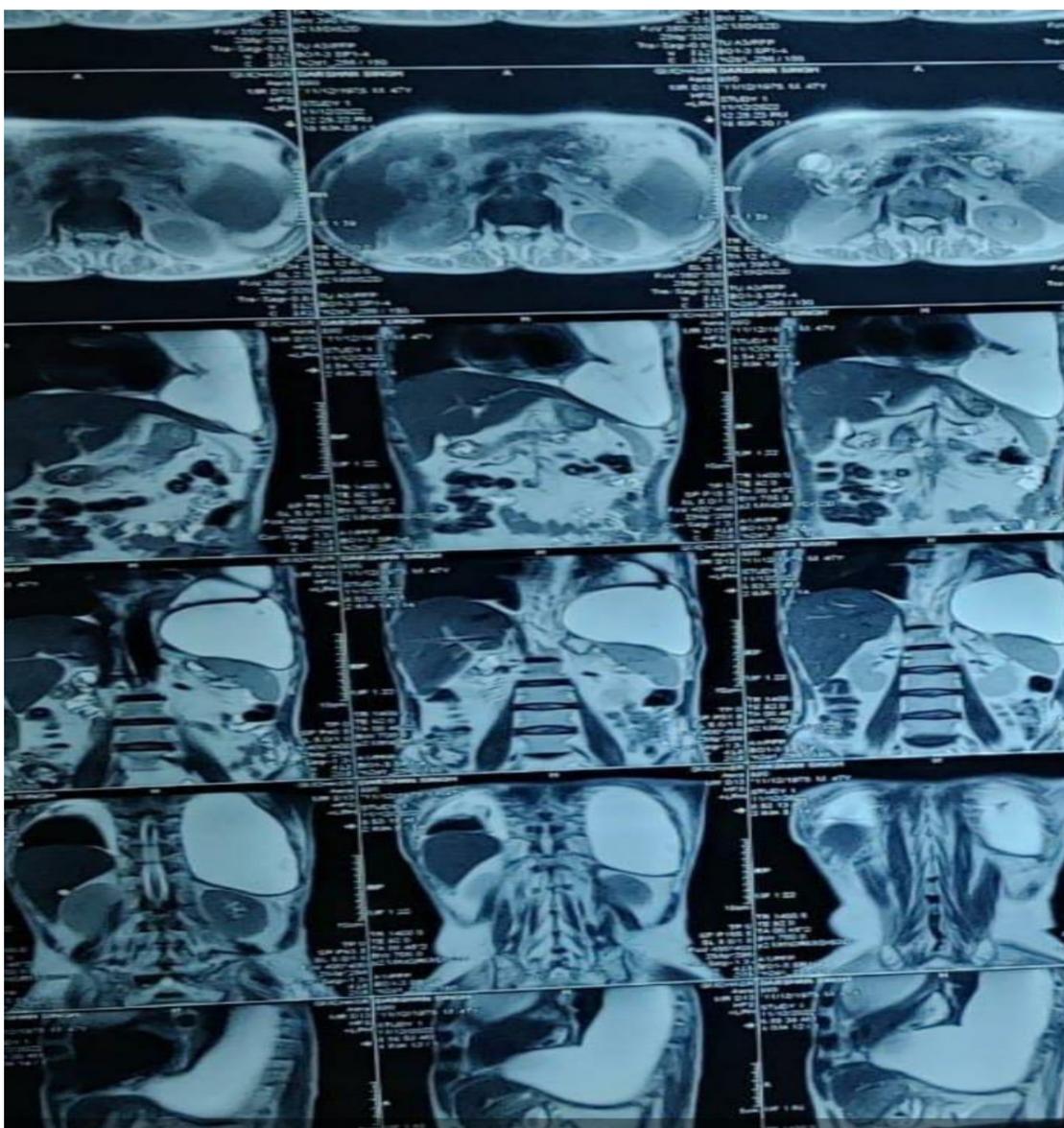
MRCP: Suggestive of atrophic pancreatitis (Cambridge grade IV) likely formation of pancreaticopleural fistula and resulting enlarged left sided pleural effusion.



