

A Case of Peripancreatic Tuberculous Lymphadenitis Mimicking Pancreatic Malignancy: Diagnosis by Endoscopic Ultrasound-guided Core Biopsy

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ABSTRACT

Pancreatic and peripancreatic Tuberculosis (TB) are uncommon and may closely resemble pancreatic malignancy on imaging. The authors report a 65-year-old immunocompetent male who presented with epigastric pain, weight loss of 5-6 kg, and malaise. Contrast-Enhanced Computed Tomography (CECT) of the abdomen showed a 2.5×1.9×1.7 cm hypoenhancing lesion in the pancreatic head with multiple enlarged peripancreatic lymph nodes showing central necrosis, raising suspicion of pancreatic malignancy. Positron Emission Tomography-Computed Tomography (PET-CT) showed an Fluorodeoxyglucose (FDG)-avid necrotic lesion adjacent to the pancreatic head and neck, along with multiple FDG-avid necrotic peripancreatic lymph nodes and mildly FDG-avid mediastinal and right hilar lymph nodes. Endoscopic Ultrasound (EUS) demonstrated conglomerated peripancreatic lymph nodes measuring approximately 3×1 cm with central calcification. EUS-guided core biopsy showed necrotising granulomatous inflammation, and GeneXpert performed on the tissue was positive for *Mycobacterium tuberculosis*. The patient was started on anti-tubercular therapy and showed significant clinical improvement, with complete resolution of the lesions on follow-up PET-CT after six months. The present case illustrates the difficulty in distinguishing peripancreatic tuberculosis from pancreatic malignancy based on imaging alone and highlights the importance of EUS-guided tissue acquisition with molecular testing in establishing the diagnosis and guiding appropriate treatment.

Keywords: Endosonography, Fine needle biopsy, Granulomatous inflammation, Peripancreatic lymphadenitis

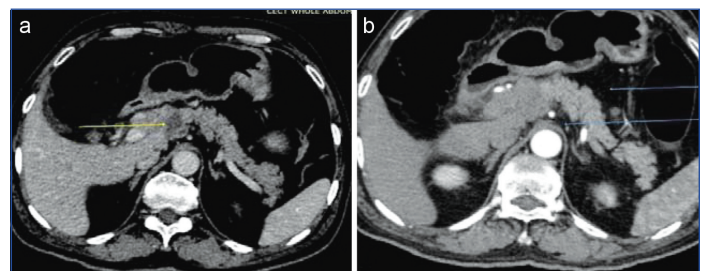
CASE REPORT

A 65-year-old male agricultural labourer from Andhra Pradesh, India, presented with abdominal pain for 20 days. His abdominal pain was of insidious in onset, progressing slowly, mainly localised to epigastrium and left hypochondrium, having a dull aching character, intermittent, and associated with meals. The patient also reported malaise, and an unintentional weight loss of approximately 5-6 kg over one month. There were no symptoms of fever, vomiting, jaundice, abdominal distention, melena, prior hospital admission, surgery, or any other significant medical illnesses. The patient denied tobacco and alcohol use.

On examination, the patient was afebrile with stable vital signs. Abdominal examination revealed epigastric tenderness without palpable mass or organomegaly. No peripheral lymphadenopathy was noted. Laboratory investigations showed haemoglobin (12.9 g/dL) level, normal leucocyte count (10050 cells/cumm) and platelet counts (3.59 lacs/cumm) and elevated erythrocyte sedimentation rate (35 mm/hr). A contrast enhanced computed tomography scan of the abdomen showed a well-defined hypoenhancing lesion with dimensions of 2.5×1.9×1.7 cm involving the head of the pancreas. Multiple enlarged peripancreatic lymph nodes with central non enhancing necrotic areas were noted, raising suspicion for pancreatic head malignancy with nodal involvement [Table/Fig-1]. Serum tumour markers including carbohydrate antigen 19-9, carcinoembryonic antigen, and alpha-fetoprotein were found to be within normal limits. All laboratory investigations have been represented in [Table/Fig-2].

