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Case Report

Exceptional association of hepatic and pancreatic tuberculosis mimicking metastatic pancreatic neoplasia: A case report and review of the literature

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ABSTRACT

Introduction: Despite the high prevalence of tuberculosis in the world and especially in endemic areas such as Morocco, isolated hepatic and pancreatic tuberculosis and pancreatic tuberculosis remain rare and pose a real diagnostic problem.

Case presentation: We report a case illustrating an exceptional association of pancreatic tuberculosis with hepatic tuberculosis in a 44-year-old immunocompetent woman, in whom the presence of a pancreatic mass on imaging suggested a neoplastic origin. The diagnosis was rectified after bacteriological and pathological study of the CT-guided percutaneous biopsy specimens.

Discussion: The symptomatology of pancreatic and hepatic tuberculosis is unspecific and polymorphic and can mimic any intra-abdominal pathology. Abdominal ultrasound is often the first imaging modality used. The diagnosis of certainty is bacteriological.

The treatment of pancreatic and hepatic tuberculosis is identical to the other extrapulmonary tuberculosis. the anti-bacillary drugs are the gold standard. Surgery is reserved for complicated forms.

Conclusion: The diagnosis of hepatic and pancreatic tuberculosis is a challenge for the clinician. The treatment is usually medical and based on antituberculosis treatment; surgery may be necessary in case of complications.

1. Introduction

Despite the high worldwide prevalence of tuberculosis especially in endemic areas like Morocco [1], isolated hepatic and pancreatic tuberculosis are extremely rare and pose a real diagnostic problem [2]. Their association is exceptional. The non-specific clinical presentation and the polymorphism may lead to the suspicion of a neoplastic origin and perform a useless and potentially morbid surgical procedure [3]. Herein, we report a case illustrating an exceptional association of pancreatic and hepatic tuberculosis in a 44-year-old woman, an immunocompetent woman, in whom the presence of a pancreatic lesion on imaging suggested a neoplastic origin. The final diagnosis of tuberculosis was made upon a bacteriological and anatomopathological study of the biopsy samples. This case has been reported following the SCARE criteria [4].

2. Case report

A 44-years-old female patient, with no particular pathological history or tuberculosis infection presented to the ER with a history of atypical epigastralgia that had been evolving for two months with fever and weight loss. Physical examination revealed a slight tenderness in the epigastric region. The standard biological workup including lipase, tumor markers (CEA, CA 19–9), hepatitis (B, C) serologies, HIV viral serologies, and liver workup were all within the normal range.

Abdominal computed tomography (CT) scan showed two lesional located respectively at the upper and lower edges of the body of the pancreas measuring 5 × 3 cm and 4 × 3 cm respectively, with irregular contours, enhanced inhomogeneously associated with multiple hepatic lesions and mesenteric adenomegaly with a necrotic center.

These two pancreatic lesions seemed communicated on the posterior surface of the pancreas with close contact with the splenic vein (Fig. 1).

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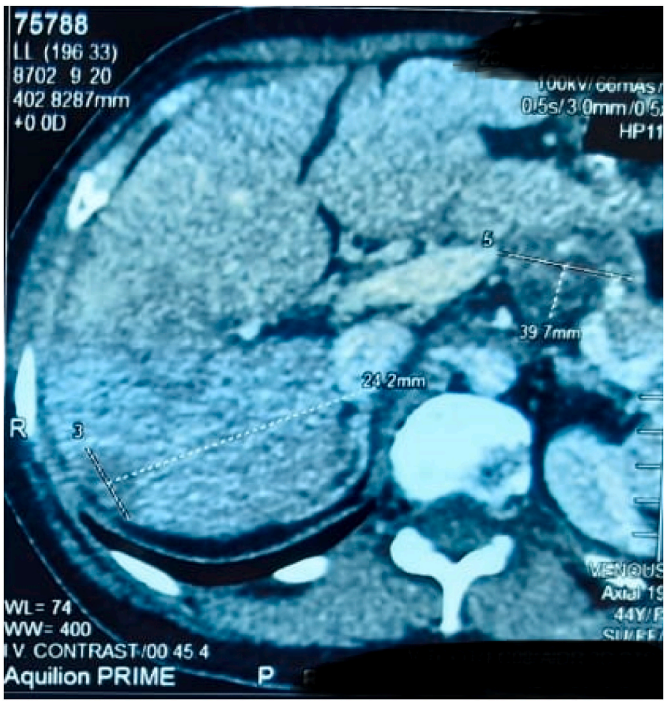

Fig. 1. Abdominal CT showing a mass located in the body of the pancreas with the presence of multiple liver lesions.

