Mesenteric vasculopathy in intestinal tuberculosis

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Background: Involvement of mesenteric vessels in intestinal tuberculosis and its role in the pathogenesis of the intestinal changes have not been studied histologically. Aim: To study mesenteric vessels in patients undergoing surgery for complications of intestinal tuberculosis. Methods: Resected intestinal specimens from 68 patients presenting with intestinal perforation and intestinal obstruction were examined; involvement of the major mesenteric vessels was evaluated. Results: Granulomas were seen in the vessel wall in one case and near the vessel wall in 11 cases, intraluminal thrombi were seen in 23 cases, and subintimal fibrosis in nine cases. Perivascular cuffing was seen in intramural and subserosal vessels in ten cases. Conclusions: Changes in the vessel wall may lead to gut ischemia, which may contribute to the development of strictures and stercoral perforation in intestinal tuberculosis. [Indian J Gastroenterol 1997; 16: 134-136]

Key words: Intestinal ischemia, vasculitis

Tuberculosis is a chronic disease where healing with fibrosis occurs simultaneously with ongoing infection. The pathogenetic events which culminate in ulcer formation, perforation and stricture are not clear: specifically, involvement of blood vessels and its contribution to these events have not been studied adequately.

Sen and Kinare1 postulated that inflammation of the aortic wall in the middle aortic syndrome was a hypersensitivity reaction to tuberculosis. Vasculitis is a feature of tuberculosis of the central nervous system2 and lungs.3 Shah and Ramakantan4 documented angiographic changes in the mesenteric vessels in intestinal tuberculosis. The histologic equivalent of these changes have not been studied.5 Also, whether these changes are merely reactive or due to direct involvement of the vessels is not known.

We therefore studied the histologic changes in the mesenteric vessels in patients operated on for ulceroconstrictive lesions of intestinal tuberculosis.

Methods

Sixty eight patients (30 men, 38 women; aged 12 to 60 years) with histologically proved intestinal tuberculosis were studied prospectively over 3 years. Their clinical presentations and operative findings and histology of the resected specimens were noted.

Fifty three patients had presented with clinical and investigational evidence of intestinal obstruction and 15 with evidence of perforative peritonitis. At surgery the perforated ulcers were in the ileum and the strictures in the ileum and ileocecal regions. The surgical procedures done were resection-anastomosis and hemicolectomy.

Mesenteric blood vessels in the resected specimens were dissected from their root to the periphery. Based on their size they were classified as large (primary branches of the superior mesenteric artery), medium (arcuate vessels), small (vasa recta), or intramural and subserosal vessels. Hematoxylin-eosin and elastic van Gieson stains were used to study the internal elastic lamina of these vessels.

Results

The lesions noted in the vessel lumen, vessel wall, and adjacent to the vessels are listed in the Table. Figs 1 to 6 are photomicrographs of typical lesions seen.

<table>
<thead>
<tr>
<th>Table: Vasculitis in specimens of intestinal tuberculosis</th>
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<tbody>
<tr>
<td>Ulcer perforation</td>
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<tr>
<td>n=15</td>
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<tr>
<td></td>
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<tr>
<td>Granuloma in vessel wall</td>
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<tr>
<td>Granuloma in adventitia</td>
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<tr>
<td>Thrombus in vessel</td>
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<tr>
<td>Subintimal fibrosis</td>
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<tr>
<td>Perivascular cuffing*</td>
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</tbody>
</table>

*Seen only in intramural and subserosal vessels

Of the 68 cases, 25 had caseating granulomas and 43 had noncaseating granulomas.

Discussion

The pathogenetic events in intestinal tuberculosis that culminate in ulceration, fibrosis, stricture formation and perforation are not known. At least some of these may be secondary to ischemia due to involvement of adjacent vessels, either directly or as a reaction. On the other handvascular changes, if present, may be only an incidental effect.

We showed changes in the large, medium and small-sized mesenteric vessels in 43 of 68 patients with intestinal tuberculosis; in five of these the presence of granulomas in or adjacent to the vessel wall suggested direct involvement. This was accompanied by formation of thrombus in the large vessels in ten cases, along with subintimal thickening and fibrosis in four cases. In addition, perivascular
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Fig 1: Granuloma within a medium-sized vessel (H&E, 160X)

Fig 2: Granuloma within a medium-sized vessel (H&E, 160X)

Fig 3: Granuloma near large-sized vessel; artery also shows subintimal fibrosis (EVG, 160X)

Fig 4: Large-sized vessel showing recanalizing thrombus (H&E, 160X)

Fig 5: Large-sized vessel showing disruption of internal elastic lamina with subintimal fibrosis (EVG, 160X)

Fig 6: Perivascular cuffing of subserosal vessel (H&E, 160X)

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cuffing was seen in the intramural and subserosal vessels in ten cases; these we believe are nonspecific lesions, occurring probably as a hypersensitivity reaction.

We postulate that these vascular changes lead to ischemia of the bowel which may contribute to the development of ulcers, perforation, fibrosis and strictures. Segments of the bowel proximal to the strictures also suffer ischemia and are subjected to high intraluminal pressures; this can lead to stercoral perforation.

References
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