

Pancreatic tuberculosis with acquired immunodeficiency syndrome: A case report and systematic review

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Abstract

Pancreatic tuberculosis (TB) is a relatively rare disease that can mimic carcinoma, lymphoma, cystic neoplasia, retroperitoneal tumors, pancreatitis or pseudocysts. Here, I report the case of a 31-year-old immigrant Burmese woman who exhibited epigastralgia, fever, weight loss and an epigastric mass. The patient was diagnosed with pancreatic TB and acquired immunodeficiency syndrome, and was treated with antituberculous drugs and percutaneous catheter drainage without a laparotomy. The clinical presentation, radiographic investigation and management of pancreatic TB are summarized in this paper to emphasize the importance of considering this rare disease in the differential diagnosis of pancreatic masses concomitant with human immunodeficiency virus infection. I also emphasize the need for both histopathological and microbiological diagnosis *via* fine-needle aspiration.

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Key words: Pancreas; Tuberculosis; Abscess; Antituberculous drugs; Human immunodeficiency virus; Fine-needle aspiration

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INTRODUCTION

Tuberculosis (TB) is an extremely common opportunistic infection in human immunodeficiency virus (HIV)-positive patients and is considered to be an acquired immunodeficiency syndrome (AIDS)-defining illness^[1]. Because of the virulence of TB, its symptoms tend to manifest at an early stage of HIV infection. The most overt feature of TB in HIV-seropositive patients is their substantially greater likelihood of extrapulmonary involvement and dissemination^[2]. Intraabdominal involvement is frequently observed in the abdominal lymph nodes (LNs), the spleen, the peritoneum, the liver, and the gastrointestinal tract^[3]. However, even in HIV-infected patients, TB of the pancreas is relatively rare with an incidence of 0.46% based on ultrasonographic findings^[4], and isolated primary pancreatic TB is particularly rare. Because of its rarity, the natural course of the disease is currently unknown. Several case reports have included a detailed review of this subject; however, we still lack a complete clinical picture of the disease^[5-7]. Here, I report a case of pancreatic TB with AIDS that initially followed a classic pattern, but in which the patient subsequently exhibited an uncommon natural history of disease. I review the current literature regarding HIV-associated pancreatic TB. To the best of my knowledge, the present systematic review includes the largest number of published case reports on this subject, and is the first to describe the natural history of disease in a patient with pancreatic TB and AIDS.

CASE REPORT

In April 2010, a 31-year-old immigrant Burmese woman was admitted at Mae Sot General Hospital for approximately 1 wk due to epigastralgia. Two day before admission, she experienced constant severe abdominal pain and fever. She also reported weight loss of 5 kg over the preceding month. Upon admission, her temperature was 39.8 °C, her pulse was 128 bpm, her respiratory rate was 18 breaths/min, and her blood pressure was 116/81 mmHg. Her weight and body mass index were 39 kg and 15.82 kg/m², respectively. She presented with oral thrush on her tongue and a pruritic papular eruption on her extremities and trunk. Her abdomen was bulging and contained a well-defined, round, smooth-surface, tender mass measuring 9 cm × 9 cm in the epigastrium. No hepatosplenomegaly or significant lymphadenopathy was observed.

A serological test for HIV was positive, and the patient's lymphocyte subset profile showed a CD4-positive cell count of 120 cells/mm³ (range, 410–1 264/mm³). A sputum examination for acid-fast bacilli (AFB) was negative, and a chest radiograph was normal. An ultrasound (US) of the upper abdomen revealed a lobulated, heterogeneous, hypoechoic cystic mass with septation and an irregular wall at the head of the pancreas (5.6 cm × 5.3 cm × 5.7 cm in size) (Figure 1). Multiple small LNs that ranged from 1.0 cm to 1.9 cm in diameter were observed in the paraaortic, peripancreatic, and porta hepatis regions.