Given the suspicion of a neoplastic origin of the lesion, an 18-Fluoro deoxy-D-glucose proton emission tomography (FDG-PET) showed two corporal pancreatic lesions intensely avid for FDG with multiple hepatic secondary lesion and hypermetabolic pathological subdiaphragmatic adenopathies (Fig. 2).

Two CT-guided liver and pancreatic biopsies were performed. The pathological examination revealed an epithelioid with giant cellular granulomatous hepatitis and pancreatitis with suppurative necrosis, the XPERT MTB/RIF test performed on the biopsy fragments was positive with the presence of mycobacteria of the tuberculosis complex without showing a mutation of the rPOB gene coding for resistance to rifampicin, thus confirming the diagnosis of tuberculosis (Figs. 3 and 4).

A thoracic CT was normal. Direct examination for *Mycobacterium tuberculosis* in sputum was negative, a gamma interferon release test was positive. After the Multi-Disciplinary Team meeting, we decided to put the patient on anti bacillary treatment based on Isoniazid 5mg/kg, Rifampicin 10 mg/kg, Ethambutol 20 mg/kg, and Pyrazinamide 25mg/kg for two months, followed by two drugs for the next four months: isoniazid and rifampicin.

The short-term evolution was marked by a favorable clinical improvement (weight gain, appetite recovery, apyrexia) with good tolerance to the treatment. an abdominal CT scan will be performed at the end of the treatment to assess the evolution of the different lesions.

3. Discussion

Tuberculosis remains a major public health problem in the world. In 2020, the number of new cases of tuberculosis was estimated to be 9.9 million, with the highest incidence observed in developing countries [5].

Isolated hepatic tuberculosis is rare [6,7]. The liver is often affected as part of a miliary tuberculous from the lungs via the hepatic artery [8].

Infection may also spread via the portal vein or lymphatics, particularly in patients with concomitant tuberculosis of the gastrointestinal tract [9].

Isolated pancreatic tuberculosis is extremely rare, even in endemic countries with less than 5% of cases, as shown in the autopsy series [10]. The first report of pancreatic tuberculosis was reported by Auerbach in 1944. In his series of 1656 autopsies of tuberculosis patients, only 14 cases had pancreatic involvement with an incidence of 4.7% [10]. This is an exceptional entity, even in the course of miliary tuberculosis. The pancreatic resistance to tuberculosis infection may have two possible explanations. The first may be anatomical, by its retroperitoneal location, the pancreas seems to be protected against direct environmental exposure, and the other explanation can be biochemical, due to the anti-bacillary effect of the secretions and pancreatic enzymes, particularly lipase. The pancreatic tuberculosis infection thus requires massive insemination by the germs, often by contiguity from a peri pancreatic lymph nodes involvement, rarely by contiguity or hematogenous dissemination from an occult site (often pulmonary) or a latent focus reactivated as a result of immunosuppression, in the case of miliary

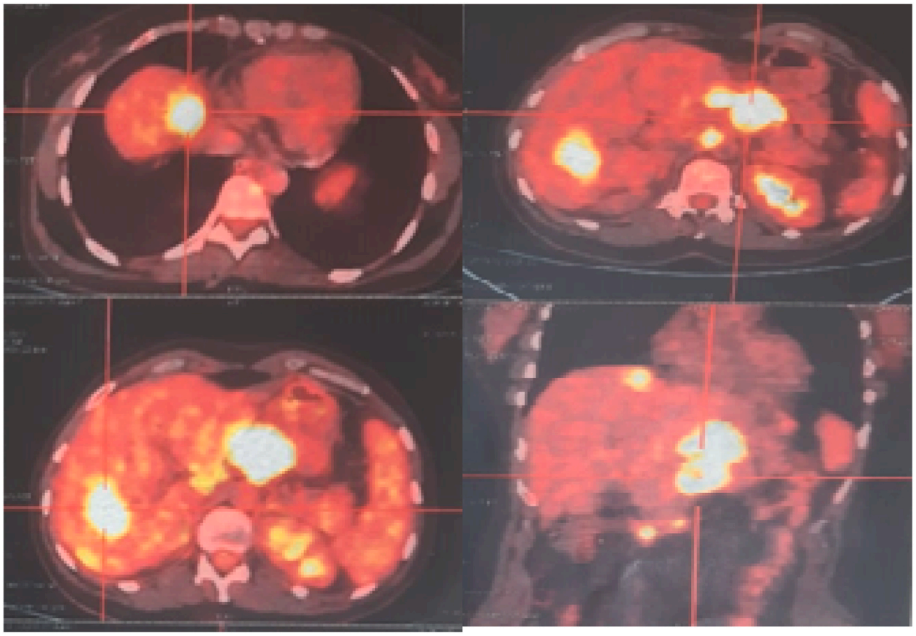

Fig. 2. FDG PET showing showed two corporal pancreatic lesions intensely avid for FDG with multiple hepatic secondary lesion.