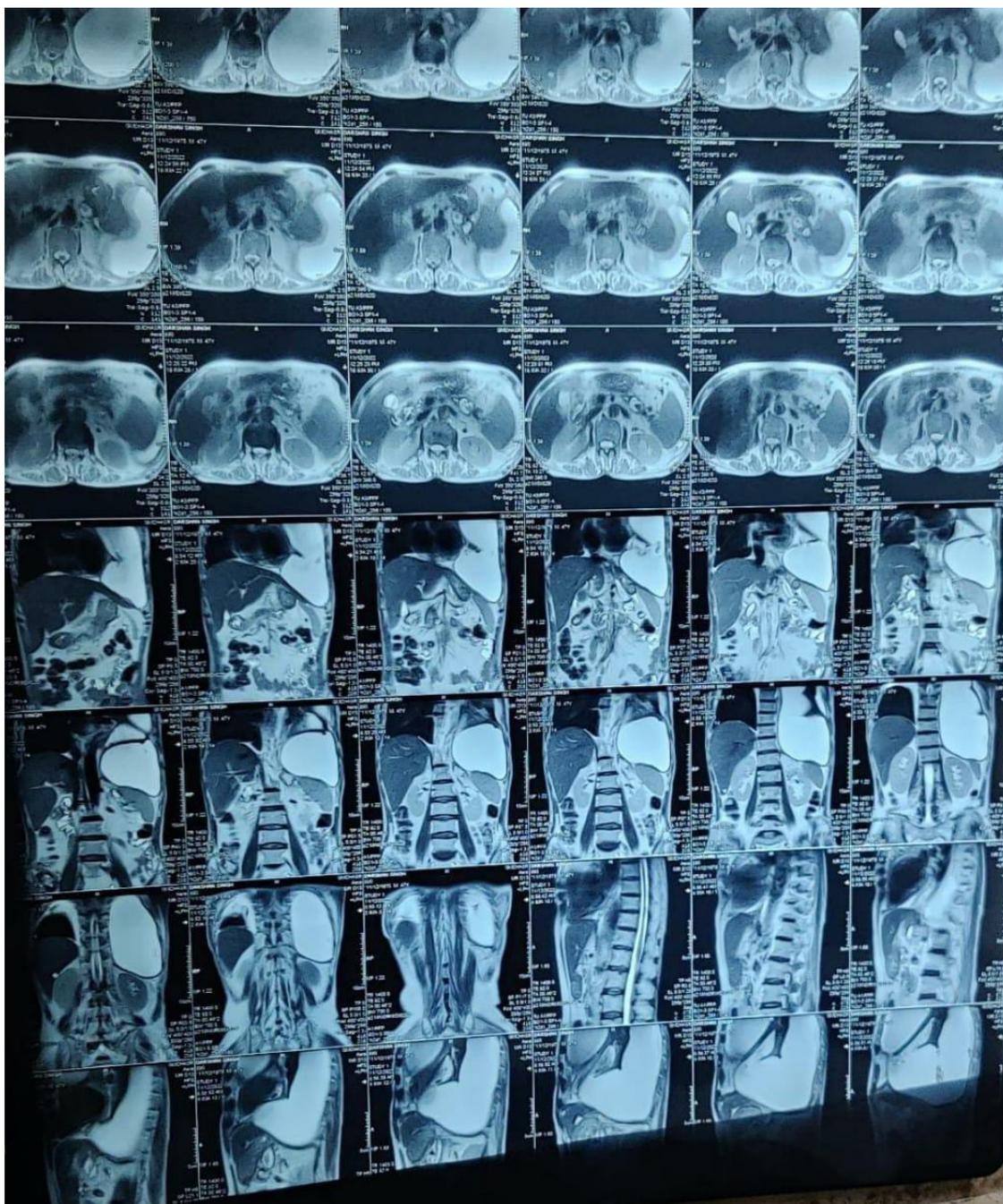
CHEST X-RAY PA VIEW SHOWING LEFT SIDED PLEURAL EFFUSION



COLA COLOURED PLEURAL FLUID AFTER THORACOCENTESIS



MRCP FILM SHOWING CHRONIC ATROPHIC PANCREATITIS CAMBRIDGE GRADE IV RESULTING IN LEFT SIDED PANCREATICO PLEURAL FISTULA



MRCP FILM SHOWING CHRONIC ATROPHIC PANCREATITIS CAMBRIDGE GRADE IV RESULTING IN LEFT SIDED PANCREATICO PLEURAL FISTULA.

