**Tuberculose ganglionnaire abdominale et hypertension portale**


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**Summary** Abdominal tuberculosis involving the portal vasculature is a rare phenomenon. We retrospectively reviewed the imaging findings of 183 cases of abdominal tuberculosis at our institution from 2002 to 2010 and found thrombosis of the splenoportal axis associated with abdominal lymphadenopathy in seven patients. However, there was no relationship between the lymph nodal size and development of thrombosis. Reversibility was noted in one patient, who had near complete recanalisation of portal vein. Mechanisms, other than direct mass effect on the splenoportal axis, may be involved, like contiguous spread of inflammation or granulomas in the vessel wall.

**Introduction** Abdominal tuberculosis is a commonly encountered entity in the developing world. It may involve the peritoneum, bowel, lymph nodes or the solid organs. Portal and peripancreatic group of nodes are frequently involved [1,2]. In spite of the common occurrence of lymphadenopathy in these locations, the incidence of portal/splenic vein thrombosis is limited to a few case reports only. We reviewed 183 cases of abdominal tuberculosis for the presence and distribution of lymph nodes as well as associated portal/splenic vein thrombosis, if any.

**Material and methods** We retrospectively analyzed the imaging records of 183 patients of abdominal tuberculosis who underwent CT examination at our hospital from 2002 to 2010. The CT examinations were performed on a single slice helical CT machine (Somatom plus 4/AR Star, Siemens, Erlangen, Germany). The images were obtained after bowel opacification with oral contrast. One hundred millilitres of intravenous contrast was also administered and the scanning was done in the portal venous phase (50–60 seconds post-injection). The ultrasound and colour Doppler were performed whenever indicated on HDI 3000, ATL Philips.

The CT images were reviewed for the presence, distribution and morphological appearance of enlarged abdominal lymph nodes. The status of portal, splenic and superior mesenteric veins was recorded. Presence of thrombosis and...
collaterals, if any, were also documented. The diagnosis of abdominal tuberculosis was made on the basis of characteristic clinical features and imaging findings. Among the seven patients with portal/splenic vein thrombosis, cytological/histological confirmation was available in six patients. Follow-up imaging was available in five of these patients, performed over a period of 6 to 18 months.

Results

One hundred and eighty-three cases of abdominal tuberculosis were reviewed and the detailed findings were recorded. Abdominal lymphadenopathy was observed in 127 patients (69.4%). The splen portal axis was patent in 176 cases (96.2%) while seven patients (3.8%) showed evidence of thrombosis. The thrombosis involved the portal vein (n = 6), splenic vein (n = 2) and superior mesenteric vein (n = 1). One patient had portal, splenic and superior mesenteric vein thrombosis whereas the rest had isolated portal vein (n = 5) or splenic vein (n = 1) thrombosis (Table 1). Six out of seven cases of portal vein thrombosis had adjoining abdominal lymphadenopathy, which were homogeneous in one (Fig. 1) and necrotic in five patients (Figs. 2 and 3). The single case of portal vein thrombosis that did not have nodes on CT, had history of the same having been observed at a prior surgery. Splenic vein thrombosis was present in a young girl who had disseminated tuberculosis with pancreatic involvement (Fig. 2).

Thrombosis was observed in four of the seven patients at the time of presentation. Three patients, however, developed venous thrombosis later in the course of their illness, while they were on antitubercular treatment (ATT). In two of these, the initial CT scan showed extensive periportal lymphadenopathy with encasement of the portal vein. The portal vein was however patent. Though the nodes regressed on ATT, portal vein thrombosis appeared (Figs. 3 and 4). The third patient presented with gastric outlet obstruction and was operated for a duodenal sticture. At surgery, portal and pancreaticoduodenal nodes were observed. Histopathological examination of these nodes was suggestive of tuberculosis. At this stage, there was no record of portal cavernoma or collaterals elsewhere, though no CT examination was performed preoperatively. Patient was started on antitubercular therapy. In a CT done 6 months later, there was complete resolution of lymphadenopathy; however portal cavernoma and retroperitoneal collaterals were identified (Fig. 5).

Five out of these seven patients underwent follow-up CT examination after a course of ATT for the assessment of adequacy of response. One patient, a young girl, died of disseminated disease while one was lost to follow-up. Four patients did not show any improvement in venous thrombosis/collaterals. In one case, however, there was near complete recanalization of the portal vein with resolution of collaterals and ascites.

Discussion

There is a resurgence of tuberculosis in the world with the onset of HIV infection. There is also a relative increase in the incidence of extrapulmonary involvement. The association of pulmonary and abdominal tuberculosis is uncommon in clinical practice. Fewer than 10% patients have this double localization [3]. Abdominal tuberculosis may involve the peritoneum, bowel, lymph nodes or the solid organs. Lymphadenopathy is seen in up to two-thirds of patients with abdominal tuberculosis [1]. The involvement of portal and peripancreatic groups is the most common [2].

