Abdominal Pain, Duodenal Ulcer, and Portal Hypertension—An Atypical Pancreatic Mass Postprint

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Abstract

A 28-year-old male patient was admitted to the Department of Gastroenterology, Peking Union Medical College Hospital, on April 26, 2019, due to "intermittent upper abdominal pain for 8 months, worsening with low-grade fever and night sweats for 1 month, and melena for 3 days."

Full Text

Preamble

Title: Abdominal Pain, Duodenal Ulcer, and Portal Hypertension: An Unusual "Pancreatic Mass"

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1 Case Summary

1.1 Present Illness

A 28-year-old male was admitted to the Department of Gastroenterology at Peking Union Medical College Hospital on April 26, 2019, with a chief complaint of "intermittent epigastric pain for 8 months, worsening with low-grade fever and night sweats for 1 month, and melena for 3 days."

In October 2018, the patient developed persistent colicky pain in the right upper quadrant after fasting, rated 5/10 on the Numeric Rating Scale, without radiation. The pain could be relieved within 30 minutes of eating and was not accompanied by fever, vomiting, or melena. In January 2019, he presented to an outside hospital where laboratory tests showed normal complete blood count; total bilirubin (TBil) 4.7 mol/L, alanine aminotransferase (ALT) 52 U/L, aspartate aminotransferase (AST) 43 U/L; erythrocyte sedimentation rate (ESR) 58 mm/h, hypersensitive C-reactive protein (hsCRP) 31.17 mg/L; mixed lymphocyte culture with interferon (N) 0.08 IU/mL, (T) 0.76 IU/mL, (M) >10.00 IU/mL; Mycobacterium tuberculosis gamma-interferon (T-N) 0.68 IU/mL, (M-N) >10.00 IU/mL. Contrast-enhanced abdominal CT revealed a mass in the pancreatic head region. Endoscopic ultrasound showed a hypoechoic mass behind the pancreatic neck with liquefaction and calcification, and splenic vein dilation. Gastroscopy revealed a giant ulcer at the duodenal bulb-descending junction. Pathology showed granulomatous inflammation adjacent to the ulcer necrotic layer with numerous epithelioid cells; acid-fast staining was negative. The patient was treated with esomeprazole for acid suppression and levofloxacin for infection. After one week, repeat gastroscopy showed no improvement. Ulcer pathology revealed suspicious caseous necrosis, equivocal positive acid-fast staining, and positive Mycobacterium tuberculosis gene detection. Considering tuberculosis as highly likely, quadruple anti-tuberculosis therapy was initiated on January 19 with isoniazid 0.3 g/day, rifampin 0.45 g/day, ethambutol 0.75 g/day, and pyrazinamide 0.75 g/day. After two weeks, thrombocytopenia developed (85 × 10 /L), prompting regimen adjustment to ethambutol 0.75 g/day + isoniazid 0.3 g/day + pyrazinamide 0.5 g TID. During this period, uric acid increased to 622 mol/L, treated with febuxostat and sodium bicarbonate. In early April, repeat tests showed normal complete blood count; TBil 77.6 mol/L, direct bilirubin (DBil) 45.3 mol/L, ALT 151 U/L, AST 100 U/L, alkaline phosphatase (ALP) 472 U/L, gamma-glutamyl transpeptidase (GGT) 816 U/L; ESR 18 mm/h, CRP 10.05 mg/L. Color Doppler ultrasound of the hepatobiliary system and spleen showed a mixed mass above the pancreatic head with moderate-to-severe stenosis of the main portal vein trunk, mild intra- and extrahepatic bile duct dilation, and splenomegaly. Considering the liver dysfunction, anti-tuberculosis therapy was suspended and hepatoprotective agents were added.

