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Case Report

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Pancreatic tuberculosis: a case report

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ABSTRACT

Tuberculosis is a bacterial infection secondary to *Mycobacterium tuberculosis* with highest burden within low socioeconomic communities. Most individuals with tuberculosis infection are immunocompromised due to underlying medical conditions such as human immunodeficiency virus. However, tuberculosis can manifest in immunocompetent individuals although the incidence is significantly less. Pancreatic tuberculosis is exceedingly rare with autopsy series revealing pancreatic tuberculosis <5% of tuberculosis infected individuals. This case report will discuss a 16-year-old female presenting with pancreatic tuberculosis masquerading as possible pancreatic malignancy. Emphasis is placed on the challenging diagnostic nature of pancreatic tuberculosis as it presents with non-specific clinical symptoms which are in keeping with a wide range of differential diagnoses. Once correctly diagnosed pancreatic tuberculosis presents the potential of cure. It is for this reason whilst a rare diagnosis clinicians should be mindful of pancreatic tuberculosis to ensure timely correct treatment is pursued and unnecessary surgical procedures avoided.

Keywords: Mycobacterium, Tuberculosis, Pancreas, Extrapulmonary tuberculosis

INTRODUCTION

Tuberculosis is a bacterial infection secondary to *Mycobacterium tuberculosis* with highest burden within low socioeconomic communities.[1] Most individuals with tuberculosis infection are immunocompromised due to underlying medical conditions such as human immunodeficiency virus. However, tuberculosis can manifest in immunocompetent individuals although the incidence is significantly less. Pancreatic tuberculosis is exceedingly rare and requires antituberculosis medical treatment. Pancreatic tuberculosis is known to mimic the appearance of pancreatic malignancy on medical imaging. Misdiagnosis of pancreatic tuberculosis as pancreatic malignancy has led to patients undergoing unnecessary major surgery for resection. Consequently, whilst rare pancreatic tuberculosis in immunosuppressed individuals or immunocompetent individuals from endemic regions should be considered to facilitate appropriate medical management and avoid unnecessary major surgical intervention.

CASE REPORT

A 16-year-old female presents with an eight-month history of fevers, shortness of breath, fatigue, weight loss, and night sweats. The patient was born in Sri Lanka and relocated to Australia at age 6 years. Initial bloods were unremarkable. The patient underwent an Quantiferon-tb gold test which returned as positive. Computed tomography (CT) chest which captured superior abdomen demonstrated likely pulmonary tuberculosis along with necrotic peripancreatic lymph nodes. A magnetic resonance imaging (MRI) of the abdomen was performed and demonstrated an 18×24 mm heterogeneously enhancing lesion concerning for a malignant lesion.

Endoscopic ultrasound (EUS) was performed demonstrating the head of pancreas lesion of concern. Initial biopsy results returned as non-specific. However, a repeat EUS revealed acid fast bacilli in keeping with pancreatic mycobacterium tuberculosis. Human

immunodeficiency virus (HIV) serology was negative and no underlying immunodeficiency could be identified. An intensive 6-month anti-tuberculosis treatment of isoniazid, rifampicin, and ethambutol was undertaken. The patient soon became asymptomatic. A repeat MRI demonstrated a reduction in size of pancreatic tuberculosis associated mass and peripancreatic necrotic lymph nodes. The patient continues to progress well and be closely monitored in the tuberculosis treatment clinic.

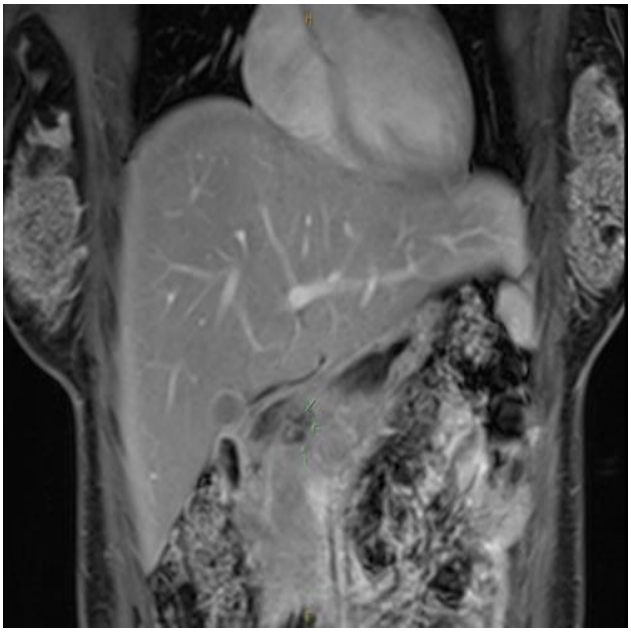

Figure 1: MRI abdomen coronal plane demonstrating head of pancreas lesion.