DISCUSSION

Pancreaticopleural fistulas a rare complication of acute and chronic pancreatitis. Pancreatic ascites or pancreatic pleural effusion are initially identified based on CT or MRI imaging and are usually due to disruption of the main pancreatic duct, often by an internal fistula between the duct and the peritoneal cavity or a leaking pseudocyst. This diagnosis is suggested in a patient with a history of acute pancreatitis in whom the ascites or pleural fluid has both increased levels of albumin [>30 g/L (>3 g/dL)] and a markedly elevated level of amylase. An ERCP or magnetic resonance cholangiopancreatography (MRCP) confirms the clinical

suspicion and radiologic findings and often demonstrates passage of contrast material from a disrupted major pancreatic duct or a pseudocyst into the peritoneal cavity. The differential diagnosis of pancreatic ascites should include intraperitoneal carcinomatosis, tuberculous peritonitis, constrictive pericarditis, and Budd-Chiari syndrome.^[8]

If the pancreatic duct disruption is posterior, an internal fistula may develop between the pancreatic duct and the pleural space, producing a pleural effusion (pancreaticopleural fistula) that is usually left-sided and often massive. If the pancreatic duct disruption is

anterior, amylase and lipase rich peritoneal fluid accumulate (pancreatic ascites). A leaking, disrupted pancreatic duct is best treated by ERCP and "bridging" stent placement and infrequently requires thoracentesis or chest tube drainage.^[8]

Treatment may also require enteral or parenteral alimentation to improve nutrition. If ascites or pleural fluid persists after two to three weeks of medical management, and the disruption is unable to be stented, the patient should be considered for surgical intervention after retrograde pancreatography to define the anatomy of the disrupted duct.^[8]

CONCLUSION

In this case patient was wrongly started on antitubercular drugs on clinicoradiological basis without any clinical improvement. After patient came to our pulmonary medicine department, we arrived at a final diagnosis of left sided pancreaticopleural fistula due to chronic pancreatitis. Patient was managed conservatively and referred to surgery department for further management.

REFERENCES

1. Tay CM, Change SK: Diagnosis and management of pancreaticopleural fistula. *Singapore Medical J*, 2013, 54: 190-194. 10.11622/smedj.2013071.
2. Anderson WJ, Skinner DB, Zuidema GD, Cameron JL. Chronic pancreatic pleural effusions. *Surgery Gynecology and Obstetrics*, 1973; 137(5): 827–830.
3. King JC, Reber HA, Shiraga S, Hines OJ. Pancreatic-pleural fistula is best managed by early operative intervention. *Surgery*, 2010; 147(1): 154–159.
4. Sut M, Gray R, Ramachandran M, Diamond T. Pancreaticopleural fistula: a rare complication of ERCP-induced pancreatitis. *Ulster Medical Journal*, 2009; 78(3): 185–186.
5. Dhebri AR, Ferran N. Nonsurgical management of pancreaticopleural fistula. *Journal of the Pancreas*, 2005; 6(2): 152–161.
6. Vyas S, Gogoi D, Sinha SK, Singh P, Yadav TD, Khandelwal N. Pancreaticopleural fistula: an unusual complication of pancreatitis diagnosed with magnetic resonance cholangiopancreatography. *Journal of the Pancreas*, 2009; 10(6): 671–673.
7. Safadi BY, Marks JM. Pancreatic-pleural fistula: the role of ERCP in diagnosis and treatment. *Gastrointestinal Endoscopy*, 2000; 51(2): 213–215.
8. Greenberger NJ, Conwell DL, Wu BU, Banks PA. Acute and chronic pancreatitis. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J. *Harrison's Principles of Internal Medicine*, 18th Edn., Vol. 2, McGraw Hill, 2012; 2634-48.