US-guided fine-needle aspiration (FNA) of the mass was performed, and 50 mL of purulent, turbid fluid was harvested. The level of amylase in the cyst fluid was 33 801 U/L. An examination of a specimen using the Ziehl-Neelsen stain revealed AFB in the proteinaceous fluid and abundant acute inflammatory cells. An analysis of the cultures identified pure growth of *Mycobacterium tuberculosis* that was susceptible to isoniazid, rifampicin, ethambutol, and streptomycin. Consistent with the patient's clinical features, she was diagnosed with AIDS with a tuberculous pancreatic abscess. Note that a pancreatic pseudocyst typically contains an amylase-rich fluid, and when pus is present instead of sterile pancreatic juice, an infected pancreatic pseudocyst is referred to as a pancreatic abscess. Antituberculous drugs (ATDs) including isoniazid, rifampicin, pyrazinamide, and ethambutol were administered on the 6th d of admission. The treatment plan included quadruple ATDs for 2 mo followed by isoniazid and rifampicin for the next 7 mo. The patient was discharged after 12 d of hospitalization in an improved and stable condition, but she was still febrile at that time. Antiretroviral treatment was not planned due to a lack of monetary support.

The patient was readmitted 4 mo later due to epigastric fullness and shortness of breath. The epigastric mass was found to be enlarged. A physical examination revealed that the epigastric mass measured 15 cm × 15 cm in size. On admission, the patient had a temperature of 38.9 °C, a pulse of 100 bpm, and a respiratory rate of 24 breaths/min; her blood pressure was 123/87 mmHg.

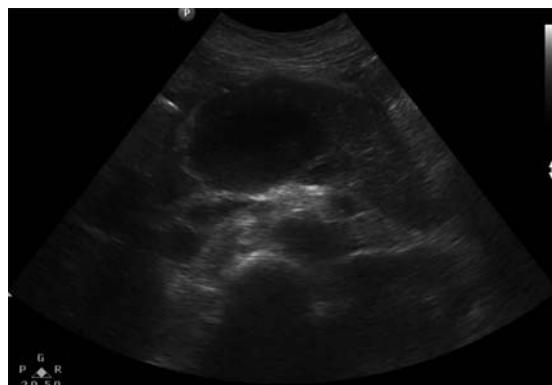


Figure 1 Ultrasound demonstrated a well-defined, lobulated cystic lesion with fluid debris at the pancreatic head. The size of the lesion is approximately 5.6 cm × 5.3 cm × 5.7 cm.

The patient's body weight was 39 kg. The patient's septic workups were unremarkable. An abdominal US revealed that the pancreatic cystic lesion had increased in size (11.8 cm × 8.7 cm × 11.6 cm) and contained a clear fluid (Figure 2A). Encasement of the celiac trunk was noted. Main portal vein compression and varices in the porta hepatis were observed (Figure 2B). A clear, serosanguinous fluid with an intracystic amylase level of 55 708 U/L was obtained from the pancreatic pseudocyst. Aerobic and mycobacterial cultures were negative. Although the patient's abdominal symptoms had improved, she still experienced an intermittent fever, cachexia, malnutrition (with a body weight of 34 kg and a serum albumin level of 21.0 g/L), and weakness within the month following admission. She continuously complied with the ATD regimen for 5 mo and 1 wk prior to discharge. Unfortunately, the patient was lost to follow-up after discharge and died 3 wk later (i.e., 6 mo after being diagnosed with pancreatic TB) in Myanmar from a suspected AIDS-related complication.

DISCUSSION

The PubMed, EMBASE databases, and Google were searched in July 2011 using the following terms in various combinations: tuberculosis, tuberculous, tubercular, mycobacterial infection and pancreas, pancreatitis, pancreatic, peripancreatic and HIV, AIDS, immunocompromised. The reference lists of the retrieved articles or of articles dealing with literature review were searched for additional studies. Only HIV-positive patients with informative clinical characteristics were reviewed.