On April 9, the patient' s abdominal pain worsened, becoming persistent and distending, most prominent in the epigastric region with radiation to the back, accompanied by low-grade fever, fatigue, and night sweats (Tmax 38.3°C,

self-resolving, without chills or rigors). Outpatient tests at our hospital showed white blood cell (WBC) count 16.66 × 10 /L, neutrophils (NEUT) 90.1%, hemoglobin (Hb) 108 g/L, platelets (PLT) 243 × 10 /L; TBil 38.3 mol/L, DBil 27.9 mol/L, ALT 110 U/L, AST 46 U/L, ALP 372 U/L, GGT 426 U/L; ESR 86 mm/h; lymphocyte culture + interferon A+B: 0+32 FC/10S6MC; CA19-9 98.5 U/mL; abdominal CT plain scan + pancreatic thin-section scan showed enlarged pancreatic head with heterogeneous density, multiple enlarged peripancreatic lymph nodes, intra- and extrahepatic bile duct dilation, splenomegaly, and possible retroperitoneal lymph node enlargement. On April 22, the patient developed tarry black stools twice daily (approximately 50 mL each), accompanied by abdominal pain and fatigue, without hematemesis. Since onset, the patient had poor appetite and mental status, dark yellow urine, and 5 kg weight loss.

1.2 Past Medical History and Other History

Before onset, the patient experienced work-related fatigue and irregular lifestyle. He denied prior tuberculosis history or contact. He smoked for 5 years (currently 5 cigarettes/day). In childhood, he consumed unpasteurized milk. Family, marital, and reproductive history were unremarkable.

1.3 Physical Examination

Vital signs were stable. BMI 15 kg/m². Pale, anemic appearance. No palpable superficial lymphadenopathy. Clear breath sounds bilaterally, regular heart rhythm, no cardiac murmurs. Abdomen was firm, slightly distended, without abdominal wall varices. Tenderness was present in the right upper quadrant and epigastric region (+), without rebound tenderness or muscular rigidity. Liver and spleen were not palpable below the costal margins. Murphy' s sign (-). No hepatic percussion tenderness. Shifting dullness (+). Bowel sounds 6/min. Mild pitting edema of both lower extremities.

1.4 Laboratory Tests

Complete blood count: WBC 18.98 × 10 /L, NEUT% 90.0%, HGB 43 g/L, PLT 200 × 10 /L. Reticulocyte percentage 5.4%. Normocytic normochromic anemia.

Stool routine: White blood cells and red blood cells (-), occult blood (-).

Blood chemistry: Albumin 22 g/L, TBil 10.4 mol/L, ALT 16 U/L, urea 2.74 mmol/L, creatinine 46 mol/L, amylase 34 U/L, lipase 14 U/L.

Immunoglobulin panel: IgG 1670 mg/dL, IgM 71.8 mg/dL, IgA 191 mg/dL.

Iron studies: Serum iron 13 g/dL, total iron-binding capacity 78 g/dL, iron saturation 12.5%, transferrin saturation 16.7%, serum ferritin 342 ng/mL.

Coagulation profile: Prothrombin time 16.2 s, INR 1.39, fibrinogen 6.28 g/L, D-dimer 4.57 mg/L FEU, activated partial thromboplastin time 31.5 s.

Infectious disease panel: HBsAg (-), anti-HCV (-), anti-TP (-), HIV antibody screening (-).

Inflammatory markers: ESR 121 mm/h, hs-CRP 96.83 mg/L.

Tumor markers: CA19-9 4.6 U/mL, CEA normal.

1.5 Ascites Examination

Ascites routine: Appearance light yellow and slightly turbid, specific gravity 1.013, total cell count 529 × 10 /L, WBC count 321 × 10 /L, monocytes 38.0%, polymorphonuclear cells 62.0%. Rivalta test (-).

Ascites biochemistry: Chylous test (+), total protein 14 g/L, adenosine deaminase 0.7 U/L, albumin 6 g/L, lactate dehydrogenase 30 U/L, glucose 5.6 mmol/L, chloride 110 mmol/L, amylase 60 U/L, lipase 363 U/L.

Ascites microbiology: Lymphocyte culture + interferon (A+B): 232+592 FC/10S6MC; Mycobacterium tuberculosis/non-tuberculous mycobacteria nucleic acid testing, bacterial smear and culture, fungal smear and culture, acid-fast staining, Nocardia smear and Actinomyces culture were all (-).

