Tuberculosis of the pancreas mimicking carcinoma

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The HIV/AIDS pandemic has affected the South African population more than most. Tuberculosis (TB) is endemic in South Africa and this has been severely aggravated by the advent of AIDS.[1] and [2] Tuberculosis presents in many guises, some common, some extremely rare. Often, it is only a high index of suspicion that helps make the diagnosis. We present herein a patient who presented with obstructive jaundice and a mass in the head of the pancreas, which resolved on anti-TB treatment, to serve as a caveat to others.

A 33-year-old female was referred to the surgery department of the Pretoria Academic Hospital. She complained of pain in the right hypochondrium, which had been present for two weeks. The pain was constant and not affected by meals. She described her stools as being green in color. The patient weighed 75 kg, which was her approximate expected weight for height. She was jaundiced. None of the typical constitutional symptoms of tuberculosis were reported on questioning, nor was the patient observed to have fever, nausea, vomiting or night sweats during her hospital stay. There was no medical or surgical history of any note. The patient did not have any known TB contacts. The patient had oral candidiasis. There was a 3 cm mass in her left supraclavicular fossa fixed to underlying structures. A vague epigastric mass was felt on clinical examination, which was otherwise unremarkable – the liver appeared to be of normal size and the gallbladder was not clinically palpable. Vital signs were normal throughout her hospital stay.

On admission, the patient's full blood count was normal, the liver functions were typical of obstructive jaundice (bilirubin 117.3 μmol/l, ALP 401 IU/l, γGT 462 IU/l, ALT 64 IU/L, AST 57 IU/L), and the CRP was 90 mg/l. Chest X-ray was normal. Due to the endemic nature of tuberculosis in South Africa, a purified protein derivative (PPD) skin test was not performed. Pancreatic enzymes were not tested. Serum albumin was 24 g/l on admission.

Abdominal ultrasound showed a large cystic mass in the head of the pancreas. Intra- and extra-hepatic bile ducts were dilated. A computed tomography (CT) scan confirmed the presence of a 4.47 × 3.37 × 4.4 cm cystic mass in the head of the pancreas (Figure 1). The mass was thick-walled with septae. There was no sign of intra-abdominal lymphadenopathy. In consultation with the radiologists, a preliminary diagnosis of a periampullary carcinoma was made.

Fine needle aspiration (FNA) of the supraclavicular lymph node was performed. This yielded copious numbers of TB bacilli. The patient also tested positive for HIV, with a CD4 count of 164. A thorough examination showed no other sites of tuberculous involvement. Due to the patient's general condition, as well as the diagnosis of tuberculosis based on the supraclavicular lymph node, a CT-guided FNA of the mass in the pancreas was not attempted. It was considered possible that the mass in the pancreas was due to TB.

The patient was started on anti-TB treatment with a standard regimen of rifampin, isoniazid and pyrazinamide. She was also referred for antiretroviral therapy. Rapid improvement of symptoms and resolution of the jaundice followed within a period of several days, as demonstrated by the rapid normalization of liver function tests (Table 1). The CT scan was repeated a month later and showed that the maximal diameter of the cystic mass had decreased to 3.71 cm and the bile ducts were no longer dilated (Figure 2). Treatment for TB,
as well as highly active antiretroviral therapy (HAART) was continued. Liver functions had normalized at the one-year follow-up.

**Figure 1.** CT scan showing the mass at presentation

<table>
<thead>
<tr>
<th>Date</th>
<th>Total bilirubin (N = 0–21 μmol/l)</th>
<th>ALP (N = 40–120 IU/l)</th>
<th>γGT (N = 0–35 IU/l)</th>
<th>ALT (N = 5–40 IU/l)</th>
<th>AST (N = 5–40 IU/l)</th>
</tr>
</thead>
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<tr>
<td>Admission</td>
<td>117.3</td>
<td>401</td>
<td>462</td>
<td>64</td>
<td>57</td>
</tr>
<tr>
<td>1 week</td>
<td>54.8</td>
<td>310</td>
<td>217</td>
<td>42</td>
<td>47</td>
</tr>
<tr>
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<td>13.0</td>
<td>99</td>
<td>147</td>
<td>315</td>
<td>211</td>
</tr>
<tr>
<td>3 months</td>
<td>12.6</td>
<td>85</td>
<td>84</td>
<td>31</td>
<td>29</td>
</tr>
<tr>
<td>1 year</td>
<td>10.0</td>
<td>81</td>
<td>37</td>
<td>29</td>
<td>22</td>
</tr>
</tbody>
</table>

