Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Gastroenterology

Pancreatic Pseudotumor Tuberculosis: A Case Report

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DOI: 10.36347/sjmcr.2022.v10i05.014 | **Received:** 03.04.2022 | **Accepted:** 14.05.2022 | **Published:** 18.05.2022

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Abstract **Case Report**

Tuberculosis is a communicable disease caused by the bacillus Mycobacterium tuberculosis. Approximately one quarter of the world's population is infected with M. tuberculosis and therefore at risk of developing tuberculosis [1]. The abdominal form is the third most common extra-pulmonary location and accounts for 3% of the different topographical forms in our country [2]. The pancreatic and peripancreatic location is very far from the frequency of peritoneal and intestinal lesions. It often takes on a pseudotumoral appearance, thus posing diagnostic problems that must be resolved urgently.

Keywords: Pseudotumor, pseudotumoral, communicable diagnostic problems.

INTRODUCTION

Tuberculosis is considered a major global disease. The digestive tract is the fourth most common site after the pulmonary, pleural and lymph node forms, and is dominated by the ileocaecal and peritoneal forms. Pancreatic involvement is nevertheless exceptional, and its diagnosis, in the absence of associated pulmonary involvement, is difficult due to the diversity and non-specificity of the clinical and radiological signs, thus simulating several pathologies, in this case neoplasia [3].

We report a case of pancreatic tuberculosis in its pseudo-tumoural form collected in the hepato-gastroenterology department "II" of the Mohamed V military hospital in Rabat. In this study, we review the diagnostic difficulties of pancreatic tuberculosis in its tumour-like form and the contribution of endoscopic ultrasound to the diagnosis.

CASE REPORT

A 24 year old woman with no particular pathological history and no notion of tuberculosis infection, admitted for exploration of recurrent epigastralgia, with posterior irradiation, of moderate intensity, without any other associated sign, evolving for 3 years in a context of conservation of the general state.

Clinical examination showed epigastric tenderness without hepatosplenomegaly or palpable mass. The lymph nodes were free and the rest of the clinical examination was unremarkable. The biological work-up showed anaemia with a haemoglobin level of 9.2 g/dl and hyperleukocytosis of 11710, mild hepatic cytolysis without associated cholestasis (transaminases 140 IU/l), and lipase was normal. Abdominopelvic CT showed a hypodense lesion over the isthmus and tail of the pancreas measuring 65*38 mm associated with partially necrotic peripancreatic, coeliomenic and retroperitoneal adenopathies, the largest of which measured 26 mm. and MRI scan showed a liquid, necrotic, multipartitioned lesion, with a central band, located at the posterior part of the isthmus and tail of the pancreas and measuring 63*33mm which extended to the back cavity of the epiplonia, with several partially necrotic peripancreatic and retroperitoneal adenopathies.

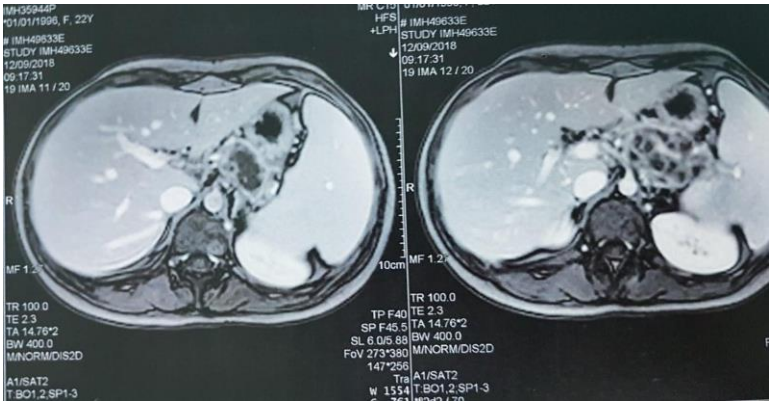

Fig-1: MRI scans showing a multicompartment fluid lesion in the pancreas

Further exploration by transbulbar and transgastric echo-endoscopy showed a heterogeneous oval hypoechogenic lesion of mixed content and cystic and tissue contours measuring 33 mm in long axis at the pancreatic corporocaudal junction, with a doubt about the presence of perilesional adenopathy. The rest of the pancreatic parenchyma was homogeneous and the main bile duct and the main pancreatic duct were of normal calibre.