Ascites tumor markers: CEA <0.200 ng/mL, CA19-9 <0.600 U/mL. Liquid-based cytology: No tumor cells identified.

1.6 Imaging Studies

Portal vein color Doppler ultrasound: Cavernous transformation of portal vein, probable embolism of main portal trunk and branches, with the pancreatic head mass encasing and narrowing the splenic vein and superior mesenteric vein.

Chest CT plain scan: Ground-glass opacity in right lower lobe dorsal segment, multiple linear opacities in both lungs, bilateral pleural effusions.

Abdominopelvic contrast-enhanced CT + pancreatic thin-section scan: Compared with previous images from April 9, new findings included massive ascites; patchy hypoenhancement in hepatic parenchyma during portal venous phase; nodular patchy hypoenhancement in hepatic segment S6, not excluding space-occupying lesion; enlarged pancreatic head with heterogeneous enhancement, worse than before; mild pancreatic duct dilation; poor opacification of main portal trunk and intrahepatic branches with cavernous transformation; multiple varices in esophagogastric and other portal venous tributaries; new low-density hypoperfusion in hepatic hilum; mild intra- and extrahepatic bile duct dilation with distal common bile duct encased and narrowed by abnormally enhancing vascular bundle after contrast; splenomegaly; possible retroperitoneal lymph node enlargement.

Electronic gastroscopy: Esophageal varices (moderate-to-severe), localized deep mucosal depression in post-bulbar duodenum. Pathology results: Post-bulbar duodenal mucosa showed acute and chronic inflammation, focal blunting of villi,

and lamina propria edema; special stains: acid-fast-TB (-), weak acid-fast staining (-).

[Figure 1: see original paper] Abdominal CT plain scan + pancreatic thin-section scan on April 9, 2019 showing enlarged pancreatic head with heterogeneous density (arrows). A. Coronal view; B. Axial view.

[Figure 2: see original paper] Abdominopelvic contrast-enhanced CT + pancreatic thin-section scan on April 29, 2019 showing enlarged pancreatic head with heterogeneous enhancement, worse than before (A, arrows); mild pancreatic duct dilation (B, arrows); poor opacification of main portal trunk and intrahepatic branches (C, arrows) with cavernous transformation; multiple varices in esophagogastric and other portal venous tributaries (D, arrows).

[Figure 3: see original paper] Gastroscopy showing severe esophageal varices with red color sign (A, arrows), post-bulbar duodenal mucosal depression with marked local mucosal congestion and edema (B, arrows).

2 Multidisciplinary Discussion

2.1 Gastroenterology

This young male patient had an insidious onset with a chronic course, presenting initially with abdominal pain that progressed to low-grade fever, fatigue, night sweats, and melena. Laboratory tests revealed severe anemia, while imaging showed a pancreatic head mass, duodenal ulcer, and portal hypertension. Analysis is as follows: (1) **Nature of the pancreatic head mass:** CT showed a heterogeneous mass in the pancreatic head region, and endoscopic ultrasound revealed a hypoechoic mass behind the pancreatic neck with liquefaction and calcification. Differential diagnosis includes: Pancreatic malignancy: The patient had prominent abdominal pain, rapid disease progression, significant wasting symptoms, elevated CA19-9, a hypoechoic space-occupying lesion in the pancreatic head region with possible duodenal invasion, encasement of peripancreatic splenic vein and superior mesenteric vein causing luminal narrowing, involvement of main portal trunk and branches, and multiple abdominal lymph nodes, raising suspicion for pancreatic malignancy. However, the patient was young, not in the typical age range for pancreatic cancer, and duodenal ulcer pathology showed tuberculosis infection without tumor evidence. Post-anti-tuberculosis therapy endoscopy showed significant improvement, inconsistent with malignancy. Pancreatic tuberculosis: The young male patient had prominent constitutional symptoms of tuberculosis (low-grade fever, fatigue, night sweats), history of unpasteurized milk consumption, positive Mycobacterium tuberculosis gamma-interferon test, positive PPD test, elevated ESR, and outside hospital duodenal ulcer pathology showing suspicious caseous necrosis, equivocal positive acid-fast staining, and positive TB-PCR. Repeat gastroscopy at our hospital after anti-tuberculosis therapy showed ulcer improvement with no further TB infection evidence, suggesting the duodenal ulcer was TB-related. Both pancreatic TB and tumors can appear as hypoechoic lesions on endoscopic ultrasound,