Enlarged portal lymph nodes may result in compression of the portal vein and/or common bile duct, leading to portal hypertension (PHT) or obstructive jaundice, but in practice, it has been reported only rarely. In 1845, Knight reported the first case of PHT and obstructive jaundice due to compression of the portal vein by tuberculous lymph nodes [4]. In 1956, Caroli and Paraf, using splenoportography, made the first preoperative diagnosis of portal vein compression and PHT in a patient with abdominal tuberculosis [5]. The British Thoracic Society research committee reported portal thrombosis after medical treatment for tuberculosis of lymph nodes in only nine of 101 patients [6]. We have come across only seven patients of PHT among 183 cases of abdominal tuberculosis.

PHT in abdominal tuberculosis can be due to portal vein thrombosis, splenic vein thrombosis or simply portal vein compression. In our patients, we observed portal vein (n = 6), splenic vein (n = 2) and superior mesenteric vein (n = 1) thrombosis. The suggested mechanisms of PHT in TB are: portal vein thrombosis secondary to lymph nodes, splenic vein thrombosis due to pancreatic tuberculosis or retroperitoneal tubercular abscess, hepatic TB causing sinusoidal compression, and hepatic outflow obstruction due to constrictive pericarditis [6—10]. A case of PHT due to compression by portal lymph nodes without portal vein thrombosis has also been reported [11]. De Backer et al. have reported a case in which caseous lymphadenopathy at the porta caused encasement and stenosis of the portal vein and subsequent collateral formation [12]. In six of our seven patients, the venous thrombosis was secondary to lymph node involvement. One patient developed splenic vein thrombosis due to pancreatic tuberculosis. Two similar cases of PHT in pancreatic tuberculosis have been reported by Schneider et al. [13] and Takhtani et al. [14].

Even though the incidence of bulky portal and peripancreatic lymphadenopathy surrounding the splen portal axis is high, venous thrombosis is observed only rarely. We also observed that the likelihood of thrombosis does not appear to be related to the size of the nodes. Several of the 126 patients with portal and peripancreatic nodes had large, bulky nodes with a patent splen portal axis. On the contrary, majority of our patients with venous thrombosis had small nodes. Two patients amongst this group with bulky lymphadenopathy did not show any thrombosis in the initial CT scan. The thrombosis developed subsequently on treatment, once the size of the nodes had decreased (Fig. 4). Similarly, of our two cases of pancreatic involvement (of 183 patients), the one with large pancreatic head and peripancreatic nodes (not shown) had a normal splen portal axis, while a small area of involvement of the dorsal aspect of the body resulted in splenic vein thrombosis in the second patient (Fig. 3). This prompts us to postulate the presence of other causative factors apart from compression, which contribute to this thrombosis. One of these may be contiguous spread of inflammation with perivascular cuffing.

Table 1  Imaging findings in patients with splenoportal axis thrombosis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>Cytology/ Histopath</th>
<th>Location of adenopathy</th>
<th>Appearance of nodes</th>
<th>Site of thrombosis</th>
<th>Ascites</th>
<th>Splenomegaly</th>
<th>Other organ involvement</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27/M</td>
<td>No</td>
<td>Peri-Pancreatic/ Coeliac axis</td>
<td>Necrotic</td>
<td>PV/ SV/SMV</td>
<td>+</td>
<td>+</td>
<td>Cæcum</td>
<td>Persistent thrombosis</td>
</tr>
<tr>
<td>2</td>
<td>36/M</td>
<td>Yes</td>
<td>Peri-Pancreatic</td>
<td>Necrotic</td>
<td>PV</td>
<td>—</td>
<td>—</td>
<td>Duodenum, lungs (AFB+)</td>
<td>Lost to follow-up</td>
</tr>
<tr>
<td>3</td>
<td>32/M</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>PV</td>
<td>+</td>
<td>—</td>
<td>D2 narrowing</td>
<td>6 months later, ascites and collaterals ↓</td>
</tr>
<tr>
<td>4</td>
<td>25/F</td>
<td>Yes</td>
<td>Peri-Pancreatic</td>
<td>Homogenous</td>
<td>PV</td>
<td>—</td>
<td>—</td>
<td>Lungs</td>
<td>Developed peritonitis and died</td>
</tr>
<tr>
<td>5</td>
<td>35/F</td>
<td>Yes</td>
<td>Peri-Pancreatic</td>
<td>Necrotic</td>
<td>PV</td>
<td>—</td>
<td>—</td>
<td>Cervical nodes</td>
<td>Regression of nodes 1 year later — portal cavernoma persistent</td>
</tr>
<tr>
<td>6</td>
<td>32/F</td>
<td>Yes</td>
<td>Right para-aortic from renal hilum to aortic bifurcation with Right hydronephrosis</td>
<td>Necrotic</td>
<td>SV</td>
<td>—</td>
<td>—</td>
<td>Pancreas</td>
<td>No change Drug resistant</td>
</tr>
<tr>
<td>7</td>
<td>8/F</td>
<td>Yes</td>
<td>Peri-Pancreatic</td>
<td>Necrotic</td>
<td>PV</td>
<td>—</td>
<td>—</td>
<td>Mediastinum</td>
<td>Regression of nodes 1 year later — portal cavernoma persistent</td>
</tr>
</tbody>
</table>

