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Pancreatic Tuberculosis: A Diagnosis Dilemma; About A Case Report and Review of the Literature

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Abstract **Case Report**

Pancreatic tuberculosis is a rare entity despite the high prevalence of tuberculosis in the world and in Morocco in particular. The pancreas, an organ normally protected against this infection, is only affected by hematogenous diffusion or not by contiguity through the peripancreatic lymph nodes. Its clinical expression can be either in the form of a pancreatic mass simulating a cancer, or an obstructive jaundice. The diagnosis, difficult to make, is often made only after surgical exploration. Here, we report a case of pancreatic pseudo-tumoral tuberculosis, presented with abdominal pain and weight loss.

Keywords: Pancreatic tuberculosis, The pancreas, obstructive jaundice, clinical diagnosis.

INTRODUCTION

Isolated pancreatic tuberculosis is an extremely rare disease and its clinical characteristics and radiological findings are similar to those of pancreatic malignancy making it a challenging clinical diagnosis [1]. The excellent evolution after anti-tuberculous therapy makes it imperative to diagnose pancreatic tuberculosis early so we can avoid unnecessary surgical procedures. We present the case of a 39-year-old girl with pseudotumor form of pancreatic tuberculosis and through this observation we will discuss the diagnostic difficulties of this pathology.

CASE REPORT

Patient Information

A 39 years old female who has been followed in the past 6 years for recurrent generalized pruritus, with no history of personal none familial tuberculosis of cancer or other comorbidities; was admitted for etiological assessment of a chronic abdominal pain localized in the HCD and epigastrium, like a stabbing pain, of moderate intensity, radiating to inter- scapular area, triggered by the taking of copious meals and relieved by fasting, without antalgic position and without any other functional digestive signs, notably no digestive haemorrhage or jaundice or transit disorder or any extra-digestive signs. All evolving in a context of feverish sensation, night sweats and altered general condition (she lost 12 kg in 6 months) and had anorexia.

CLINICAL FINDINGS

Clinical examination showed a conscious, stable patient with a BMI=16kg/m2, OMS score 1 with signs of malnutrition such as depleted fatty panicle; abdominal examination objective a sensitivity of the right hypochondrium and epigastrium with presence of a firm, painless mass without inflammatory signs. Biology workup revealed a mild elevated liver enzymes and cholestasis with ALAT= 3,4*N, ASAT= 2,8*N, GGT= 6*N ans PAL= 2,9*N; hemoglobine level was right at 12,3g/dL with a positif PCR at 16 g/dL. Tumor markers were positive with carcinoembryonic antigen (CEA) = 2*N, carbohydrate antigen 19-9 (CA19-9)= 5*N and AFP normal at 3 ng/mL.

Diagnostic Assessment

Ultrasonography supplemented by abdominal CT scan has shown a pancreatic process measuring 31x20mm spontaneously hypodense, discreetly enhanced after injection of contrast (figure 1). It's associated with a caudal atrophy, infiltration of the surrounding fat.

Posteriorly, it infiltrates the retro-portal lamina and encompasses the superior mesenteric artery, the celiac trunk as well as the initial segments of the splenic and common hepatic arteries which remain permeable. It is separated from the aorta by a fatty separating line, encompasses the VMS which remains patent. Anteriorly, it comes into intimate contact with the small

gastric curvature and segment III of the liver with loss of the separating fatty interface in places. The liver has a normal size, regular contours without nodular lesions. In view of this aspect, the neoplastic origin was retained and the inflammatory origin was evoked last. The echo-endoscopy supports the diagnosis of malign tumor by showing a hypoechogenic lesion at the level of the pancreatic body in intimate contact with the superior mesenteric artery framing the splenic artery. Fine needle aspiration was performed and revealed inflammatory cells without neoplastic cells. The patient underwent exploratory surgery for diagnosis, and a mass with necrosis was seen in the head and body of the pancreas with multiple nodes seen. Biopsies of the pancreas and lymph nodes showed inflammatory changes with multiple granuloma and caseous necrosis without evidence of malignancy.