but calcification favors TB. From a unified disease perspective, the pancreatic head mass was most likely TB. While endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA) is increasingly valued for diagnosing pancreatic masses with good safety and ability to obtain multiple tissue samples for improved diagnostic accuracy, this patient' s lesion had numerous tortuous dilated vessels, posing high bleeding risk, so EUS-FNA was not pursued.

(2) **Nature of ascites:** The patient had massive ascites that was light yellow and slightly turbid, with serum-ascites albumin gradient >11 g/L, ascitic fluid specific gravity <1.018, negative Rivalta test, CT showing cavernous portal vein transformation with multiple varices in tributaries, splenomegaly, and moderate-to-severe esophageal varices on gastroscopy, suggesting portal hypertension-related ascites from pancreatic lesion involvement of the portal trunk and branches. Additionally, markedly elevated ascitic WBC count and significantly higher lymphocyte culture + interferon (A+B) levels in ascites than serum suggested concurrent tuberculous ascites. In summary, the ascites was considered mixed, including both portal hypertension-related and tuberculous components.

(3) **Cause of gastrointestinal bleeding:** The patient developed melena during the course. Outside hospital gastroscopy in January 2019 had shown duodenal ulcer, suggesting possible bleeding from that source. However, melena was newly developed after 3 months of anti-tuberculosis therapy when endoscopic duodenal ulcer had significantly improved, making it an unlikely explanation. Repeat gastroscopy at our hospital in April revealed new moderate-to-severe esophageal varices with red color signs, suggesting esophageal variceal rupture as the likely bleeding source. Propranolol 10 mg BID was prescribed for vasodilation and portal pressure reduction, with endoscopic therapy available if needed. The temporal concordance of gastrointestinal bleeding, esophageal varices, and ascites suggested progressive portal hypertension from pancreatic lesion-induced portal trunk stenosis/occlusion. Although imaging suggested possible portal trunk thrombosis, anticoagulation was not initiated due to recent bleeding and high risk.

(4) **Cause of jaundice:** The patient had predominantly direct bilirubin elevation with cholestatic enzymes, plus imaging showing pancreatic head mass with intra- and extrahepatic bile duct dilation, suggesting obstructive jaundice from the pancreatic lesion. However, jaundice appeared after anti-tuberculosis drug initiation and resolved spontaneously after discontinuing all anti-tuberculosis medications, without parallel improvement in pancreatic head mass size or bile duct dilation, suggesting drug-induced liver injury with intrahepatic cholestasis rather than obstructive jaundice.

2.2 Radiology

Based on abdominopelvic contrast-enhanced CT imaging analysis, the following features and differential considerations apply: (1) Compared with previous images from April 6, the pancreatic head region showed enlargement with ill-defined borders and heterogeneous enhancement, suggesting a space-occupying lesion such as pancreatic malignancy, inflammatory pancreatic lesions like pancreatic TB, or autoimmune pancreatitis. Autoimmune pancreatitis typically shows diffuse pancreatic enlargement ("sausage-like"appearance) involving head, body, and tail; focal autoimmune pancreatitis can involve only the head but rarely invades duodenum, inconsistent with this patient' s imaging. CT cannot reliably differentiate pancreatic malignancy from TB, as both can present as low-density masses involving surrounding tissues, lymph nodes, and vessels. Vascular involvement (narrowing and thrombosis) is more common in malignancy, but increasing literature reports pancreatic TB involving portal vein and its branches, which is not uncommon. Calcification is a common CT feature of TB. This patient' s CT showed a large, ill-defined pancreatic head mass with scattered punctate calcifications, possibly representing confluent peripancreatic lymph node TB encasing the pancreatic head. Compared with pancreatic TB, malignancy more frequently shows pancreatic duct dilation, which was not prominent in this patient.