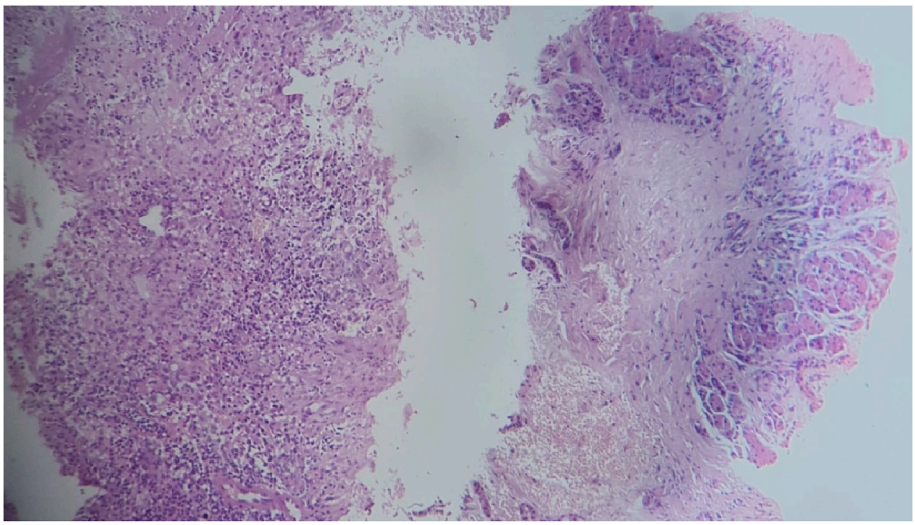

Fig. 3. Microphotograph showing pancreatic parenchyma dissociated by a granulomatous infiltrate composed of epithelial cells and giant cells focally centered by suppurative necrosis.

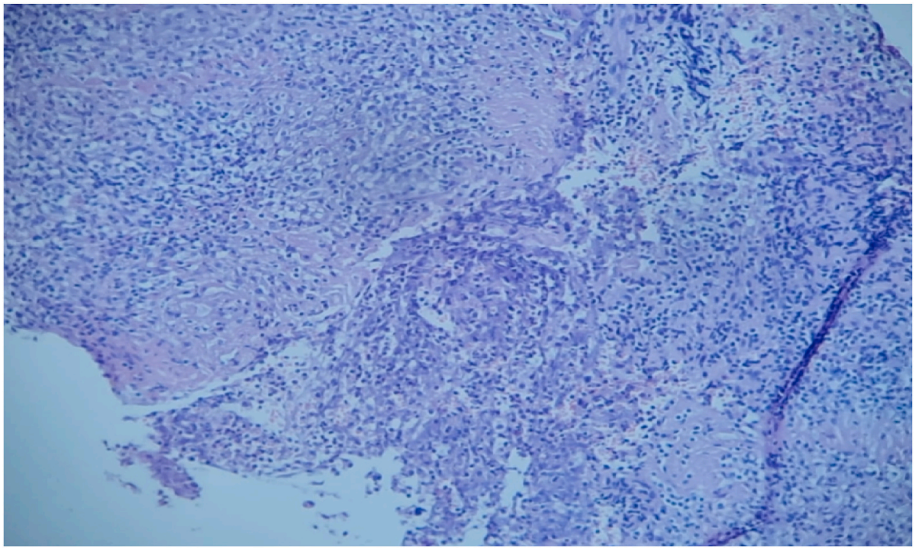

Fig. 4. Microphotograph showing liver parenchyma dissociated by a granulomatous infiltrate composed of epithelial cells and giant cells focally centered by suppurative necrosis.

tuberculous disease with multivisceral involvement [11].

The symptomatology of pancreatic and hepatic tuberculosis is not specific and may mimic any intra-abdominal pathology.

Affected patients may remain asymptomatic. In a study of 42 cases by Kim et al., 19 patients (45.2%) were asymptomatic, while the others had various nonspecific symptoms [12].

