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Original Article

Vascular anatomy of strictured small bowel

Magnus Jayaraj Mansard,[1] Upender Rao,[1] Pradeep R,[1] Anuradha Sekaran,[2] G.V. Rao,[1] D.N. Reddy[3]

Department of Surgical Gastroenterology,[1]
Department of Pathology,[2]
Department of Gastroenterology,[3]
Asian Institute of Gastroenterology,
6-3-661, Somajiguda,
Hyderabad- 500082.

Correspondence:
Dr. Magnus Jayaraj Mansard
Email: jeymagnus@gmail.com

ABSTRACT

Aim: To investigate the role of ischemia in the pathogenesis of small bowel strictures.
Methods: Vascular anatomy of 39 small bowel strictures was studied using modified Spalteholtz method. Ten normal small bowel segments were studied as controls.
Results: 71.8% of small bowel strictures showed block in the mesenteric vessels (p=0.008). Subset analysis of tuberculous strictures showed block in the mesenteric vessels in 70.8% of strictures (p=0.0098).
Conclusions: Ischemia plays a significant role in the pathogenesis of small bowel strictures. Mesenteric vasculopathy has a significant association with tuberculous strictures of the intestine.

KEYWORDS: Intestinal stricture, tuberculous stricture, tuberculous vasculopathy, mesenteric ischemia, intestinal obstruction

Introduction

In a patient with no history of previous surgery, or obstructing hernia or metastatic disease, benign stricture of the small bowel is an important cause of small bowel obstruction. In India, where tuberculosis is endemic, intestinal tuberculosis is probably the most common cause of small bowel stricture formation.[1] Ischemic strictures have also been noted to be quite common in Indians between 20 and 40 years of age.[2] Crohn's disease, which has been rare in the subcontinent in the past, is being noted with increasing frequency over the last two decades.[3] Other less common causes of stricturing bowel disease include radiation induced stricture and NSAID induced stricture. A large percent of strictures with no identifiable etiology are grouped as 'idiopathic' strictures.

The strictures have been postulated to be the result of mucosal and transmural inflammatory fibrosis of the intestinal wall due to tuberculosis and Crohn's disease. Stricturing type of Crohn's disease, in contrast to the inflammatory and penetrating types seldom responds to medical treatment and requires surgical management.[4] Similarly the hyperplastic type of tuberculous strictures are known to progress in severity and may require resectional surgery.[5] Elucidation of the pathophysiology of these strictures might help in the better understanding and treatment of these diseases.

While ischemic strictures have mesenteric vascular block as the obvious cause of the stricture, the role of ischemia in the etiology of the other strictures remains to be elucidated. Endarteritis has been identified as the cause of radiation induced strictures. Anthony et al[6,7] investigated the vascular anatomy of small bowel specimens prepared by the Spalteholtz's method in meticulously designed experiments. They demonstrated the role of ischemia in the etiology of strictures caused by Crohn's disease and by indomethacin. Tuberculosis is known to cause vasculitis in all systems, especially in the lungs[8] and the central nervous system.[9] However, the role of

ischemia in the pathophysiology of intestinal tuberculosis and in the "idiopathic' group of strictures remains to be elucidated. This study was undertaken to identify the role of ischemia in the pathogenesis of these strictures.

Methods

Patients with strictures of the small intestine who underwent resectional surgery at our institute between August 2006 and December 2008 were included in the study. The strictures were resected with preservation of the main vascular pedicle with the mesentry. Immediately after resection of the small bowel strictures, the most representative stricture was chosen for routine histopathological examination. Of the other strictures, the one showing the earliest form of the disease was chosen for the study.

The specimen to be studied was cleared of its luminal contents. The main vascular pedicle supplying the strictured part was identified in its mesentry by transillumination. The chief vessel supplying the strictured part was cannulated using an 18G needle and India ink was injected. The dye was injected till it could be noted in the serosal capillaries running on the surface of the bowel. The vascular pedicle was ligated to prevent extravasation of the contrast. The specimen was mounted on a thermocol pad and preserved in formalin solution overnight.