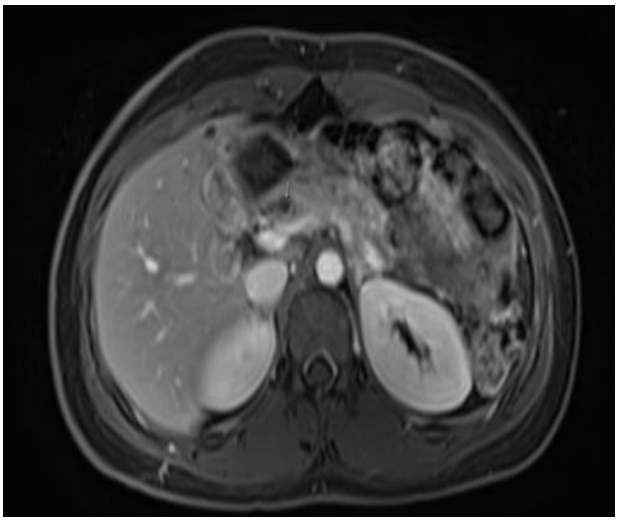

Figure 2: MRI abdomen axial plane demonstrating head of pancreas lesion.

DISCUSSION

Tuberculosis is a bacterial infection secondary to *Mycobacterium tuberculosis*.[1] Tuberculosis is of highest burden within low socioeconomic communities and males within their fifth decade of life.[1] Tuberculosis patient cohort consists mostly of two major groups.[1] The first being individuals from endemic tuberculosis regions and immunocompromised individuals from non-endemic regions.[1] Most individuals with tuberculosis infection are immunocompromised due to underlying medical conditions such as HIV.[1,2] Tuberculosis can manifest in immunocompetent individuals although the incidence is significantly less.[1,2] Tuberculosis most commonly manifests as pulmonary disease but more rarely can establish infection within our bodily organs or systems.[1,2] Abdominal tuberculosis infection is most observed in the ileocaecal region and solid organs such as the spleen, liver, and kidneys.[2] Pancreatic tuberculosis is exceedingly rare with autopsy series revealing pancreatic tuberculosis <5% of tuberculosis infected individuals.[2]

Tuberculosis transmission is routinely via aerosol transmission.[3] The infected individual disperses tuberculosis particles into the air through coughing, singing, or sneezing.[3] The tuberculosis droplet particles may remain suspended in the air for an extended period before being inhaled by a non-infected individual.[3] Following contact with the mycobacterium tuberculosis an individual will either develop a tuberculosis infection or not.[3] Of the individuals who develop a tuberculosis infection there are three potential outcomes.[4,5] The first outcome being that the host self clears the infection, and no residual tuberculosis infection remains.[4,5] The second is the development of latent tuberculosis whereby the disease is contained by the immune system and remain asymptomatic but fail to clear acid-fast bacilli.[4,5] The third outcome is the individual develops symptomatic and progressive tuberculosis infection.[4,5] It should be noted individuals with latent tuberculosis maintain the potential to progress into symptomatic disease.[4,5]

Our case report demonstrated a young female who spent their first six years of life in a tuberculous endemic country. Interestingly, our patient did not demonstrate symptoms until they had lived in Australia, a non-endemic tuberculous country, for nine years. Nor did the patient demonstrate a HIV infection or other underlying causes of immunodeficiency. It is likely the patient had latent tuberculosis which transitioned into a symptomatic and progressive tuberculosis infection. It is not possible to be certain when the patient's pulmonary tuberculosis disseminated to become extrapulmonary pancreatic tuberculosis. However, it is possible dissemination occurred when the patient started becoming symptomatic suggesting progressive tuberculosis infection.