(2) The new findings of poor portal trunk opacification with suspected thrombosis, cavernous transformation, multiple esophagogastric and portal tributary varices, splenomegaly, and ascites suggested portal hypertension secondary to pancreatic head mass involvement of the portal vein.

(3) The low-density hypoperfusion in hepatic hilum and distal common bile duct encasement/narrowing by abnormally enhancing vascular bundle after contrast explained the mild bile duct dilation. Based on these imaging features, the pancreatic head mass was favored to be pancreatic TB or peripancreatic lymph node TB, though pancreatic malignancy could not be excluded.

2.3 Pathology

The patient had two pathological examinations at our hospital: (1) In January 2019, outside hospital duodenal post-bulbar ulcer pathology was reviewed at our hospital, showing chronic small intestinal mucosal inflammation with epithelioid cell granulomatous nodules and coagulative necrosis, most commonly seen in mycobacterial infection. Non-tuberculous mycobacteria (NTM) infection can be difficult to differentiate pathologically from TB, also showing proliferative reactions with epithelioid cells and Langhans giant cell granulomas, but with weaker tissue reactions and less necrosis. Pancreatic or duodenal TB patients often cannot find or culture Mycobacterium tuberculosis in tissues. Typical pancreatic TB pathology shows caseous necrosis, epithelioid cells, Langhans giant cell granulomas, and positive acid-fast staining, with caseous necrosis and pos-

itive acid-fast staining being most diagnostic, though granulomas are common but non-specific. This pathology' s special stains did not identify pathogens; both acid-fast and weak acid-fast staining were negative, without definitive etiologic evidence for TB infection. However, even in confirmed TB cases, including pancreatic or duodenal TB, biopsies rarely show caseous necrosis, while acid-fast bacilli are more common in necrotic foci and difficult to find in granulomatous tissues. Degenerating bacilli on pathology sections may have weakened or absent staining, causing false negatives. With advances in molecular diagnostics, positive TB-PCR has become increasingly important for TB diagnosis. This patient' s positive TB-PCR at the outside hospital provided strong supportive evidence.

(2) In late April 2019, repeat duodenal post-bulbar mucosal pathology at our hospital after 3 months of anti-tuberculosis therapy showed no definite epithelioid granulomatous lesions or necrosis, with reduced local inflammatory background compared with January, suggesting treatment efficacy. Based on clinical presentation and our pathology results, duodenal ulcerative lesions were considered most likely TB infection.

2.4 Infectious Diseases

This young male had a chronic course with prominent constitutional TB symptoms, history of raw milk consumption, positive PPD test, elevated lymphocyte culture + interferon levels in both ascites and blood (higher in ascites), and duodenal ulcer pathology showing epithelioid cell granulomatous nodules with coagulative necrosis, consistent with TB infection, suggesting active TB infection in duodenum or retroperitoneum. The patient had negative infectious disease panel and normal immunoglobulin levels, without identified immunodeficiency. Pre-illness fatigue and irregular lifestyle may have been predisposing factors. Therefore, besides anti-tuberculosis therapy, lifestyle modification was needed. Abdominal TB treatment is identical to pulmonary TB: standard quadruple intensive therapy for 2 months with first-line drugs (isoniazid 5 [4-6] mg/kg/day, rifampin 10 [8-12] mg/kg/day, ethambutol 15 [15-20] mg/kg/day, pyrazinamide 25 [20-30] mg/kg/day), followed by maintenance therapy with isoniazid and rifampin for at least 4 months. Some patients experience adverse drug reactions including gastrointestinal symptoms, hepatotoxicity, allergic reactions, hyperuricemia, ototoxicity, and nephrotoxicity, with hepatotoxicity being most common and mostly manageable with supportive care. This patient developed liver dysfunction, thrombocytopenia, and hyperuricemia during quadruple therapy. Isoniazid, rifampin, and pyrazinamide can all cause hepatotoxicity; liver function normalized after rifampin discontinuation, suggesting rifampin-induced injury. Hyperuricemia was attributed to pyrazinamide, whose metabolite pyrazinoic acid inhibits uric acid excretion, potentially causing arthralgia and myalgia. Poor response to febuxostat and sodium bicarbonate suggested pyrazinamide discontinuation. Both rifampin and isoniazid can cause thrombocytopenia, with combined use potentially worsening myelosuppression. This patient' s platelets

