Pseudotumoral Pancreatic Tuberculosis
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Abstract

Tuberculosis is a major public health problem in our country. Abdominal tuberculosis ranks third among the extrapulmonary localisations and account for 3% of the various topographic forms in Morocco. The pancreas and peripancreatic locations are much rarer than peritoneal and intestinal lesions. They present a pseudotumoral appearance that creates a diagnostic emergency. We report a 54 year old woman immunocompetent with pseudotumoral pancreatic tuberculosis. Diagnosis was revealed by abdominal pain predominantly epigastric, radiating in the back and associated with decreased appetite, weight loss, and asthenia. Definitive diagnosis was obtained by highlighting follicle Koester on pancreatic mass and lymph node biopsies per operative. Evolution was favorable after antituberculosis chemotherapy. The diagnosis of pancreatic tuberculosis requires a high level of presumption, we must thinking about in front of a pancreatic mass in a patient living in an endemic country.

Keywords: Pancreas, Tuberculosis, extrapulmonary, presumption.

INTRODUCTION

Tuberculosis is an infectious disease caused by Mycobacterium tuberculosis, also known as Koch’s bacillus and found most often in the lungs [1]. This disease is a public health problem not only in emerging countries but also in developed ones since its reemergence with the appearance of acquired immunodeficiency syndrome (AIDS). It continues to be common in Morocco despite regular decreases in its incidence in recent years, due first to the low incidence of AIDS and second to the national antituberculosis program [2]. Intra-abdominal infection is unusual. When it occurs, it most often involves the gastrointestinal tract, peritoneum, lymph nodes, liver, and spleen [1, 2]. Pancreatic infection is rare and occurs most often in the context of miliary or disseminated tuberculosis. In immune-deficient patients, pancreatic tuberculosis can appear as a pancreatic mass resembling a pancreatic carcinoma and lead to surgical treatment.

CASE REPORT

A 54-year-old woman with no history of tuberculosis was admitted, complaining of epigastric pain associated with mucocutaneous jaundice. He had lost 8 kilograms in the previous 2 months, had no particular medical or surgical history and no family history of tuberculosis, and was taking no medical treatment. Abdominal examination showed epigastric sensitivity, with no mass observed on palpation, no hepatosplenomegaly, and no ascites. Peripheral lymph nodes were appeared normal. Pulmonary auscultation was normal.

The blood count showed a hemoglobin level of 14 g/dL (normal: 12-16). WBC and the lymphocytes was normal. Hepatic blood test, the serum lipase and serum albumin was normal. Results of the intradermal tuberculin skin test and the interferon gamma release assay (quantIFERON-TB) were positive. Cultures for acid-fast bacilli from the sputum were negative for Mycobacterium tuberculosis. Serology for HIV 1 and 2 was also negative. Chest and abdominal x-rays were normal. Serum CA 19-9 was also normal: 0.6 U/mL (normal: 0-40).

Abdominal ultrasound showed tissue mass of the body of the pancreas measuring 3.4 cm, without ductal dilatation. Abdominopelvic computed tomography (CT) showed an oval mass of the body of the pancreas measuring 18 × 29 mm, well limited hypodense, discreetly enhanced after injection of the contrast medium, without dilatation of the wirsung canal with discrete atrophy of the pancreatic parenchyma of the tail of the pancreas downstream. No infiltration of the surrounding fat, nor of the vascular structures.

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Associated with a voluminous coelio-mesenteric lymphadenopathy of fuzzy contours measuring 25 × 28 mm widely necrosed arriving in contact with the superior mesenteric artery and vein which are respected. Liver height was normal and homogeneous. The chest was also normal (Figure-1). These findings suggested a pancreatic first an adenocarcinoma. EUS-FNA was performed using 21G needle and three samples were collected. A hypoechoic, heterogeneous, lesion of 3.8 × 3.5 mm was observed in pancreatic body. The cytopathological examination ruled out malignancy but was inconclusive. We completed with a laparoscopy which showed: a tissue mass of 4 cm in the body of pancreas with a coelio-mesenteric lymphadenopathy. The liver and peritoneum appeared normal. Biopsies from the pancreatic mass and lymph node revealed multiple granulomas with areas of caseating necrosis and clusters of multinucleated giant cells. Stains for fungi and mycobacteria were negative as was a polymerase chain reaction assay for Mycobacterium tuberculosis; however, pan-susceptible M. tuberculosis was isolated from the culture after four weeks of incubation.

Based on this finding, a diagnosis of pancreatic tuberculosis was made. The patient was treated with a quadruple antituberculosis therapy: rifampicin 600 mg/d and isoniazid 300 mg/d for 9 months, and ethambutol 25 mg/kg/d with 1.5 g of pyrazinamide daily for the first 2 months.