From the next day, the specimen was prepared by the Spalteholtz technique to render it transparent. This included placing the solution in hydrogen peroxide solution to bleach the formalin stain and progressive dehydration by placement in increasing concentrations of alcohol solution and xylene. The specimen was finally placed in methylsalicylate solution which rendered it transparent. On transillumination, the India ink in the blood vessels stood out making the vascular anatomy of the strictured part of the bowel available for study. Specimens undergoing such preparation were returned back for routine histopathological examination at any point of time, if the examination of the first specimen happened to be inconclusive for the diagnosis of the cause of the stricture.

Segments of normal small bowel incidentally resected during surgery for other conditions during the study period were also studied using the Spalteholtz method and were used as controls. Informed patient's consent and Institute review board approval were obtained. For statistical analyses, the Fisher's exact test was applied using the SPSS 11.0 statistical software. An associated probability of 5% or less was considered significant.

Results

During the study period, 127 patients underwent resectional surgery for benign small bowel stricture disease. They were classified into 5 groups depending on the pathological diagnosis of the strictures (**Table 1**). Eight patients were in a miscellaneous group with strictures caused by radiation and by omental bands or hernia rings compressing a loop of bowel. Fifty six patients who had multiple strictures were included in the study. Nine patients were excluded because of difficult mesenteric cannulation due to thickened mesentry. Eight other patients were excluded as the specimens were taken back for histopathological examination when the first specimen's examination was inconclusive.

Of the remaining 39 patients included in the study, 24 patients had tuberculosis, 7 patients had Crohn's disease, 3 patients had ischemic strictures and 5 patients had nonspecific strictures. **Table 1** and **Figure 1** show the distribution of strictures of different etiologies.

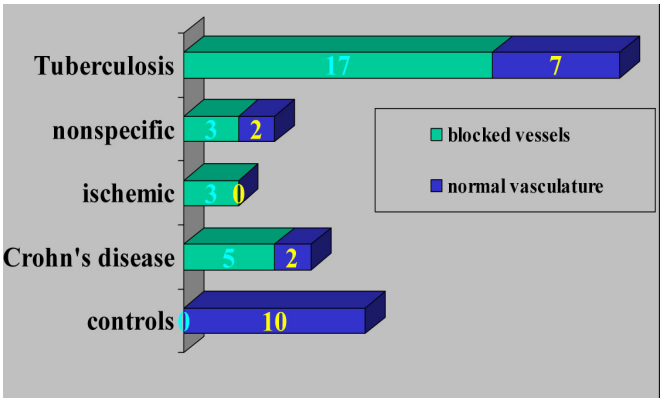

Figure 1:

Table 1: Shows the distribution of strictures of different etiologies

	Tuberculous stricture	Crohn's disease	Ischemic stricture	Miscellaneous group	Nonspecific stricture	Controls
Total number of patients with strictures	61	26	6	8	26	-
Number of patients with multiple strictures	27	14	3	0	12	-
Number of patients included in the study	24	7	3	0	5	10
Number of patients with blocked mesenteric vessels	17	5	3	0	3	2

All normal segments of small bowel used as controls showed normal vasculature (**Figure 2**). 71.8% of patients with strictures of the small bowel showed block in the mesenteric vessels leading to the stricture (**Figure 3**) (p=0.008). 70.8% of patient with tuberculous strictures were found to have block of the mesentric blood vessels in the region of the stricture (p= 0.0098).