decreased to 85 × 10 /L at the outside hospital but normalized after rifampin discontinuation, suggesting rifampin-induced myelosuppression. Per guidelines, anti-tuberculosis options included continuing isoniazid and ethambutol, adding an injectable anti-tuberculosis drug (amikacin preferred over streptomycin due to resistance rates, adverse effects, and availability), plus a later-generation fluoroquinolone such as levofloxacin. Close monitoring of blood counts, liver/kidney function, and hearing was required to watch for adverse effects.

2.5 General Surgery

Based on history and ancillary tests, pancreatic head mass was considered, with TB infection most likely, not excluding lymph node confluence. Pancreatic TB and peripancreatic lymph node TB have no specific clinical manifestations, both presenting with constitutional symptoms (low-grade fever, night sweats, fatigue, weight loss) and gastrointestinal symptoms (abdominal distension, pain, diarrhea). Due to strong bactericidal pancreatic secretions, primary pancreatic TB is rare. The pancreatic head region has abundant lymph nodes and lymphatics, making it a predilection site for lymph node TB, which can directly spread to the pancreatic head region. Therefore, peripancreatic lymph node TB is relatively common and often coexists with pancreatic TB. Pancreatic CT can show diffuse enlargement, multiple nodules, or focal types, with punctate calcifications within masses. Peripancreatic lymph node TB appears as enlarged peripancreatic lymph nodes, mainly in head/neck and body regions, with calcifications possible and ring enhancement on contrast scans; partial lymph node fusion shows "florid" changes. Based on imaging features, peripancreatic lymph node TB with concurrent pancreatic TB was favored. Pancreatic cancer rarely calcifies, shows heterogeneous enhancement, and is often accompanied by significant pancreatic duct and bile duct dilation. The current diagnosis favored TB infection without definite surgical indication. Medical anti-tuberculosis therapy could continue with close monitoring of pancreatic and peripancreatic lesions to determine further management based on treatment response.

3 Treatment

From April 27, the patient received anti-tuberculosis therapy with isoniazid 0.3 g/day PO + ethambutol 0.75 g/day PO + levofloxacin 0.5 g/day IV + amikacin 0.4 g/day IV, plus propranolol 10 mg BID for portal pressure reduction, Losec for acid suppression, furosemide for diuresis, iron supplementation, and IV nutritional support. The patient became afebrile with significantly improved abdominal pain, occasional postprandial bloating, no night sweats or fatigue, normal bowel movements, and 5 kg weight gain. Pre-discharge tests on May 20 showed WBC 3.81 × 10 /L, NEUT% 66.6%, HGB 82 g/L, PLT 84 × 10 /L; albumin 39 g/L, TBil 8.1 mol/L, ALT 36 U/L; ESR 35 mm/h, hs-CRP 2.54 mg/L.

4 Final Diagnosis

Pancreatic and peripancreatic lymph node tuberculosis, most likely, with duodenal and portal vein involvement, complicated by portal hypertension.

5 Treatment and Follow-up

After discharge, the patient continued regular anti-tuberculosis therapy with isoniazid 0.3 g/day PO + ethambutol 0.75 g/day PO + levofloxacin 0.5 g/day PO + amikacin 0.4 g/day IV, without fever, abdominal pain, or bloating, and stable weight. On August 7, 2019 (after nearly 6 months of total anti-tuberculosis therapy), follow-up tests showed WBC 3.31 × 10 /L, NEUT% 52.3%, HGB 133 g/L, PLT 61 × 10 /L; normal liver and kidney function; ESR 6 mm/h, hs-CRP 0.22 mg/L. Abdominopelvic contrast-enhanced CT + pancreatic thin-section scan showed resolution of ascites; previously seen large patchy hypoenhancement in hepatic parenchyma and nodular hypoenhancement in segment S6 were no longer evident; pancreatic head enlargement with heterogeneous enhancement remained largely unchanged; cavernous portal vein transformation with multiple collateral vessels was slightly more prominent; mild intra- and extrahepatic bile duct dilation had improved.