A whole body PET-CT was performed, which showed several fluorodeoxyglucose-avid necrotic lymph nodes adjacent to the pancreatic head and neck. An ill-defined fluorodeoxyglucose-avid necrotic lesion was indenting the superior border of the pancreatic neck which could either represent an exophytic lesion

of the pancreas or conglomerated suprapancreatic lymph nodes. Additionally, a few mildly enlarged fluorodeoxyglucose-avid mediastinal and right hilar lymph nodes were noted [Table/Fig-3].

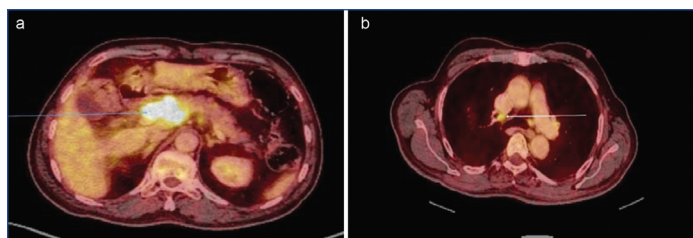


[Table/Fig-1]: a) Contrast-enhanced computed tomography (axial view) showing a hypoenhancing lesion in the pancreatic head (yellow arrow); b) Contrast-enhanced computed tomography (axial view) showing peripancreatic necrotic lymph nodes (blue arrows)

Parameters	Patient value	Reference range
Haemoglobin	12.9 g/dL	13-17 g/dL
Total leukocyte count	10050 cells/cumm	4,000-11,000 cells/cumm
Platelet count	3.59 lacs/cumm	150,000-450,000 /cumm
ESR	35 mm/hr	<15 mm/hr
CA 19-9	3.32 U/mL	<34 U/mL
CEA	1.49 ng/mL	<3.4 ng/mL
AFP	3.23 ng/mL	<7 ng/mL
HIV serology by ELISA	Non reactive	Non reactive
Mantoux test	Positive (15 mm)	Negative

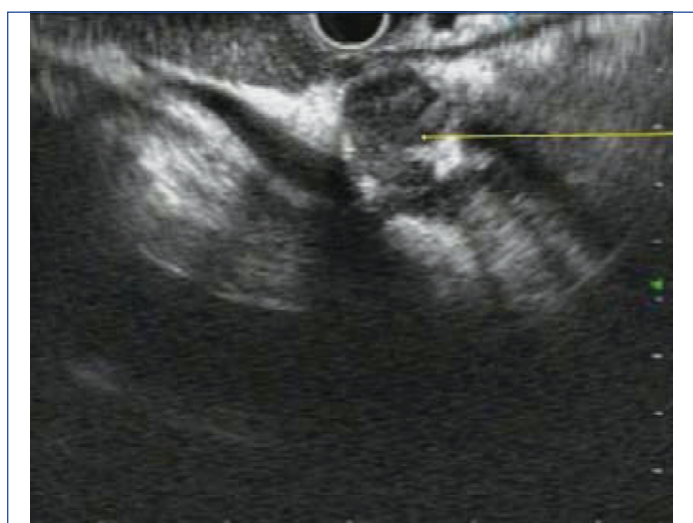
[Table/Fig-2]: Laboratory investigations.

Footnote: ESR: Erythrocyte sedimentation rate; CA 19-9: Carbohydrate antigen 19-9; CEA: Carcinoembryonic antigen; AFP: Alpha-fetoprotein; HIV: Human immunodeficiency virus; ELISA: Enzyme-linked immunosorbent assay