A total of 43 cases of HIV-positive patients with pancreatic TB (including the present case) were reviewed^[2,5-38]. Of these cases, 40 were primarily analyzed^[2,5-23,25-38]. Table 1 illustrates the clinical characteristics of the pancreatic TB patients with AIDS. Most of the patients appeared to be affected between the age of 30 and 40 (this was the case in 53.7% of cases); the median age was 33 years with a range of 19–61 years, *n* = 41, and most of the patients (75.6%) were ≤ 43 years old. Five of the 40 patients (12.5%) had

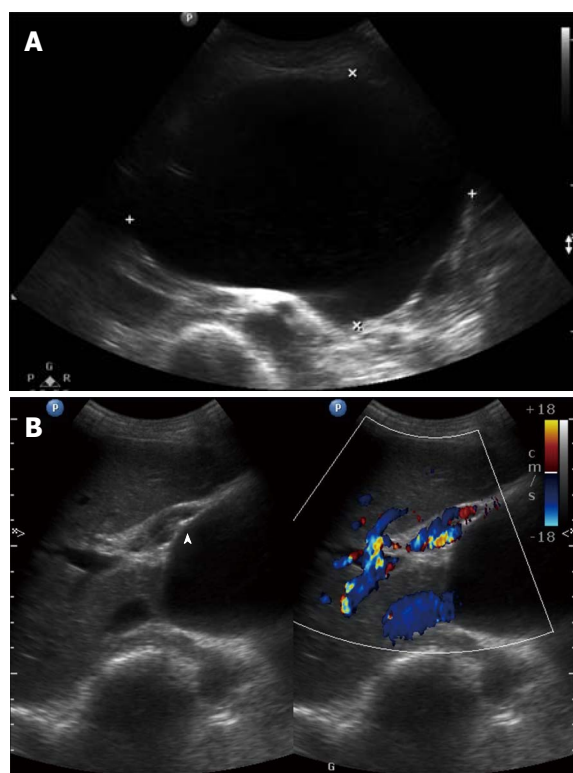


Figure 2 An abdominal ultrasound was performed 4 mo after discharge and revealed an increase in the size of the pancreatic cystic lesion (to 11.8 cm × 8.7 cm × 11.6 cm) with a clear fluid content (A). Portal vein compression (arrowhead) and varices in the porta hepatis were observed (B).

Table 1 Clinical characteristics, diagnostic studies, investigations yielding definitive diagnosis, preoperative diagnoses and definitive diagnosis of patients with pancreatic tuberculosis and acquired immunodeficiency syndrome

Clinical characteristics	n (%)
Gender ^[2,5,20,22-28,30-32,34-38]	
Male	28/39 (71.8)
Female	11/39 (28.2)
Known HIV-infected cases ^[2,5-23,25-38]	11/38 (28.9)
Positive tuberculin skin tests ^[2,5,6,9,12,13,16,29-31,33,34,38]	7/14 (50.0)
Symptoms ^[2,5-23,25-38]	
Abdominal pain	38/40 (95.0)
Fever	36/40 (90.0)
Weight loss	21/40 (52.5)
Anorexia	12/40 (30.0)
Night sweats	10/40 (25.0)
Nausea or vomiting	9/40 (22.5)
Diarrhea	7/40 (17.5)
Dysphagia	2/40 (5.0)
Cough	2/40 (5.0)
Dysuria, polyuria, and confusion	1/40 for each (2.5)
Signs ^[2,5-23,25-38]	
Epigastric mass	8/40 (20.0)
Significant superficial lymphadenopathy	7/40 (17.5)
Hepatomegaly	7/40 (17.5)
Oral thrush	6/40 (15.0)
Jaundice	6/40 (15.0)
Splenomegaly	2/40 (5.0)
Oral hairy leukoplakia, pruritic papular eruption, ascites, and disorientation	1/40 for each (2.5)
Diagnostic studies	n (%)
Evidence of TB on chest	10/31 (32.3)
X-rays ^[2,5,6,8-10,12-15,17-20,23-25,27-32,34-37]	