Symptomatic patients may show signs related to the pancreatic mass including acute or chronic abdominal pain, abdominal mass, anorexia, weight loss, asthenia, fever, night sweats, or back pain [13]. Nagar et al. in their study of 32 patients reported that the most common symptom was epigastric pain [14]. Xia et al. also reported that the most common symptom was abdominal pain (75%), other symptoms were

Table 1
Assessment of various symptoms of pancreatic tuberculosis in the different studies.

Studies	2003Xia et al.	2009 Nagar et al.	2009 Song et al.	2011 Ibrahim et al.	2012Puri et al.	2013Rao et al.	2014Kim et al.	2015Rana et al.	2019Alsaif et al.
Number of patients	16	32	21	14	19	14	42	16	9
Pain	75%	91%	67%	100%	100%	57%	29%	100%	89%
Fever	50%	81%	14%	100%	63%	100%	7%	0	33%
Night sweats	50%	*	5%	21%	5%	*	*	0	11%
Weight loss	69%	53%	33%	100%	63%	36%	3%	100%	55%
Anorexia	69%	*	23%	*	31%	28%	*	*	11%
Jaundice	31%	9%	5%	*	21%	7%	*	75%	11%

anorexia/weight loss (69%), weakness (64%), fever (50%), and jaundice (31%) [15]. Table 1 summarizes the various symptoms of pancreatic tuberculosis in the different studies reported in the litterature.

Abdominal ultrasound is often the first imaging modality used. It may reveal non-specific signs of pancreatic tuberculosis, such as homogeneous or heterogeneous swelling of the pancreas [16]. Ultrasound can also help to evaluate extra-pancreatic signs such as ascites, liver, spleen lesions, and lymph nodes [17].

Abdominal CT was performed in almost all reported studies but failed to diagnose pancreatic tuberculosis in the majority of cases [18].

The CT features of pancreatic tuberculosis are not specific and include:

- Pancreatic mass:

On CT, a hypodense, hypovascular mass is seen [13], sometimes multicystic [19], single or multiple with coronal contrast. crown [16]. This pancreatic mass may be accompanied by adenopathies, sometimes voluminous hypodense peripancreatic adenopathies, which are very suggestive [19].

These features may resemble those of inflammatory cystic lesions or neoplastic lesions of the pancreas [13].

Although the majority of lesions involve the head and/or body of the pancreas isolated involvement of the tail of the pancreas has been reported less frequently [20].

In our patient, abdominal CT also showed two lesions hypodense in the body of the pancreas with multiple adenopathies peripancreatic adenopathies, partially necrotic.

- Bile ducts and pancreatic duct:

The common bile duct and pancreatic duct are normal in cases of pancreatic tuberculosis, regardless of the location of the lesion. However, in pancreatic adenocarcinomas (located in the center of the head of the pancreas), the pancreatic duct is dilated [21].

- Vascular invasion:

Invasion of the abdominal vessels has often been reported as a point of distinction between pancreatic tuberculosis and malignancy [22]. Nevertheless, vascular involvement cannot be used as a criterion to distinguish pancreatic tuberculosis from malignancy.

- Calcifications:

Calcifications in pancreatic lesions and lymph nodes peripancreatic lymph nodes may suggest pancreatic tuberculosis rather than pancreatic malignancy [23].

Echo-endoscopy seems to be the examination of choice [24]. In recent years, endoscopic diagnosis by fine-needle aspiration has proven to be an excellent tool for the proved to be an excellent tool for the cytological diagnosis of pancreatic tuberculosis with a sensitivity of 80%–95%, which can help to avoid unnecessary surgery [21,25].

FDG- PET scan has also been evaluated in patients with pancreatic tuberculosis, and the results can closely mimic pancreatic cancer [17].

The sensitivity of percutaneous ultrasound- or CT-guided biopsy for pancreatic tuberculosis is 50%–80% [21,25].

Some authors recommend percutaneous biopsy for any cystic mass. Some authors recommend percutaneous biopsy for any cystic mass of the pancreas, while others question its value. Moreover, this examination carries a risk of tumor dissemination [26].

The final diagnosis is bacteriological and is based on the detection of *Mycobacterium tuberculosis* on Ziehl's stain which is positive in 77% of cases [27].

Identification by PCR (DNA polymerase chain reaction) can detect the presence of "Koch's bacillus" with a sensitivity of 60–80% [28].

The diagnosis can be supported by the pathological study performed on the surgical specimen after exeresis surgery [29] or after percutaneous biopsy.

For our patient, the Xpert MTB/RIF test (real-time PCR) performed on the fragments of the liver and pancreatic biopsy allowed the detection of mycobacterial DNA.

The workup for other tuberculosis localizations (clinical examination, chest X-ray, Xpert MTB/RIF in sputum) is often negative [28].