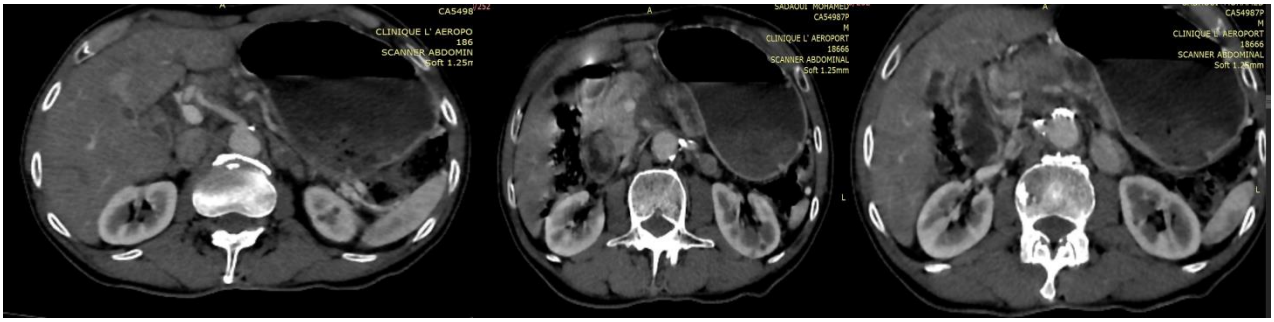

Figure: CT scan showing a pancreatic process locally infiltrating the body with vascular engulfment

Therapeutic Intervention

Anti-tuberculosis chemotherapy was initiated (2RHZE/4RH).

Follow Up and Outcomes

The clinical evolution was good with complete clearance of the lesion after 6 months of anti-tuberculosis treatment.

DISCUSSION

Pancreatic tuberculosis is a very rare pathology, even in endemic countries with less than 5% of cases, as shown in autopsy series [3]. 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, an incidence of 4.7% [4]. Bhansali reported no cases of pancreatic tuberculosis in a series of 300 cases of abdominal tuberculosis in India [5]. Xia and colleagues reported 16 patients from China with tuberculosis of the pancreas and peripancreatic lymph nodes; the predominant symptoms were abdominal pain (75%–100%), anorexia, weight loss (69%), malaise, weakness (64%), fever and night sweats (50%) [6]. It is not yet clear how the infection can only affect the pancreas. Pancreatic secretions have been reported to have an antitubercular effect in vitro, suggesting a potential protective mechanism. Nonetheless, several possible mechanisms for pancreatic location of tuberculosis have been discussed. These include hematogenous spread, disseminated tuberculosis in the setting of advanced immunosuppression, and reactivation of previous abdominal tuberculosis located in adjacent lymph nodes [7].Clinical manifestations includes abdominal pain (75 - 100%), anorexia and weight loss (69%), fever and night sweats (50%), anemia (50%), and obstructive jaundice (30%) and infrequently, pancreatic tuberculosis may present as acute pancreatitis with radiographic findings of pancreatic enlargement and oedema [8, 9]. Laboratory abnormalities including mild anemia, lymphocytopenia, elevated transaminases and alkaline phosphatase have been seen in approximately 50% of cases. [10]. Radiologic features including ultrasonography (US), CT or endoscopic ultrasound (EUS) usually show a single tissue process in 62.5% of cases and show usually a heterogeneous appearance. It is located often in the head (56%) and is associated with a peripancreatic lymphadenopathy in 75% of cases. Pancreatic lesions resulting from mycobacterial tuberculosis infection are often heterogenous and multicystic and can mimic pancreatic cystic neoplasm [11-13]. Invasive diagnostic techniques such as CT/US-guided percutaneous biopsy and surgical biopsy are more reliable and definitive in contrast to noninvasive techniques. In fact, tissue obtained from biopsy can be evaluated for pathologic and microbiological examination; histologically, the presence of caseous granulomatous inflammation and positive stain for acid-fast bacilli are suggestive of tuberculosis. Typical epithelioid and gigantocellular granuloma is found in 60% of cases and rarely caseous necrosis is seen. Microbiological ex- amination is used equally to confirm the diagnosis and is based essentially on cultures for mycobacteria, which take up to 6 weeks to grow [14-17]. Once the diagnosis of tuberculosis infection is confirmed, specific treatment must be instituted and proves beneficial in the majority of cases. This treatment must be prolonged, continuous, have good tissue penetration, intra- and extra-cellular bactericidal power and use several drugs in order to reduce the risk of emergence of resistant strains. The duration of treatment is usually 6 to 9 months, but in case of resistant strains, especially to isoniazid or rifampicin, it could be extended to at least 12 months with antibiotics that are effective in the antibiogram

[18]. The prognosis of pancreatic tuberculosis is good if the diagnosis is made early and if antituberculosis treatment is prescribed. However, in a review of the literature, a mortality rate of 7% has been reported in immunocompetent patients [18].

CONCLUSION

Primary pancreatic tuberculosis is extremely rare and diagnosis is a real challenge. Imaging studies are non- specific and do not allow differentiation with an adenocarcinoma. The diagnosis must be considered in the presence of a pancreatic mass in a young patient, living in an endemic country or immunocompromised, which will allow a biopsy to be performed in the first instance and will avoid a complex and unnecessary laparotomy.

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