Once tuberculosis infection is established it can remain in the pulmonary system or may spread to other areas of the body. Disseminated tuberculosis is known to spread haematogenously.[2] However, less commonly tuberculosis can spread via lymphatics or directly from bordering sites of infection.[2] The pancreas produces deoxyribonuclease and lipase promoting an anti-mycobacterial effect.[2,6] As such the pancreas presents an unfavourable environment

for tuberculosis infection to establish itself.[2] Subsequently, it is unusual for an immunocompetent individual to develop pancreatic tuberculosis infection.[1,2] It is not unreasonable to assume those with pancreatic tuberculosis are at an increased risk of exocrine and endocrine pancreatic dysfunction.[1] However, given the low incidence of pancreatic tuberculosis there is minimal literature available surrounding the impact of tuberculosis on the pancreatic function.[1]

Pancreatic tuberculosis can be challenging to diagnose as it presents with non-specific clinical symptoms which are in keeping with a wide range of differential diagnoses.[7] Key differential diagnoses include malignancy either primary or metastatic disease, sarcoidosis, or Castleman disease.[2] Common clinical symptoms include fever, night sweats, weight loss, and fatigue.[2] Depending on the extent and anatomical location of the pancreatic tuberculosis individuals may present with jaundice, early satiety, abdominal pain, dyspepsia, gastrointestinal haemorrhage, pancreatitis, or diabetes mellitus.[2] On physical examination evidence of weight loss along with clinical jaundice, epigastric tenderness, epigastric mass, or splenomegaly may be present.[1,2] Unfortunately, the clinical presentation closely resembles presentation of a pancreatic malignancy and can be challenging to delineate.[1,7] Additionally, less than 50% of individuals with pancreatic tuberculosis have no known past medical history of tuberculosis.[2]

Given pancreatic tuberculosis ability to masquerade as other pathologies, such as pancreatic malignancy, microbiological analysis in extrapulmonary tuberculosis remains a crucial step in establishing a formal diagnosis to ensure correct treatment is pursued.[1] Several case series demonstrate initial misdiagnosis of pancreatic tuberculosis as pancreatic malignancy, which was only realised at time of histology following a Whipple procedure or distal pancreatectomy.[2,8,9] When a diagnosis of pancreas tuberculosis is suspected tuberculin skin test (TST) and Quantiferon-tb gold test (QFT) may yield a useful investigation to identify if the individual has a current or latent mycobacterium tuberculosis infection.[1,10] In approximately 70% of patients with pancreatic tuberculosis TST was positive.[1] Given that TST and QFT are unable to distinguish between current or latent tuberculosis infection these investigations are best used as an adjunct to imaging and biopsy.[10]

Medical imaging such as ultrasound (US), CT, and MRI are modalities to further delineate the origin of a pancreatic lesion.[2] Pancreatic tuberculosis on imaging may present as a well-defined hypodense hypovascular mass with irregular boarders.[2] Additionally, there may be central liquefaction with associated lymphadenopathy.[2] These features are non-specific to pancreatic tuberculosis and can represent a vast array of differential diagnoses ranging from inflammatory, infective, to malignant pathology.[2] EUS with fine needle aspiration cytology (FNAC) is the preferred modality of histological confirmation due to its minimally invasive nature.[1,2,11-15] Whilst FNAC only identifies acid-fast bacilli in 20-40% of biopsies, if also sent for culture and polymerase chain reaction (PCR) assay recognition of mycobacterium is tuberculosis is identified in up to 77% of biopsies.[2,16]

If EUS FNAC yields inconclusive results other options to obtain histopathology can be considered. These include CT imaging guided percutaneous biopsy or surgical excisional biopsy.[2,8,9] Given the retroperitoneal location of the pancreas CT imaging guided percutaneous biopsy of a pancreatic lesion carries an elevated risk of damage to surrounding structures.[2] However, may be considered in biopsy of necrotic lymph nodes or other areas of potential tuberculosis disease to establish diagnosis.[2] Unfortunately, other minimally invasive procedures such as endoscopic retrograde cholangiopancreatography (ERCP) with biliary cytology seldom yield a result to confirm pancreatic tuberculous.[2,18] Surgical excisional biopsy of pancreatic lesion or necrotic peripancreatic lymph node also remain options for obtaining tissue for histopathology.[2,8,9] Importantly once diagnosis of tuberculosis is confirmed or highly suspected consideration of possible predisposing immuno-deficiencies should be considered.[1,2] Subsequently, HIV infection should be considered and excluded on serology.[1,2]