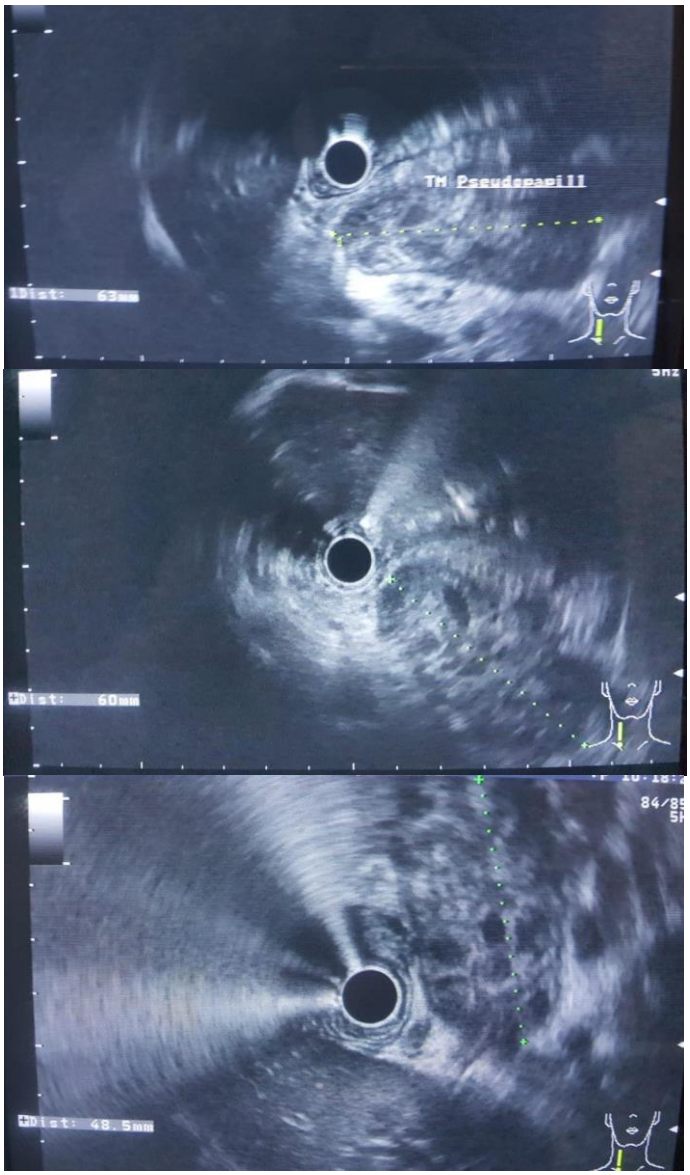

Fig-2: Echo-endoscopic images showing the cystic lesion in the pancreas

A pseudo papillary solid tumour of the pancreas was strongly suspected. The indication of a caudal splenopancreatectomy was retained and the patient benefited from a laparotomy, which in addition to the isthmo-caudal cystic lesion and peri-lesional adenopathies, revealed the presence of whitish pinhead granulations, disseminated over the entire pancreatic parenchyma, more suggestive of a pancreatic tuberculosis.

An extemporaneous anatomopathological examination of the peripancreatic adenopathy confirmed the diagnosis of tuberculosis, with evidence of epithelioid and gigantocellular granulomas centred by caseous necrosis.

The diagnosis of pancreatic tuberculosis was accepted, and an antibacillary chemotherapy was started according to the following scheme: 2 months of quadritherapy (Rifampicin, Isoniazid, Pyrazinamide and Ethambutol followed by the combination of Rifampicin and Isoniazid for 4 months.

The clinical course was favourable, with disappearance of abdominal pain and considerable regression of the cystic lesion on CT scan at 3 and 6 months.

DISCUSSION

The rarity of pancreatic and peri-pancreatic lymph node tuberculosis is well known and argued´ by the work of Paraf who had recorded´ only 11 cases out of 562 autopsies of patients with´ this´ of´ tuberculosis [5]. This localization appears to be peculiar to adults [4].