Abnormal CT scans ^[2,5-10,12-16,19-38]	
Pancreatic/peripancreatic mass with or without diffuse pancreatic enlargement	34/36 (94.4)
Only diffuse pancreatic enlargement	1/36 (2.8)
Small nodular lesions	1/36 (2.8)
Topographic lesions of the pancreatic mass from all imaging studies ^[2,5-10,12,13,15,16,18-25,27,29-38]	
Head of the pancreas	31/36 (86.1)
Body of the pancreas	8/36 (22.2)
Neck of the pancreas	3/36 (8.3)
Tail of the pancreas	2/36 (5.6)
Diagnostic clues of a simultaneous duodenal fistula	
Abnormal upper gastrointestinal study ^[13]	1/1 (100.0)
Abnormal gastroscopy ^[14,17,27,33,38]	2/5 (40.0)
Investigations yielding definitive diagnosis (n = 40) ^[2,5-23,25-38] (%)	
FNA with AFB staining and/or mycobacterial culture ^a	23 (57.5)
Laparotomy	14 (35.0)
Therapeutic diagnosis with evidence of miliary TB ^b	1 (2.5)
Discharge from pancreaticoduodenal fistula	1 (2.5)
Autopsy	1 (2.5)
Preoperative diagnoses (n = 15) ^[2,5,7-13,16-17,19,22,25] (%)	
Pancreatic mass	4 (26.7)
Pancreatic cancer	3 (20.0)
Pancreatic abscess of unknown pathogen	3 (20.0)
Acute pancreatitis	2 (13.3)
Tuberculous pancreatic abscess ^c	2 (13.3)
Lymphoma or retroperitoneal tumor	1 (6.7)
Definitive diagnosis (n = 40) ^[2,5-23,25-38] (%)	
Tuberculous pancreatic abscess without duodenal fistula	26 (65.0)
Tuberculous pancreatic abscess with duodenal fistula	2 (5.0)
Acute tuberculous pancreatitis without duodenal fistula	2 (5.0)
Acute tuberculous pancreatitis with duodenal fistula	1 (2.5)
Focal acute tuberculous pancreatitis with chronic pancreatitis	1 (2.5)
Pancreatic TB	8 (20.0)

TB: Tuberculosis; AIDS: Acquired Immunodeficiency Syndrome; HIV: Human Immunodeficiency Virus; CT: Computed tomography. ^aOne was performed from the left supraclavicular lymph node instead of the pancreatic mass, and one patient underwent both fine needle aspiration and laparotomy due to clinical deterioration. ^bNo histopathological specimens. ^cDiagnosis was based on a computed tomography scan only in one patient.

a confirmed history of TB; in 3 cases, the patient had had a positive purified protein derivative skin test, and there were 2 cases of pulmonary TB. The median CD4 count (n = 21) was 54/mm³ (range, 0-718/mm³); 76.2% and 90.5% of the patients had a CD4 count ≤ 120 or ≤ 190/mm³, respectively. The median duration of abdominal pain (n = 31) before presentation was 30 d (range: 3 d-1 year).

The results of the diagnostic studies conducted in cases of pancreatic TB with AIDS are summarized in Table 1. The types of abdominal imaging studies (n = 43) used included computed tomography (CT) scanning (n = 36), US (n = 27), endoscopic US (n = 1), and magnetic resonance imaging (n = 1). In at least 20 cases, both US and CT scans were performed. A CT scan usually confirmed or completed the US findings and revealed a pancreatic/peripancreatic mass with or without diffuse pancreatic enlargement; this was true in all but 2 cases.

The masses ranged from cystic or hypodense masses to complex soft tissue masses or mixed solid/cystic masses. The CT data for the remaining cases indicated only diffuse pancreatic enlargement or small nodular lesions (< 1 cm) in the pancreas^[24]. The pancreatic masses were 2-8 cm in diameter. Abdominal lymphadenopathy was detected as follows: peripancreatic LN ($n = 9$), retroperitoneal LN ($n = 7$), paraaortic/aortocaval LN ($n = 4$), porta hepatic/hepatic hilum LN ($n = 4$), mesenteric LN ($n = 2$), periportal LN ($n = 2$), unspecified abdominal LN ($n = 2$), celiac LN ($n = 1$), and splenic hilum LN ($n = 1$). Hepatomegaly and splenomegaly were noted in 10 and 9 patients, respectively. Concomitant hepatic and splenic granulomas and concomitant splenic and kidney granulomas were each observed in one patient. Other associated conditions included ascites ($n = 3$), extrahepatic bile duct obstruction and dilatation ($n = 2$), pancreatic duct dilatation ($n = 1$) or compression ($n = 1$), and ileocecal thickening ($n = 1$).

