ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 11 | November - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

UNVIELING THE HIDDEN PATHWAY:A RARE CASE OF PANCREATICOPLEURAL FISTULA PRESENTING AS RECURRENT PLEURAL EFFUSION

Medicine

Dr. Edwin Sam Varghese Junior Resident

Dr. Shama Prakash K Professor And Head Of Unit, Department Of General Medicine

ABSTRACT

Pancreaticopleural fistula (PPF) is a rare diagnosis requiring a high index of clinical suspicion as it presents with respiratory rather than abdominal symptoms. Elevated pleural fluid amylase clinches the diagnosis. Magnetic Resonance Cholangiopancreatography (MRCP) is the investigation of choice. First line treatment is conservative while operative management is considered if medical management fails. Here we briefly describe a case of recurrent pleural effusion secondary to a pancreaticopleural fistula and how we approached and managed the case.

KEYWORDS

CT-Computed tomography, ERCP-Endoscopic retrograde cholangiopancreatography, IAC-Indian Academy of Cytologists, IV-Intravenous, Kg-Kilogram, MRCP-Magnetic resonance cholangiopancreatography, PPF-Pancreaticopleural fistula.

INTRODUCTION:

Pleural effusion in acute pancreatitis is self-limiting, however in chronic pancreatitis maybe due to a PPF.PPF typically presents as massive pleural effusion. As PPF is rare, there is still ambivalence in diagnostic as well as management protocols.

Incidence And Pathogenesis:

PPF occurs in 0.4% of patients presenting with pancreatitis and 4.5% of patients with pancreatic pseudocyst[4].Other causes include idiopathic pancreatitis, cholelithiasis, trauma and pancreatic duct anomalies.

Incidence of pleural effusion in PPF: Left sided-42-67%, Right sided-19-40% and Bilateral-14-17%[4].

Development of PPF is usually a consequence of leak from pseudocyst or due to direct pancreatic duct leak[5].If leak occurs anteriorly, pancreaticoperitoneal fistula develops causing ascites. If leak occurs posteriorly, secretions enter retroperitoneum and may dissect through aortic/oesophageal hiatus to form a PPF. Rarely pancreaticoperi cardial, pancreaticobronchial or pancreatico-oesophageall fistula's may develop[4].

Clinical Features: Men between age of 40 and 50 years are mainly affected with history of chronic alcoholism in 50%[2].

Most common symptom is breathlessness. PPF related pleural effusion is typically refractory to drainage and tends to accumulate rapidly[1].

Other symptoms include: Cough, chest pain, fever, weight loss and haemoptysis. Abdominal pain is rare.

Diagnosis:

Pleural fluid amylase is significantly elevated (>1000 U/L).

Computed tomography (CT), Endoscopic Retrograde Cholangiopancreatography (ERCP) and MRCP are important diagnostic modalities with sensitivity being 47,78 and 80% respectively[4].

MRCP is the radiological investigation of choice[3] as it is non-invasive and we can visualize fistula even beyond strictures and study duct morphology.

Management:

Conservative management is treatment of choice especially in patients with co-morbidities[2].Surgery though definitive, is reserved for those who fail to respond to optimal medical or endoscopic treatment and for those with complete ductal obstruction or in cases where a stent may not be placed.

Medical therapy is with Therapeutic thoracocentesis/Intercostal chest drain insertion with somatostatin analogues. Optimal management includes ERCP with stenting of fistulous pancreatic duct[5].

Case Study:

A 44-year-old male known case of Systemic Hypertension and Type 2 Diabetes Mellitus (on medications) with history of significant alcohol consumption and smoking for 20 years presented to us with Progressive breathlessness (Modified Medical Research Council Grade I->III),Dry cough and unintentional weight loss of 7 kg for 1 month. For the same complaints, he had visited a hospital in a rural setting at his hometown where he was diagnosed with left sided pleural effusion and he was treated with oral antibiotics and thoracocentesis of 3 litres of haemorrhagic fluid was done which showed 80% of lymphocytes. He was symptomatically better but however symptoms recurred in 5 days and repeat Chest x ray showed reaccumulation of fluid, hence was referred to our centre in view of possible malignancy for further management. He also has a past history of pancreatitis and right sided pleural effusion which was managed conservatively with IV Antibiotics and therapeutic thoracocentesis 5 months back.

MATERIALS AND METHODOLOGY:

On examination he was moderately built, but poorly nourished and had tachypnoea. Systemic examination was unremarkable except for decreased intensity of breath sounds in bilateral lower lung fields with decreased vocal fremitus and resonance. Chest X-Ray done showed blunting of both costophrenic angles. Serum lipase sent was grossly elevated.Ultrasound Abdomen done showed large hypoechoic lesion (6.4 X 6.1 cm) in epigastric region posterior to pancreas with bilateral large pleural effusions. Therapeutic and Diagnostic thoracocentesis of 1 litre of fluid done from left side revealed fluid to be brown coloured and of exudative nature with IAC system of reporting fluid cytology being Category II-Benign. Contrast enhanced CT thorax done showed fistulous tract containing hypodense fluid extending from pancreatic tail through aortic hiatus communicating with bilateral pleural cavities-PPF. Visualised pancreas was atrophic with segmental prominence of MPD. Sputum Culture sensitivity sent showed growth of Klebsiella pneumoniae and Pseudomonas aeruginosa. Repeat diagnostic thoracocentesis was done and sent for Amylase levels and culture. Pleural fluid culture sent showed no growth however amylase levels were found to be grossly elevated and hence diagnosis of PPF induced pleural effusion was confirmed. Patient was treated with IV Antibiotics according to sensitivity pattern and underwent diagnostic and therapeutic ERCP which confirmed the diagnosis and pancreatic sphincterotomy with MPD stenting was done beyond the disrupted segment following which prompt drainage of pancreatic secretions and contrast was noted.