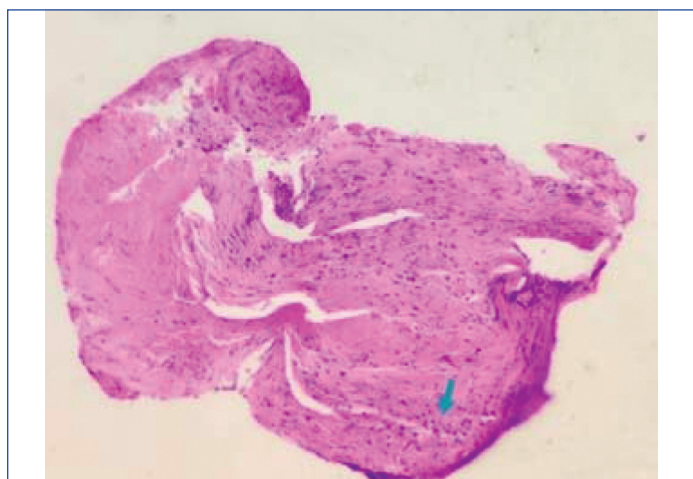
[Table/Fig-3]: Baseline Positron emission tomography-computed tomography (axial) demonstrating fluorodeoxyglucose-avid necrotic lesions: a) Around the pancreatic head and neck (blue arrow); b) Mediastinal lymphadenopathy (white arrow).

In view of concern for pancreatic malignancy, EUS was performed. At the duodenal bulb station, multiple conglomerate peripancreatic lymph nodes measuring approximately 3×1 cm and showing central calcification were visualised [Table/Fig-4]. EUS-guided core biopsy was performed using a 22-gauge Franseen fine-needle biopsy needle using the slow-pull technique with multiple to-and-fro movements under continuous EUS guidance. Three passes were performed to obtain adequate core tissue for histopathological examination and GeneXpert.



[Table/Fig-4]: Endoscopic Ultrasound (EUS) image showing conglomerate peripancreatic lymph nodes with central calcification (yellow arrow).

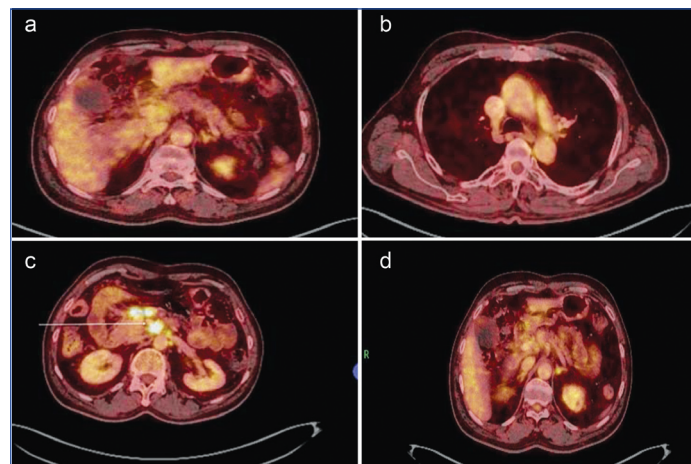
Histopathological examination demonstrated extensive necrosis with poorly formed granulomas, consistent with necrotising granulomatous inflammation indicating tuberculosis [Table/Fig-5]. GeneXpert MTB performed on the biopsy specimen detected *Mycobacterium tuberculosis* complex. Based on the histopathological, microbiological, and clinicoradiological findings, a diagnosis of peripancreatic tuberculous lymphadenitis was established. Subsequent evaluation revealed a positive Mantoux test with an induration of 15 mm at the end of 48 hours. Human



[Table/Fig-5]: Histopathology demonstrating necrotising granulomatous inflammation with poorly formed granuloma (blue arrow) (H&E, ×40).

immunodeficiency virus serology was non-reactive, and there was no clinical evidence of immunosuppression [Table/Fig-2].

The patient was commenced on standard anti-tubercular therapy. He demonstrated significant symptomatic improvement and weight gain during follow-up. Repeat PET-CT performed after six months of therapy showed complete resolution of the previously identified abdominal lymphadenopathy and fluorodeoxyglucose-avid lesions in mediastinum and right hilum [Table/Fig-6].



[Table/Fig-6]: Follow-up positron emission tomography-computed tomography (axial) after six months of anti-tubercular therapy showing complete resolution of previously noted lesions. a) Resolution of abdominal lymphadenopathy; b) resolution in size and metabolic activity of mediastinal lymph nodes as compared to before anti-tubercular therapy; c) Baseline PET-CT demonstrating an FDG-avid lesion in the pancreatic head/peripancreatic region with multiple FDG-avid necrotic peripancreatic lymph nodes (white arrow); d) Follow-up PET-CT after six months of anti-tubercular therapy showing complete metabolic resolution of the pancreatic/peripancreatic lesion and abdominal lymphadenopathy.