The diagnoses for the pancreatic TB cases are summarized in Table 1. FNA was attempted in 29 cases, but was successful in only 25 (86.2%), 2 of which did not include a documented AFB smear or mycobacterial culture. Only one specimen was successfully obtained using endoscopic US-guided aspiration^[38], whereas the other specimens were obtained percutaneously^[6,10,15,18,21,25-32,34-37]. In the cases in which AFB smears and/or mycobacterial cultures of the FNA specimens were conducted, 20 of 20 (100.0%) and 18 of 19 (94.7%), respectively, were positive. In the sole culture-negative FNA sample, *Mycobacterium tuberculosis* (*M. tuberculosis*) DNA was identified *via* the polymerase chain reaction (PCR) method. ATD susceptibility was studied in 9 samples, and only one was resistant to streptomycin; the others were sensitive to the prescribed first-line ATDs. PCR was performed in 4 cases, and all 4 were positive for *M. tuberculosis* DNA. The probability of positive results from at least one mycobacterial culture or AFB smear for body fluid (excluding the pancreatic mass) was 32.5% (13/40). In these cases, mycobacterial cultures of sputum ($n = 8$), blood ($n = 3$), bronchoalveolar lavage ($n = 2$), urine, ascites fluid, pleural effusion, stool, or preauricular LN ($n = 1$ for each) were taken, as were AFB smears of urine, bone marrow, or supraclavicular LN ($n = 1$ for each). Active pulmonary TB was documented in 9 cases.

The preoperative diagnoses made in all 15 cases in which laparotomies were conducted are summarized in Table 1. A provisional diagnosis of acute pancreatitis with a pseudocyst was made in one autopsy case^[14]. Pancreatic TB was suspected in each of the cases in which FNA was performed; an AFB smear and/or mycobacterial culture was performed in each of these cases. The definitive diagnoses of pancreatic TB are listed in Table 1.

A laparotomy and/or FNA was performed in every case except for one that was diagnosed after autopsy and another in which a presumptive diagnosis was made based on an imaging study and a positive mycobacterial sputum culture^[23]. There were 13 cases in which an exploratory

laparotomy for a biopsy and/or open drainage was considered due to an uncertain diagnosis ($n = 11$) or a deteriorating tuberculous pancreatic abscess ($n = 2$). A distal pancreatectomy/ splenectomy was performed in one case for an unknown reason. In a second case, a Whipple's operation was performed to treat suspected pancreatic cancer.

Thirty-four patients received ATD treatment. The most commonly used treatment was a quadruple ATD regimen that consisted of isoniazid, rifampicin, pyrazinamide, and ethambutol ($n = 15$). The initial response to the ATDs usually occurred rapidly, manifesting between 72 h and 2 wk later; however, fever and cachexia occasionally persisted for years. The duration of ATD treatment ranged from 3 wk to 2 years. Of the 37 documented patients, 33 (89.2%) survived and were dischargeable after their admission. The remaining 4 patients deteriorated and died during their initial hospitalization (yielding a 10.8% in-hospital mortality rate) due to upper gastrointestinal bleeding^[12], severe sepsis^[14], viral meningoencephalitis^[30], or an unreported cause ($n = 1$ for each)^[31]. Twenty-four patients were followed from 3 wk to 32 mo (median, 9.50 mo), and 9 died during the follow-up period from AIDS-related causes, including full-blown AIDS ($n = 5$)^[25,26], disseminated cryptococcosis^[9], non-Hodgkin's lymphoma^[2], pyogenic chest infection^[18], and *Pneumocystis jirovecii* pneumonia ($n = 1$ for each)^[7]. The probability of survival after discharge was determined *via* Kaplan-Meier analysis. A 35.9% long-term survival rate emerged with a median survival period of 18 mo. No recurrence of TB was found in the pancreas or in other organs. In 15 cases, follow-up abdominal imaging data were available. In all but one of these cases (the present case), the size of the pancreatic mass decreased. Antiretroviral drugs were prescribed in only 6 cases; most of the reports did not mention any antiretroviral treatment.