In the majority´ of cases, pancreatic and peripancreatic tuberculosis evolves as part of a multidiscal involvement, but there are primary and isolated forms in 25% of cases [1, 6, 7].

In the majority of cases, the clinical and radiological findings resemble those of pancreatic malignancy [6]. The finding of a pancreatic mass, the presence of peripancreatic adenopathy and calcifications, and vascular invasion are common features of tuberculosis and pancreatic malignancy. Currently, no study has established a reliable imaging technique to accurately distinguish tuberculosis from other pancreatic pathologies, and a diagnosis of certainty can only be made based on histopathological or microbiological evidence of the disease [7]. Therefore, pancreatic tuberculosis should be considered in patients with a pancreatic mass, especially if the patient resides in an endemic country. This consideration is also important for patients who are immunocompromised, have a history of tuberculosis or have other extra- pancreatic tuberculosis involvement such as the lungs [8, 9].

Other differential diagnoses of pancreatic tuberculosis are pancreatitis. Wegner in the face of granulomatous inflammation and the absence of caseous necrosis [6].

In our case, the young age of the patient, the female sex and the imaging data, in particular echo-endoscopy, make it possible to discuss certain anatomical-clinical forms of pancreatic tumours and in particular the pseudopapillary and solid tumour of the pancreas or Frantz tumour, which has a mixed composition with solid and cystic zones, a low-grade malignant potential and a favourable prognosis [10].

It is a rare tumour that accounts for less than 2% of exocrine pancreatic tumours and less than 5% of cystic pancreatic tumours. It usually affects young women in the second or third decade of life with a mean age of 28 years and a sex ratio of 10:1 [11].

Clinically and biologically, the signs of PPSD are not specific. The circumstances of discovery are multiple: it may be a palpable abdominal mass, an incidental finding on an imaging examination or vague abdominal pain. PPSWT rarely causes signs of digestive or biliary compression. It is rarely discovered in the context of a complication such as intratumoral haemorrhage or intraperitoneal rupture [11].

Morphologically, ultrasound, CT and MRI can describe cystic, mixed and solid forms. They usually show a well-limited, poorly vascularised mass that develops preferentially in the corporal-caudal region of the pancreas (64% of cases).

On echo-endoscopy, the lesion is echogenic, heterogeneous, with a hypoechoic peripheral halo. This examination can show the backflow of the Wirsung canal. However, the value of echo-endoscopy is diminished by the generally large size of the tumour [10].

The imaging data of the tumour, compared with the epidemiological data, are sufficient to evoke the diagnosis which will be confirmed by the anatomopathological analysis of the surgical specimen.

The only curative treatment for PPSD is radical surgical removal. It consists of a left pancreatectomy with, if possible, conservation of the spleen, cephalic duodenopancreatectomy (CPD), partial pancreatectomy, or even total pancreatectomy depending on the location of the tumour and its relationship with neighbouring organs [10, 11].

In our case, the diagnosis of pancreatic tuberculosis was confirmed on histology by the demonstration of epithelioid and gigantocellular granulomas centred by caseous necrosis during the

extemporaneous examination of a peripancreatic adenopathy.

Antibacillary treatment involves a triple or quadruple combination for a period of 6 to 12 months depending on the protocol (6 months in our case [2]).

Progression under antibacillary treatment is usually dramatic, with amendment of general signs where they exist and rapid improvement of ultrasound and CT images.

CONCLUSION

Because of its rarity and sometimes misleading presentation, the diagnosis of pancreatic tuberculosis may be overlooked or made late. The clinical presentation and radiological finding of a pancreatic mass may suggest a malignant pancreatic tumour with very different prognostic implications and therapeutic approach. Therefore, pancreatic tuberculosis should be considered in the differential diagnosis of a pancreatic mass, especially in patients with a history of tuberculosis or in subjects from endemic areas. The diagnosis is ultimately based on histopathological and/or bacteriological findings obtained by biopsy of the pancreas, at best under echo-endoscopy, which should be considered more often in order to avoid unnecessary laparotomy or surgical resection. Tuberculosis treatment is curative in the majority of cases, the evolution under treatment is usually spectacular, and the prognosis is generally good.

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