SMV: superior mesenteric vein; PV: portal vein; SV: splenic vein.
Abdominal tuberculous lymphadenopathy

Figure 1  Twenty-five-year-old female with biopsy proven tubercular adenopathy. Contrast-enhanced CT of the abdomen shows (a) portal cavernoma formation with; (b) homogeneous nodes at the porta (arrow) and thrombus within the portal vein. Focus of calcification is also seen in one of the nodes. Ultrasound of the same patient (c) shows multiple tortuous collaterals as well as the periportal nodes (arrow).

Figure 2  Thirty-two-year-old female with pancreatic tuberculosis and thrombosis of the splenic vein (a, b). Hypodense lesion with ill-defined margins is seen in the posterior aspect of the distal body of pancreas. Splenic vein is not seen. Extensive perisplenic collaterals are seen. (c) Low density retrocaval and para-aortic nodes are present. Right-sided hydronephrosis also present.

Figure 3  Thirty-five-year-old female on follow-up after antitubercular therapy. (a, b) Initial CECT of the abdomen shows rim-enhancing periportal nodes. Portal vein shows normal enhancement, although it is encased and narrowed by the periportal nodes (arrow in b). Follow-up CECT of the abdomen after 1 year (c, d) shows regression of the periportal nodes but formation of portal cavernoma, which persists after 1.5 years.
granulomas in the vessel wall with subintimal fibrosis and intraluminal thrombi [15].

The PHT due to tuberculous lymph nodes has been reported to be reversible in only one case previously, where only compression of the portal vein was seen [11]. In all other reported cases in literature, the PHT was secondary to portal or splenic vein thrombosis and was persistent in spite of the regression of lymphadenopathy. Our cases showed a similar course with the venous thrombosis being irreversible over a follow up of 6–18 months in six of the seven patients. In one case with nodal and duodenal involvement, there was diminution in the portal collaterals with recanalisation of the portal vein on follow-up (Fig. 5). The ascites seen previously also regressed. The improvement in the venous thrombosis, however, did not result from a decrease in the size of the nodes, as this patient did not show sizable lymphadenopathy even in the initial CT scan. The lymph nodes, in this case, were documented at a prior surgery.

Although gastro-intestinal bleeding due to ruptured gastric varices in a case of hepatic tuberculosis has been reported [9], all our patients were incidentally detected to have PHT and varices on investigation with no instance of upper gastro-intestinal bleed as the primary presentation. All our patients remained asymptomatic on follow-up and were not treated for the PHT, as has also been the case in most of the previous reports. The appropriate treatment of asymptomatic patients in splenic vein thrombosis remains controversial [16]. Loftus et al. have noted no difference in survival or bleeding between patients treated with splenectomy and those who were only observed [17]. On the other hand...

Figure 4 Twelve-year-old female on follow-up after antitubercular therapy. (a) Initial CECT of the abdomen shows rim-enhancing periportal nodes. Portal vein shows normal enhancement, although it is encased and narrowed by the periportal nodes with small filling defect within suggesting portal vein thrombosis (arrow). Follow-up CECT of the abdomen after 1 year (b) shows regression of the periportal nodes but formation of portal cavernoma, which persists after 1.5 years.

Figure 5 Thirty-two-year-old male with past history of resection for tubercular duodenal stricture causing gastric outlet obstruction. CECT of the abdomen shows (a, b) portal vein thrombosis with cavernoma formation and peripancreatic collaterals. Mild wall thickening of the antropyloric region is seen. Follow-up scan (c, d) after 9 months shows resolution of portal cavernoma formation with normal opacification of the portal vein, signifying a reversible phenomenon.
hand, some recommend prophylactic surgery for splenic vein thrombosis when thrombosis is due to benign diseases as these may demonstrate a high rate of gastric variceal hemorrhage [18]. Our single case of splenic vein thrombosis remained asymptomatic on an 18-month follow-up.

In conclusion, abdominal tuberculosis rarely causes PHT, secondary to portal or splenic vein thrombosis. Though usually irreversible, these patients tend to remain asymptomatic and no specific treatment may be required for the thrombosis. Although it is beyond the scope of this article, our observations prompt us to hypothesize if the high incidence of extrahepatic portal vein obstruction encountered in the developing world could at least, in some patients, be related to a prior tubercular infection. However, in absence of a definite history and no stigmata of nodal involvement apart from calcification in some cases, this cause-effect relationship cannot be established.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

References