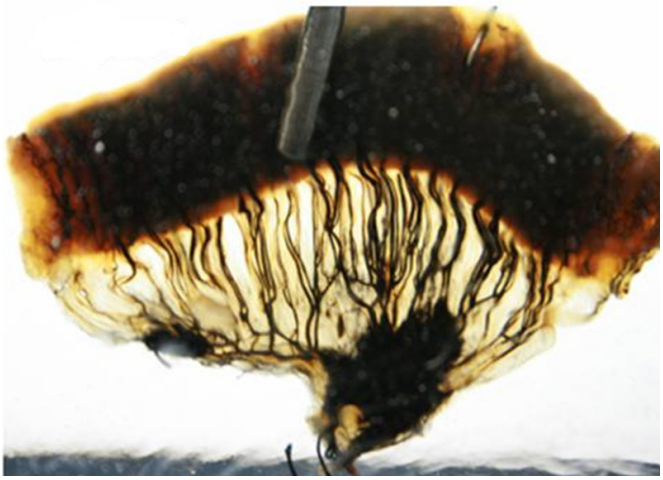

Figure 2: Normal bowel segment displaying normal vascular anatomy.

Figure3: Strictured bowel segment showing evidence of vessel blockade (marked by arrow) at the site of stricture.

Discussion

Crohn's disease is an inflammatory bowel disease which can involve any part of the gastrointestinal tract with transmural inflammation. The pathogenesis of Crohn's induced inflammation is a complex process which still evades comprehension. The involvement of ischaemia as a primary mechanism operating in Crohn's disease has been proposed by Wakefield et al[10], who on the basis of immunohistological evidence associating granulomas with the vasculature, suggested that ischaemic mucosal injury occurs as a consequence of occlusive granulomatous vasculitis.Further studies of the mesenteric vasculature of the strictured part of the bowel by Anthony et al[7], showed block in the small end arteries supplying the mesenteric side of the strictured bowel. Similar ulceration and structuring induced experimentally in rats by indomethacin were also noted to have associated arterial thrombosis. Hence, ischaemia plays an important role on pathogenesis of both NSAID induced strictures and Crohn's strictures.

Gastrointestinal tuberculosis is another granulomatous inflammatory disease where the sequence of pathogenetic events that culminate in ulceration and stricture formation are not known. While the sputogenous theory implicates direct infection of the intestinal mucosa by tubercle bacilli in swallowed sputum,[11] the hematogenous theory proposes a vascular genesis of tuberculosis.[11] Angiographic findings documented by Shah and Ramakantan showed vascular changes in the mesentry of the intestine affected by tuberculosis.[12] Pathological studies of the vessel wall in the small bowel mesentry also showed vasculitis changes incriminating ischemia as a chief cause for the ulceration and stricture associated with tuberculosis.[13,14] End arteritis has been documented in some of the earliest reports describing the pathology of intestinal tuberculosis.[15] Our study has also showed vascular blockade in a significant number of patients with tuberculous stricture, supporting the hypothesis that tuberculous ulcers and strictures are a result of vasculitis resulting in infarction of the intestinal wall.[12-14]

In the studies by Anthony et al[15], they demonstrated two types of vessels supplying the intestine: a) short mesenteric vasa recta that were found to be end arteries and b) large anastomotic vessels at the antimesenteric margin of the small intestine. The predilection of the mesenteric margin and the longitudinal nature of the ulcers were sited to be due to involvement of the shorter mesenteric vessels by Crohn's and indomethacin induced vasculitis. The circumferential nature of the 'napkin' ring strictures caused by tuberculosis which are very discrete with surrounding normal mucosa[16] point towards involvement of the longer anastomotic vessels supplying the entire circumference of the intestine. In our study, the methodology used was similar to that used by the previous studies. However, the distinction between the different types of vessels involved by the different diseases could not be made out because of the smaller sample of Crohn's strictures available for study.

Vascular blockade has also been noted in the 'nonspecific' strictures studied. The vascular blockade not in the strictures described as 'ischemic' after histopathology were found to be similar to those found in tuberculous stricture. Whether these are different forms of the early stages of tuberculous strictures, as has been proposed in the past,[12] requires further studies for clarification.

Conclusion

Small intestinal strictures, especially those caused by tuberculosis, are associated with blocked vessels in the mesentry. Though the temporal cause-effect relationship could not be demonstrated, it can be conceived from this study and with supporting evidence in the literature that ischemia plays a role in the pathogenesis of these strictures.

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