6 Discussion

This case involved a young male with chronic course, prominent clinical features of abdominal pain, fever, night sweats, and weight loss. Laboratory and imaging studies revealed a pancreatic head space-occupying lesion, most likely TB, possibly invading duodenum, with pancreatic lesion involvement of portal vein causing portal hypertension, secondary ascites, splenomegaly, esophagogastric varices, and subsequent gastrointestinal bleeding. After nearly 6 months of anti-tuberculosis therapy, clinical symptoms resolved, ascites disappeared, and inflammatory markers normalized, though the pancreatic head lesion showed no further progression.

Abdominal TB involving gastrointestinal tract, peritoneum, lymph nodes, and/or solid organs accounts for approximately 5% of all TB cases [1]. Peritoneal TB, intestinal TB, and hepatic TB are relatively common, while duodenal TB and pancreatic TB are rare, mostly secondary to pulmonary TB [2-3]. Pancreatic TB' s clinical and imaging features are often difficult to differentiate from pancreatic malignancy [4]; therefore, previous pancreatic TB diagnoses often came from surgical specimens of patients suspected to have pancreatic malignancy [5]. With advances in imaging technology and increased methods for obtaining pancreatic tissue, pancreatic TB diagnosis rates have significantly improved in recent years [3,6]. Currently, no multicenter clinical epidemiological studies have investigated pancreatic TB incidence and prevalence.

Possible etiologies of pancreatic TB include [7]: (1) Primary pancreatic TB

(rare); (2) Lymphatic and hematogenous dissemination, mostly from occult pulmonary TB hematogenous spread or retroperitoneal lymph node dissemination, with possible abdominal and peripancreatic lymph node TB, lesions favoring pancreatic head and uncinate process, possibly related to abundant blood supply and lymphatics in this region; (3) Disseminated TB with concurrent active pulmonary TB and abdominal TB, often with gastrointestinal involvement. This patient had no prior TB history or contact, and chest CT showed no definite pulmonary TB signs. Imaging and endoscopy suggested lesions involving pancreatic head and peripancreatic lymph nodes with unclear duodenal boundaries, and duodenal ulcer pathology confirmed TB. Therefore, pancreatic TB was considered likely disseminated from peripancreatic lymph node TB, invading duodenum and portal venous system.

Case series show pancreatic TB is more common in young to middle-aged males, with non-specific clinical manifestations [8], including constitutional TB symptoms (low-grade fever, fatigue, night sweats, appetite loss, weight loss) and gastrointestinal symptoms (abdominal distension, pain, jaundice). Some patients may have palpable abdominal masses and superficial lymphadenopathy [9]. Multiple imaging modalities can evaluate pancreatic TB: (1) Color Doppler ultrasound: simple and radiation-free, most pancreatic TB shows isoechoic or hypoechoic lesions but can be limited by bowel gas, potentially missing smaller lesions; can detect peripancreatic lymphadenopathy, ascites, bile duct dilation, and vascular lesions [9]. (2) Contrast-enhanced abdominal CT: more accurately assesses lesion extent, pancreatic calcifications, hepatobiliary lesions, and vascular involvement. Pancreatic TB mostly appears as low-density masses with peripheral enhancement [6], often in pancreatic head, involving surrounding lymph nodes and vessels, mimicking pancreatic malignancy, cystic lesions, or mass-forming pancreatitis, making differentiation difficult. In a small series of 9 pancreatic TB patients, CT showed pancreatic head masses in 5, pancreatic tail mass in 1, cystic lesions in 2, and pancreatic calcifications or splenic vein thrombosis in 2 [10]. Over half of patients have lesion calcifications [6,11], and many case reports document vascular involvement including portal vein, superior mesenteric vein, and hepatic artery. (3) Endoscopic ultrasound (EUS): high clinical value, accurately assesses pancreaticobiliary lesion size and extent, lymphadenopathy, ductal dilation, calcifications, and vascular invasion, most importantly enabling tissue acquisition via fine-needle aspiration for pathology and microbiology, especially for smaller lesions [12]. EUS shows pancreatic TB as hypoechoic lesions, difficult to differentiate from pancreatic adenocarcinoma, though cancer patients are older with higher bilirubin, more common bile duct dilation, and higher pancreatic duct dilation rates [11]. (4) MRI: mainly used to evaluate ductal compression by masses; pancreatic TB may appear hypointense on T1-weighted and hyperintense on T2-weighted images [13]. (5) PET-CT: rarely used for pancreatic TB as it cannot reliably differentiate TB from pancreatic cancer, as TB can also show high uptake [14]. (6) ERCP: can evaluate intraductal lesions and place stents for obstruction relief.

