Answer

Nonocclusive Ischemic Colitis

Computed tomography showed concentric bowel wall thickening with inflammatory changes from the midileum to the proximal transverse colon and patent major mesenteric vessels. Colonoscopy showed severe ulcerating inflammation of the distal ileum and proximal colon. A diagnosis of inflammatory bowel disease was made and conservative management was instituted. Failure to respond led to diagnostic laparotomy. This revealed an abruptly narrowed 60-cm segment of distal ileum confluent with the ascending and transverse colon, showing a thickened, woody appearance with nodularity and fibrin deposition on the serosa (**Figure 2**). The major mesenteric arteries were pulsatile. Resection with a primary ileocolic anastomosis was performed. Histological analysis showed mural ischemia and necrosis of the distal ileum and colon with organization and regeneration of the mucosa. Arterioles showed mild medial hypertrophy. Arterial or venous occlusion was not evident. Thrombophilia and vasculitis screen results were negative. A diagnosis of nonocclusive ischemia of distal ileum and proximal colon was made.

Ischemic colitis[1] has a prognosis more favorable than that of its small-bowel counterpart.[2] It is typically a disease of elderly persons and can broadly be divided into arterial or venous and occlusive or nonocclusive types. Arterial-occlusive etiology includes luminal thrombosis on a background of mesenteric atherosclerosis with embolic disease from atrial fibrillation or following subendocardial myocardial infarction.[3,4] Other causes of occlusion include vasculitis, radiation end-arteritis, complications of abdominal aortic aneurysm, aortic dissection, hypercoagulable states, and strangulation of the mesentery.[3,4] Venous occlusion usually occurs with mesenteric venous thrombosis, strangulation, or severe venous stasis.[3] Transmural infarction inevitably occurs in occlusive ischemia. A sharp transition point is seen in arterial occlusion while a vaguer penumbra is seen with venous etiology.[2] Nonocclusive causes include hemodynamic shock, vascular spasm, venous congestion, and luminal distention.[2-4] The degree of hypoperfusion, length of segment affected, severity, and sequelae are variable and dependent on the severity, rapidity, and duration of the insult, resolution, chronicity, collateral circulation, comorbidities, and overall organ function.[2,3] Damage resulting from nonocclusive ischemia may be classified as mucosal, mural, or transmural.[2,3] Presentation typically involves sudden-onset colicky abdominal pain, vomiting, distention, and bloody diarrhea. Signs range from mild tenderness to generalized guarding and shock in the event of perforation and frank peritonitis.

Figure 2. Operative photograph.

Occlusive infarction has a 90% mortality, whereas nonocclusive disease carries a 10% mortality. In a study involving 150 patients with nonocclusive ischemia, 45% had reversible disease, 13% had ischemic stricture, and 19% had gangrene or perforation.[3] Nonocclusive ischemia is seen in cardiogenic, hemorrhagic, or septic shock and can be precipitated by drugs including cocaine, amphetamines, and vasopressors such as noradrenaline and digitalis. It usually affects the watershed territory of colonic perfusion at the splenic flexure and the distal sigmoid colorectal interface.[3] In mural ischemia, sequelae range from complete resolution to chronic ulceration and stricture formation. With transmural ischemia, infarction with perforation, peritonitis, and shock usually occur.[2,3]

The definitive diagnosis is usually made on the basis of macroscopic findings and is confirmed by histological analysis from colonoscopy or laparotomy, including an angiogram in conjunction with the sequence of events leading to the presentation.

Accepted for Publication: January 2, 2009.
Correspondence: Dinesh N. Ratnapala, MB, ChB, Department of Surgery, Redcliffe Hospital, Locked Bag 1, Redcliffe, Queensland 4020, Australia (dineshbrisbane@yahoo.com).
Author Contributions: *Study concept and design:* Ratnapala and Lambrianides. *Acquisition of data:* Ratnapala. *Analysis and interpretation of data:* Ratnapala, Lisle, Munn, and Lambrianides. *Drafting of the manuscript:* Ratnapala and Lambrianides. *Critical revision of the manuscript for important intellectual content:* Ratnapala, Lisle, Munn, and Lambrianides.
Financial Disclosure: None reported.

REFERENCES

1. Marston A, Pheils MT, Thomas ML, Morson BC. Ischaemic colitis. *Gut.* 1966;7(1):1-15.
2. Bailey RW, Bulkley GB, Hamilton SR, Morris JB, Smith GW. Pathogenesis of nonocclusive ischemic colitis. *Ann Surg.* 1986;203(6):590-599.
3. Khan AN, MacDonald S, Chandramohan M, Barker C. Colitis, ischemic. http://emedicine.medscape.com/article/366808-overview. Accessed March 6, 2008.
4. Sreenarasimhaiah J. Diagnosis and management of intestinal ischaemic disorders. *BMJ.* 2003;326(7403):1372-1376.

©2009 American Medical Association. All rights reserved.
Downloaded From: http://archsurg.jamanetwork.com/ on 07/30/2016