Pancreatic TB is extremely rare, even in countries in which TB is highly prevalent. Pancreatic TB most often occurs as a complication of miliary TB^[9]. The low frequency of pancreatic TB may be partly due to the biological resistance of the pancreas to tubercular infection. Pancreatic enzymes, including lipases and deoxyribonucleases, have antimycobacterial effects^[28]. However, the incidence of pancreatic TB has recently increased. In India, Bhansali did not discover a single case of pancreatic TB in a review of 300 cases of abdominal TB in 1977^[39]; however, a recent study of collective data from 1999-2004 from the same endemic area detected pancreatic TB in 8.3% of the 384 patients who were diagnosed with abdominal TB^[40]. Globalization, the HIV pandemic, and the worldwide resurrection of *M. tuberculosis* are all responsible for this increasing incidence^[41]. Pancreatic TB typically presents in the following patient types: in patients who reside in endemic tuberculous zones, sporadically in no-risk healthy patients, and in patients who are immuno-compromised^[26]. In AIDS cases, tuberculous pancreatic abscesses are most common, accounting for 70.0% of cases. In addition,

71.1% of cases include no previous serological evidence of HIV infection, and 76.2% of patients are severely immunocompromised hosts with a CD4 cell count of $\leq 190/\text{mm}^3$. If the diagnosis is delayed, pancreatic TB can be fatal; the disease has a 10.8% mortality rate (which is comparable to the mortality rate of 9.1% in immunocompetent patients)^[42]. However, pancreatic TB responds well to standard ATDs.

Diagnosing pancreatic TB is challenging. From 1989-1998, 13 of 21 cases were diagnosed postoperatively or postmortem, although this was true for only 2 of 19 cases in subsequent years. This trend indicates increasing surgeon awareness of cases in HIV-infected patients. Pancreatic TB can be classified radiologically as follows: the most common form is mass-forming (with or without diffuse pancreatic enlargement) and accounts for 94.4% of cases, but there is also a diffuse form and a small, nodular form^[24]. The masses can be radiographically similar to pancreatic tumors, abscesses, lymphomas, or pseudocysts. US scans of the abdomen are simple, non-invasive, cost-effective, and readily available; thus, they are usually used as an initial diagnostic tool and exhibit excellent sensitivity. Furthermore, US scans can reveal focal hypoechoic lesions^[12,13,18,20,21,32,34,35,38] and heterogeneously hypo-isoechoic lesions^[5,12,14,18] (primarily in the head of the pancreas), diffuse enlargement of the pancreas^[14,20,23] and enlarged peripancreatic^[12,21,25] and other abdominal LNs^[2,11,18,20,21,31]. Based on the 90.6% sensitivity of association (half of all pancreatic TB patients are HIV-positive), Nagar *et al*^[40] recommended that TB of the pancreas be a differential diagnosis for pancreatic masses associated with peripancreatic lymphadenopathy. Occasionally, biliary dilatation results from the obstruction of the common bile duct^[18,35], pancreatic duct dilatation^[12], air bubble(s) within the mass^[12], encasement of the celiac artery, or compression of the portal vein with collaterals (as in the present case). Tubercular etiology should be suspected in the case of certain ancillary findings, including characteristic lesions in other solid viscera such as the liver, kidney, or spleen; hepatomegaly^[12,31], splenomegaly^[12,31], ascites^[12,20]; peritoneal nodularity; mural thickening in the ileocecal area^[20]; pulmonary TB^[2,11,12,14,18,19,23,28,32], or pleural effusion^[14]. Because of its high sensitivity, a CT scan should be used to rule out associated pathology and to plan for further disease management. However, radiographic signs of pancreatic TB are neither specific nor pathognomonic, and most radiographic findings can be observed in pancreatitis of any cause or pancreatic carcinoma^[20].