Our case report demonstrated an example of the classic non-specific presentation of pancreas tuberculosis. The patient did not have a known history of tuberculosis. Whilst the positive QFT and CT chest demonstrated evidence of likely pulmonary tuberculosis given there was no immunodeficiency to suggest high risk of disseminated tuberculous, pancreatic malignancy remained a diagnosis of suspicion. On imaging it was challenging to delineate between tuberculosis and a pancreatic malignancy, thus an EUS FNAC was required. Interestingly, our patient's FNAC returned as positive for acid-fast bacilli. Although, it is acknowledged that a second EUS FNAC was required due to the initial FNAC returning inconclusive results.

Management for pancreatic tuberculosis is predominately medical in nature and presents the potential of cure.[1,2,8] Both pulmonary and extrapulmonary tuberculosis receive the same treatment.[1,2] Medical therapy with anti-tuberculous is recommended for 6 to 12 months.[2] Commonly the initial 2 to 4 months of anti-tuberculous treatment includes ethambutol, pyrazinamide, isoniazid and rifampicin. Which is then followed by isoniazid and rifampicin for 6 to12 months.[2,18-20] On occasion due to multidrug resistant tuberculous and anti-tuberculous medication side effects, such a hepatoxicity, different anti-tuberculous medications are required.[2,21] Pancreatic tuberculosis maintains a good response rate to anti-tuberculous medical therapy.[3]

Invasive or surgical interventions are not the mainstay of pancreatic tuberculous treatment but rather a tool to achieve diagnosis or symptom control. Invasive

procedures such as ERCP and biliary stent placement may be pursued to address symptoms such as biliary obstruction as an adjunct to anti-tuberculous treatment.[2,17-20] Surgical procedures such as laparoscopy or laparotomy may be pursued to obtain an excisional biopsy to facilitate confirmation of diagnosis if other less invasive methods fail to provide a clear diagnosis.[2,8,9,13] The documented pancreatic tuberculous cases that underwent surgical resection such as Whipple procedures and distal pancreatectomy, were in the setting of unrecognised pancreatic tuberculous with a presumed diagnosis of pancreatic malignancy.[2,8,9]

In our case report the patient responded to anti-tuberculous treatment with resolution of symptoms and a reduction in size of the tuberculosis associated pancreatic mass. Fortunately, the second EUS FNAC yielded a diagnostic result allowing the commencement of appropriate treatment. As such the patient did not require further invasive or surgical procedures for an excisional biopsy. Additionally, a clear diagnosis negated the concern for pancreatic malignancy and requirement for formal surgical resection. Our case report highlights the importance to consider the diagnosis of pancreatic tuberculosis in a space occupying pancreatic lesion. All be the diagnosis of pancreatic tuberculosis rare, if misdiagnosed the patient may undergo unnecessary surgical procedures and delay required treatment.

CONCLUSION

Tuberculosis is a bacterial infection secondary to *Mycobacterium tuberculosis* with highest burden within low socioeconomic communities. Most individuals with tuberculosis infection are immunocompromised due to underlying medical conditions such as HIV. However, tuberculosis can manifest in immunocompetent individuals although the incidence is significantly less. Pancreatic tuberculosis is exceedingly rare with autopsy series revealing pancreatic tuberculosis <5% of tuberculosis infected individuals. The pancreas produces deoxyribonuclease and lipase promoting an anti-mycobacterial effect. As such the pancreas presents an unfavourable environment for tuberculosis infection to establish itself. Pancreatic tuberculosis can be challenging to diagnose as it presents with non-specific clinical symptoms which are in keeping with a wide range of differential diagnoses. Once diagnosed treatment for pancreatic tuberculosis is predominately medical in nature and presents the potential of cure. Given pancreatic tuberculosis tendency to masquerade as other pathologies, such as pancreatic malignancy, microbiological analysis in extrapulmonary tuberculosis remains a crucial step in establishing a formal diagnosis to ensure timely correct treatment is pursued and unnecessary surgical procedures avoided.

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