DISCUSSION

Pancreatic and peripancreatic tuberculosis are uncommon forms of extrapulmonary tuberculosis that frequently pose a diagnostic challenge because of their close resemblance to pancreatic neoplasms [1-4]. Common symptoms include abdominal pain, anorexia, weight loss, fever, and in some cases, obstructive jaundice. Lack of specificity in the clinical picture is a common cause of delayed diagnosis. Radiologically, pancreatic tuberculosis may appear as a hypoenhancing pancreatic mass, cystic lesion, or necrotic lymphadenopathy. The presence of peripancreatic necrotic lymph nodes may suggest an infectious aetiology; however, these findings are not sufficient to exclude malignancy [3,5]. Furthermore, fluorodeoxyglucose uptake on PET-CT is frequently observed in both malignant and inflammatory conditions, limiting its discriminatory value [6].

Several cases reported in the literature have described pancreatic or peripancreatic tuberculosis mimicking pancreatic malignancy [2-4]. Patients commonly presented with abdominal pain, weight loss, fever, and a pancreatic mass or peripancreatic lymphadenopathy, with many cases initially suspected to represent pancreatic cancer [1]. Sharma V et al., highlighted the difficulty in distinguishing pancreatic tuberculosis from malignancy based on clinical and radiological findings alone, particularly when a pancreatic mass is associated with lymphadenopathy [1]. In another report, pancreatic tuberculosis presenting as a pancreatic mass was diagnosed by EUS-guided tissue acquisition, demonstrating the value of minimally invasive sampling in avoiding unnecessary surgical intervention [2]. Similar cases have demonstrated that necrotising granulomatous inflammation in EUS-guided biopsy specimens, particularly when accompanied by microbiological evidence of tuberculosis, can establish the diagnosis in patients with imaging features suspicious for malignancy [7,8].

In the current case, the patient's age, substantial weight loss, pancreatic head lesion, and fluorodeoxyglucose-avid lymphadenopathy strongly favoured a diagnosis of pancreatic

malignancy. Normal tumour marker levels and presence of necrotic lymph nodes, however, prompted further evaluation. EUS is well suited to evaluate pancreatic lesions and adjacent lymphadenopathy. In addition to providing high resolution imaging, it facilitates tissue acquisition through fine needle aspiration or biopsy [7,8]. Tissue diagnosis is essential because imaging findings alone cannot reliably differentiate pancreatic tuberculosis from malignancy [6-8]. In the present case, EUS-guided core biopsy helped in establishing the presence of necrotising granulomatous inflammation. The dramatic clinical and radiological response to antitubercular therapy further supported the diagnosis. Importantly, tissue diagnosis prevented unnecessary pancreatic surgery. In the present case, the diagnosis was established by combining EUS-guided core biopsy, which demonstrated necrotising granulomatous inflammation, with GeneXpert confirmation of *Mycobacterium tuberculosis*. The positive GeneXpert result provided rapid microbiological confirmation and increased diagnostic confidence in differentiating tuberculosis from pancreatic malignancy. The subsequent complete clinical and radiological response to anti-tubercular therapy further supported the diagnosis. The present case underscores the importance of considering tuberculosis in the differential diagnosis of pancreatic masses, particularly in endemic regions. EUS-guided tissue acquisition should be pursued whenever imaging findings are atypical or when a potentially treatable benign condition is suspected.

CONCLUSION(S)

Peripancreatic tuberculosis can closely mimic pancreatic malignancy clinically and radiologically, even in immunocompetent individuals. In patients with a pancreatic mass and necrotic peripancreatic

lymphadenopathy, tuberculosis should be considered, particularly in endemic regions. EUS-guided core biopsy with histopathological and microbiological evaluation, including GeneXpert, can provide a definitive diagnosis and prevent unnecessary surgical intervention. Early recognition and appropriate anti-tubercular therapy can result in complete clinical and radiological resolution.

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