In a landmark study in 1941, Auerbach reported on 1656 autopsies on patients with TB. Of these, 297 had miliary TB (18%) and only 14 of the 297 cases had any evidence of pancreatic involvement (4.7%). In 1977 Bhansali reported on 300 cases of abdominal TB. There were no cases of pancreatic TB.4 Pancreatic involvement in TB is extremely rare. This is thought to be due to the anti-tuberculous effect of pancreatic enzymes.5

Many case reports have been published, but very few studies of pancreatic TB have been made. Several authors have collectively reviewed these case reports. Some authors have suggested diagnostic protocols.6 However, due to the paucity of cases, no consensus has been achieved. The HIV/AIDS pandemic may very well occasion larger clinical studies and lead to accepted protocols.

Due to the rarity of a tuberculous mass in the head of the pancreas, and the fact that both clinically and radiologically it mimics a periampullary carcinoma,[7], [8] and [9] these patients are often diagnosed at laparotomy. The presence of tuberculous cervical lymphadenopathy in our case obviated this.
The mechanism of spread of tubercle bacilli to the pancreas is uncertain. It is speculated that this is either through hematogenous spread from an occult lesion in the lung or abdomen, or via direct spread from contiguous lymph nodes. A third theory is that dormant bacilli in an old lesion can be reactivated during an immunosuppressed state.10

Clinical features of pancreatic TB are non-specific. In a meta-analysis of 12 published reports, Lo et al. listed constitutional symptoms (weight loss, fever, malaise, night sweats), epigastric pain, nausea, vomiting, diarrhea, right upper quadrant pain and obstructive jaundice as the most common presenting manifestations.11 Most of the cases analyzed by them had no extra-pancreatic tuberculous lesions. Biochemistry and hematology are usually not helpful in making the diagnosis. In our case the diagnosis of obstructive jaundice was not specific.

The initial diagnosis of a pancreas mass has often been made on abdominal sonar. This mass has been described as being solid, cystic, low density, hypo-echoic, calcified, etc.12 In most cases an abdominal CT scan has also been performed, which has confirmed the presence of a mass.

Pombo et al. retrospectively reviewed the CT scans of six patients with proven TB of the pancreas.6 They concluded that in HIV-negative patients there is a non-specific focal mass lesion, whereas in HIV-positive patients there is a greater spectrum of findings, including focal mass lesions, multiple small low-attenuation pancreatic nodules or diffuse enlargement of the gland. They also listed a number of ancillary findings to support the diagnosis, including low-attenuation peripancreatic and periportal lymphadenopathies with peripheral rim-enhancement. The CT scan of our patient, who was HIV-positive, revealed a rather homogenous cystic focal lesion in the head of the pancreas.
Various methods have been described to confirm the diagnosis. In a review of 51 case reports in the literature by Demir et al., seven were diagnosed by CT-guided FNA and 44 underwent diagnostic laparotomy. Several case reports detail patients who have undergone pancreaticoduodenectomy to make the diagnosis. Trial of therapy may be indicated in those patients who are at high risk for TB and where there is evidence of TB elsewhere in the body, as was done in our patient.

TB of the pancreas is rare. However, one of the great mimics of the past is experiencing a resurgence in the AIDS era, and it would be wise to consider the diagnosis of TB even in such unlikely organs as the pancreas.

References


10 J.B. Stambler, M.I. Kilbaner, C.M. Bliss and J.T. LaMont, Tuberculous abscess of the pancreas, Gastroenterology 82 (1982), pp. 922–925.