He was then initiated on IV Octreotide and Pancreatic enzyme supplementation. Once symptomatically better he was discharged after IV antibiotic therapy of adequate duration with oral antibiotics and pancreatic enzyme supplements. Patient was then followed up after 2 weeks with repeat Complete blood counts and Chest X-Ray which showed normal total counts with resolution of bilateral pleural effusions.

Table 1-Laboratory Investigations

Haematology: Total counts	10440
INR	1.1
Biochemistry: Fasting blood Sugar	103
Post prandial blood sugar	97
Liver function test, Renal function test, TSH	Within normal limits
Serum. Albumin	3.5
Serum Lipase/Serum LDH	1539 (Normal-<300),324
Electrocardiogram,2D Echocardiography	Within normal limits, Ejection fraction-60%
Microbiology: HIV/HbsAg, HCV	Non-reactive
Pleural fluid: Cytology	Category II-Benign
Sugar/Protein/LDH/ADA, Amylase	121/4.5/693/20.5/2183
Cultures: Pleural fluid	Negative
Sputum	Klebsiella pneumoniae and Pseudomonas Aeruginosa

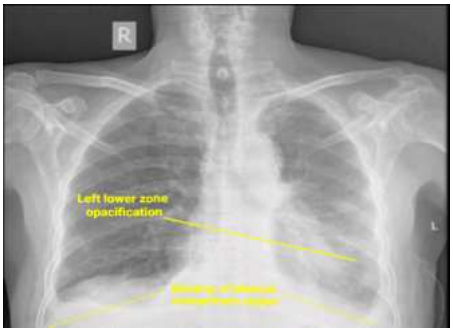

Figure 1- Initial Chest X-RAY

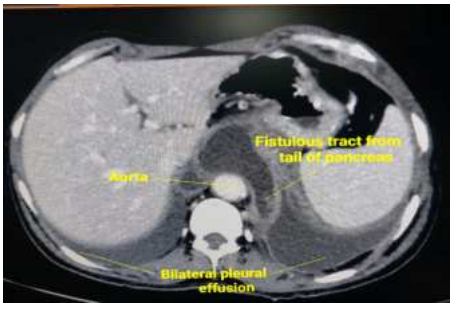

Figure 2- Axial section of CT Thorax

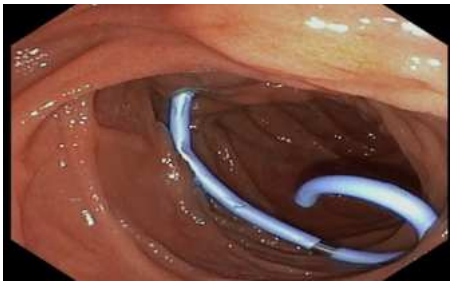

Figure 3- Main pancreatic duct with stent insitu

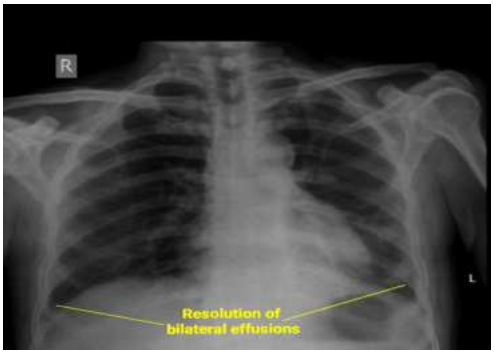

Figure 4- Follow up Chest X-RAY

DISCUSSION:

PPF induced pleural effusion is typically rapidly accumulating, recurrent and exudative in nature. Numerous pathologies such as Pulmonary tuberculosis, Lung Adenocarcinoma, Liver cirrhosis and leukaemia/lymphoma may cause elevated pleural fluid amylase[3].Hence diagnosis requires high pleural fluid amylase in the appropriate clinical setting.

Summary:

Middle aged males with history of chronic alcoholism/pancreatitis and recurrent pleural effusions may need to be suspected of having a PPF if pleural fluid analysis showed elevated amylase. Diagnosis requires a well-structured approach with strong clinical suspicion. While medical management remains mainstay of treatment, there are others who support early surgery to reduce recovery time and increase success rate[5].

REFERENCES:

1) Khadka, M., Bhusal, S., Pantha, B., Gautam, R., Gautam, K., & Chaudhary, A. (2024). Pancreaticopleural fistula causing pleural effusion: a case report and review of the literature. Journal of medical case reports, 18(1), 131. https://doi.org/10.1186/s13256-024-04457-8
2) Raab, S., Aigner, C., Kurz, F., & Shamiyeh, A. (2024). Minimally invasive treatment of an internal pancreaticopleural fistula with massive pleural effusion: a case report. Journal of medical case reports, 18(1), 430. https://doi.org/10.1186/s13256-024-04761-3
3) Aswani, Y., & Hira, P. (2015). Pancreaticopleural fistula: a review. JOP : Journal of the pancreas, 16(1), 90–94. https://doi.org/10.6092/1590-8577/2915
4) Tay, C. M., & Chang, S. K. (2013). Diagnosis and management of pancreaticopleural fistula. Singapore medical journal, 54(4), 190–194. https://doi.org/10.11622/smedj.2013071
5) Machado N. O. (2012). Pancreaticopleural fistula: revisited. Diagnostic and therapeutic endoscopy, 2012, 815476. https://doi.org/10.1155/2012/815476