The symptoms disappeared after 4 months of treatment, and the patient regained 6 kg. An abdominal ultrasound performed two months after the start of the chemotherapy revealed improvement of the pancreatic lesion. By the end of treatment the patient improved gradually, with resolution of the pain and malaise and a further weight gain of 10 kg during the next 9 months. Follow-up CT showed resolution of this inflammatory mass and the lymph node (Figure-2). After a year and a half of follow-up, she remained well and free of symptoms.
DISCUSSION

Pancreatic tuberculosis is rare, even in countries with endemic tuberculosis [3, 4]. It is often associated with miliary pulmonary involvement [5]. Tuberculosis infection is experiencing a worldwide upsurge following the spread of the human immunodeficiency virus (HIV) [6]. Tuberculosis of the pancreas is rare [7]; its prevalence is 2.1 to 4.5% in the autopsy series [7, 8]. It occurs mainly on immunocompromised terrain and exceptionally in an immunocompetent subject [9, 10]. The pathogenesis of tuberculous involvement of the pancreas is poorly understood [6, 11, 12]. This impairment is thought to result from the diffusion of the bacillus by the hematogenous or lymphatic route from an occult focus (most often pulmonary) or from a latent focus reactivated by immunosuppression [8, 12]. Enzymes produced by the pancreas (pancreatic lipases and deoxyribonucleases) are thought to confer resistance to invasion by "Mycobacterium tuberculosis", which explains the rarity of pancreatic involvement in tuberculosis [11, 12].

Pancreatic tuberculosis affects men and women equally [8, 13]. The median age of occurrence in the literature is 38 years, the extremes being 22 to 71 years [8]. The symptomatology of pancreatic tuberculosis is not specific: chronic abdominal pain, pancreatic mass (evoking an exocrine or endocrine cancer), retentional jaundice, digestive hemorrhage or a pancreatic abscess and, exceptionally, a table of acute pancreatitis [1]. Imaging (ultrasound and computed tomography) has only a role in orienting the fact of non-specificity [14-16]. On the other hand, it makes it possible to eliminate the differential diagnoses (biliary cause, tumor...) and to look for other associated attacks (lymph node, ascites...) [17, 18].

The assessment in search of other localization of tuberculosis (intradermal reaction to tuberculin, BK sputm, urinary BK ...) is often negative [16]. Genomic amplification (PCR) can detect in 24 to 48 hours the presence of the "Koch bacillus" with a sensitivity of 60 to 80% when the direct examination is negative [19].

Isolated pancreatic tuberculosis raises diagnostic difficulties due to the lack of specificity of the paraclinical assessment. Diagnostic certainty is provided by the anatomopathological study either by surgical biopsy or by percutaneous puncture (echo or CT scan) or by perchoendoscopy when the granuloma is associated with caseous necrosis [6, 9, 19].

The diagnosis of pancreatic tuberculosis is challenging because of the rarity of the disease and of the nonspecific signs and symptoms of the disease. Regarding abdominal computed tomography (CT) findings, pancreatic TB usually presents as hypodense lesions in the head of the pancreas and may have calcifications [20]. However, there is no distinctive feature to discriminate between pancreatic TB and pancreatic carcinoma. EUS-FNA has emerged as a valuable modality for evaluation of the pancreato-biliary system. It helps determine the size of the lesion but also the presence of lymphadenopathy, ductal dilatation, calcifications, and vascular invasion. It provides an opportunity to sample these lesions and helps in obtaining material for cytological and microbiological evaluation. In cases of pancreatic TB, EUS usually shows a well-defined hypoechoic lesion and sometimes calcifications. EUS can also detect extra-pancreatic findings [21]. The presence of granulomas is the most common finding on histological/cytological examination with acid-fast bacilli observed in only a minority of patients. Diagnostic TB-PCR results are positive in 43% to 80% of cases [20].

In our patient, the cytopathological examination ruled out malignancy but was inconclusive. So the diagnosis of pancreatic tuberculosis was retained on an intraoperative pancreatic biopsy highlighting a gigantocellular epithoid granuloma with caseose necrosis.

Treatment is based on antibacillary antibiotic therapy: rifampicin, isoniazid, pyrazinamide and / or ethambutol for 2 months then dual therapy with isoniazid and rifampicin [7, 12, 19]. The total duration of treatment is 6 months when the pancreatic involvement is isolated. It can be extended to 9 months depending on other locations [9]. Excellent cure rates are reported with standard antitubercular therapy for 6–12 months [21]. Surgery is reserved for the treatment of complications (duodenal fistula, cold abscess, duodenal or biliary stenosis, etc.) [11, 18, 19, 22-24]. The prognosis for pancreatic tuberculosis is good in the majority of cases (90%) if the diagnosis is instituted early, recurrences are rare [11]. Mortality is around 7% in immunocompetent patients [9].

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