Histologic diagnosis is the gold standard for pancreatic TB. Pancreatic tissue

can be obtained via: (1) EUS-guided fine-needle aspiration (EUS-FNA) and EUS-guided fine-needle biopsy (EUS-FNB) are mature, widely used diagnostic techniques playing important roles in pancreatic TB diagnosis with good safety, minimal trauma, few complications, allowing repeated multi-site sampling with pathology diagnostic rates exceeding 70% [15]. Recent EUS-FNA/FNB developments include needle types, sizes, and sampling techniques such as Fork-tip and Franseen needles that obtain larger tissue samples with preserved architecture, reducing needle passes and improving diagnostic rates [16]. (2) CT- or ultrasound-guided percutaneous pancreatic fine-needle biopsy is less precise than EUS-FNA/FNB for small lesions. Ultrasound is radiation-free with real-time needle guidance to avoid vital organs and vessels but can be affected by bowel gas and bone; CT precisely shows lesion location and morphology, improving sampling accuracy but with radiation, lack of real-time vessel avoidance, and more complications [17]. (3) Surgical exploration: considered when other techniques cannot obtain diagnostic tissue and malignancy is highly suspected. Pathology of pancreatic TB may show caseous necrosis, granulomas, positive acid-fast staining, and positive TB-PCR, with granulomas in >50% of cases [18-19] but non-specific; positive acid-fast staining is more diagnostic but rarely reported [10]; TB-PCR positivity is higher (43-80%) [6,19] and diagnostically significant.

Pancreatic TB treatment is identical to pulmonary TB. Studies show satisfactory outcomes with standard 6-12 month anti-tuberculosis therapy [6], often extended in clinical practice to >1 year [11,19]. This patient' s pancreatic head lesion had not significantly shrunk at follow-up, requiring further evaluation after longer therapy. Approximately 17.6% of patients experience adverse anti-tuberculosis drug reactions, most commonly hepatotoxicity [20]. Drug-resistant TB is another major reason for regimen changes, including mono-resistant, poly-resistant, multidrug-resistant, extensively drug-resistant, and rifampin-resistant TB, requiring susceptibility testing and local resistance data to guide regimen selection [21].

7 Expert Commentary

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Pancreatic TB is a rare disease with non-specific clinical symptoms. Diagnosis often relies on imaging, but some cases are difficult to differentiate from pancreatic malignancy radiologically, requiring histopathologic confirmation. This patient presented primarily with a pancreatic mass with concurrent duodenal, peripancreatic lymph node, and abdominal vascular involvement, complicated by ascites, gastrointestinal bleeding, and jaundice. The complex, rapidly progressive condition posed significant diagnostic challenges due to difficulty obtaining pancreatic histologic or cytologic specimens. For such rare diseases with multiple complications and risky biopsy procedures, multidisciplinary comprehensive evaluation is essential for diagnosis and treatment planning. Histologic evidence in this case could not definitively confirm TB; even during empiric

anti-tuberculosis therapy, close follow-up was needed to watch for possible malignancy. The anti-tuberculosis course was not smooth, with multiple adverse reactions: hepatotoxicity (intrahepatic cholestasis), hyperuricemia, and thrombocytopenia. After identifying the causative agents, the regimen was adjusted and anti-tuberculosis therapy restarted. In summary, this pancreatic TB case presented diagnostic and therapeutic difficulties with multiple complications, providing important guidance and reference for clinicians.

Conflict of Interest: None

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