A definitive diagnosis is usually based on a histopathological or microbiological examination of a specimen that is obtained from the pancreas or based on peripancreatic LNs exhibiting chronic granulomatous inflammation with caseous necrosis and multinucleated giant cells. In addition, an AFB smear using Ziehl-Neelsen or auramine staining can also be employed for this purpose. Nevertheless, in pancreatic TB cases with advanced HIV-associated immunosuppression and

AIDS, there is a striking paucity of granuloma formation with little cellular recruitment, and the AFB findings are copious^[43]. In non-HIV-infected patients, the success rate of FNA in diagnosing pancreatic TB is 50.0% for specimens that are obtained percutaneously^[42]. This rate is much lower than the 85.7% rate in HIV-infected cases in the present study, which reflects the operator-dependent nature of the procedure. Therefore, it is reasonable to attempt a second percutaneous image-guided FNA by a more experienced operator or an endoscopic US-guided FNA if the first attempt fails. The extremely high identification rate (100.0%) of acid-fast smears of FNA specimens from HIV-positive individuals, compared with the 23%-38% detection rate associated with mixed specimens from HIV-negative individuals^[28,44], indicates the much higher pathogen burden and lower immunological response in these immunocompromised hosts. As a result, and given the simplicity and availability of the test and the rapid results that it produces, FNA with an acid-fast smear should be a required test for pancreatic TB with immunodeficiency. A PCR assay, when used to detect mycobacterial DNA, yields highly specific same-day results. Although its sensitivity to TB in FNA specimens has not yet been determined, the PCR assay is increasingly used as an adjunct to special staining techniques and mycobacterial cultures. It may yield positive results even when specimen cultures are negative^[31,32].

In cases of AIDS with suspected pancreatic TB, mycobacterial smears and cultures from the sputum, bronchoalveolar lavage, blood, urine, stool, bone marrow, superficial LNs, ascites, or pleural effusion specimens should be performed due to the augmented yield of these sites. The more aggressive the investigation, the more likely it is that a primary infection will be discovered. In a study of abdominal TB in HIV infection, all of the cases exhibited at least one extraabdominal TB site (with 3.9 sample sites/case). The incidence of positive mycobacterial cultures from various sites was as follows; extraabdominal LNs (87%), sputum (82%), blood (74%), stool (63%), ascites (67%), bone marrow (56%), and urine (35%). Disseminated TB was present in 93% of cases^[3]. Therefore, the total number of case reports of primary or isolated pancreatic TB may be overestimated, and it may thus be exceedingly rare.

In most cases of pancreatic TB, medication is the preferred treatment; surgery and the drainage of fluid are not preferred. A standard multiple ATD regimen with directly observed therapy for 6-12 mo is usually effective^[5]. In the present case, the fluid was clear and sterile even though the pancreatic mass was subsequently further enlarged. However, if the tuberculous pancreatic mass is enlarged or causes symptoms even after the ATDs have been employed for a reasonable period, minimally invasive procedures should be considered. These might include percutaneous catheter drainage (preferably in patients with no pancreatic duct-pseudocyst communications and pancreatic duct strictures, those with immature

or infected pseudocysts, those at high surgical risk, or those who exhibit malnourishment^[45] or endoscopic internal drainage. Although the prognosis for this disease is good in immunocompetent patients (only 1 out of 58 cases that were reported in the Chinese-language literature resulted in death^[41]), the prognosis is grave in AIDS patients due to the underlying disease^[25], particularly in settings where antiretroviral therapy is unavailable^[46].

Pancreatic TB is extremely rare, has various clinical presentations, and tends to masquerade as a pancreatic malignancy, cystic tumor, or pseudocyst. In HIV-infected patients, a differential diagnosis of a pancreatic mass that is combined with a histopathological and microbiological diagnosis *via* FNA can often prevent unnecessary surgery.

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