Given its rarity and sometimes misleading presentation, the diagnosis of pancreatic tuberculosis may be overlooked or made late.

The main differential diagnosis remains the pancreatic neoplasia as the radiological findings are not specific even with FDG-PET that was performed in front of the suspicion of metastatic pancreatic neoplasia as presented in our case report.

In the majority of cases, the clinical and radiological findings mimic pancreatic neoplasia [30].

Kim et al. [31] reported that in about 52% of cases, pancreatic tuberculosis was misdiagnosed for pancreatic cancer, lymphoma, or retroperitoneal lymph node metastases. Some of these patients have even undergone unnecessary surgery. Saluja et al. [33] reported that even 2 out of 7 patients with pancreatic tuberculosis underwent palliative radiotherapy or chemotherapy due to a false diagnosis.

The pathological study and the Xpert MTB/RIF test on the biopsy as performed in our case report confirms the diagnosis of tuberculosis and eliminate the diagnosis of pancratic neoplasia.

Pancreatic tuberculosis is an easily treatable and curable disease unlike other causes of pancreatic mass (such as pancreatic adenocarcinoma) which often have a poor prognosis [34,35].

The treatment of pancreatic and hepatic tuberculosis is identical to the other extrapulmonary tuberculosis [16]. It is based on a treatment with a standard two-month quadruple therapy regimen (isoniazid, rifampicin, pyrazinamide, and ethambutol) during the intensive phase, followed by a continuation phase (isoniazid and rifampin) for four months.

The usual dosages for rifampin, isoniazid, pyrazinamide and ethambutol are ethambutol are 10 mg (8–12 mg/kg/day), 5 mg (4–6 mg/kg/day), 25 mg (20–30 mg/kg/day) and 15 mg (15–20 mg/kg/day) respectively [17]. The presence of obstructive jaundice does not alter the treatment regimen and dosages, but it is prudent to closely monitor these patients for drug-induced liver injury [17].

Surgery is reserved for the treatment of complications (duodenal fistula, abscess cold abscess, extensive duodenal or biliary stenosis) [28].

The choice of the appropriate modality to follow patients with tuberculosis is not clear [17]. No definitive guidelines are available in the literature regarding the role of repeat imaging. Singh et al. [36] have suggested radiological follow-up radiology to document regression of the mass but the time intervals are not exactly specified. They suggested that if no radiological improvement occurs even after anti-tuberculosis treatment, the mass should be resected for pathological confirmation [37]. Xia et al. [15] performed a follow-up in 11 of 16 patients and found a complete resolution of the lesions, although the time was variable between 78 and 186 days with a mean of 132 days. Therefore, we conclude that repeated imaging at the end of treatment will help us to monitor and understand the natural disease process [37].

Without treatment, tuberculosis of the pancreas may progress to complications, which may be inaugural to the disease.

Pancreatic tuberculosis may lead to acute or chronic pancreatitis, diabetes, intra-abdominal hemorrhage, or be responsible for biliary obstruction [17].

The evolution under anti-bacillary treatment is usually favorable, with regression of clinical signs and rapid regression of the lesions on ultrasound and CT scan during the first six months [38,39].

The prognosis of pancreatic and hepatic tuberculosis is good in the majority of cases (90%) if the diagnosis is made early. Recurrences are rare and mortality is around 7% in immunocompetent patients [28].

Pancreatic and hepatic tuberculosis can be fatal in case of delayed diagnosis. The mortality rate is estimated at 10.8% [37].

4. Conclusion

The diagnosis of hepatic and pancreatic tuberculosis represents a challenge for the clinician due to the heterogeneous clinical presentation and the non-specificity radiological findings that may mimic a neoplastic pathology. The diagnosis of certainty is based on a bacteriological and pathological study of the samples obtained by a percutaneous biopsy to avoid a surgical procedure with a high morbimortality.

The treatment is in most cases medical and based on the treatment of antituberculosis treatment; surgery may be necessary in case of complications.

Ethical approval

No ethical approval necessary.

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Author contribution

Sara Gartini: Writing, review and editing of the manuscript. **Abdelbassir Ramdani, Meriem Rhazari, Afaf Thouil, Hatim Kouismi, Mohammed Aharmim:** Contributed for diagnose and treatment of the patient. **Jamal Eddine Bourkadi: S**upervised the writing of manuscript.

Registration of research studies

Our paper is a case report; no registration was done for it.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Guarantor

Gartini Sara.

Declaration of competing interest

The authors declared no potential conflicts of interests with respect to research, authorship and/or publication of the article.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.amsu.2022.103717.

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