

Colonic Obstruction Due to Sigmoid Muscular Hyperplasia

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Abstract

We report a case of acute colonic obstruction initially presumed to be secondary to acute diverticulitis, necessitating emergent surgical intervention. Pathologic examination failed to reveal evidence of inflammation, fibrosis or neoplasia. Marked hypertrophy of the sigmoid circular muscle layer was documented and thought to be the etiology of the colonic obstruction.

Key Words: Colon obstruction, sigmoid colon, muscle hypertrophy, barium enema.

Diverticular disease is endemic in the Western world, affecting approximately one third of the population over the age of sixty years (1). Most often, diverticula remain clinically silent. Diverticular disease becomes clinically evident as lower gastrointestinal bleeding or lower abdominal pain, which may be complicated by perforation and abscess formation. When lower abdominal pain becomes manifest, there is a tendency to attribute this symptom to superimposed inflammation or diverticulitis. In 1963, Morson (2) showed that fully one third of the specimens resected with a clinical and radiological diagnosis of diverticular disease failed to show any evidence of inflammation. Diverticular disease is primarily a condition of disordered muscle function, the clinical importance of which is often underestimated. In fact, until the mid-1970s, surgical sigmoid myotomy was not uncommonly performed in patients with complicated and uncomplicated diverticular disease, with varied results (3, 4).

Not uncommonly, acute inflammation of the sigmoid colon manifests itself with colonic obstruction; it is usually partial in nature and related to the accompanying inflammation and edema. Complete bowel obstruction due to diverticular disease, on the other hand, is rare. Where complete obstruction occurs in diverticular disease, it is most frequently the result of complicating perforation, infection with abscess formation, edema and spasm (5). We present a rare case of complete colonic obstruction secondary to sigmoid muscular hyperplasia in the absence of inflammatory changes — a condition which necessitated emergent surgical intervention.

Case Report

A 76-year-old man with a history of schizophrenia was admitted to the hospital for treatment of pneumonia. At the time of admission, he was noted to have mild abdominal distension. A reliable bowel habit history could not be obtained from him. With the institution of intravenous antibiotics, his pneumonia resolved. However, his abdominal distension progressed over the ensuing five days, with development of obstipation. Acute bowel obstruction was suspected.

Radiographic examination of the abdomen revealed an enormously distended colon extending to the left iliac fossa in the erect position. No small bowel dilation was noted. A barium enema confirmed an obstruction in the sigmoid colon. Scattered diverticula were identified in the region of the obstruction (**Fig. 1**). No extravasation of contrast material was seen. The impression was that of acute colonic obstruction secondary to diverticulitis. It was felt that an underlying colonic neoplasm could not be excluded with certainty.



Fig. 1. Obstruction to the retrograde flow of barium in the sigmoid colon. Several scattered diverticula (arrowheads) can be seen. The adjacent descending colon (*) is markedly distended with a small amount of barium.

At laparotomy, a firm mass was palpated in the sigmoid colon. To decompress the massively distended bowel, a diverting transverse colostomy was performed. A follow-up barium enema via both the rectum and transverse colostomy again demonstrated the obstructed sigmoid colon. The unfilled segment of colon measured 8.0 cm in length. Following sigmoid resection, the patient made an uneventful recovery.

Pathological examination of the specimen revealed marked stenosis of the sigmoid lumen (1.2 cm in diameter) and a grossly thickened bowel wall (1.7 cm in width). The stenosed segment (arrow) contained three large diverticula (**Fig. 2**). The colon proximal to the stenosed segment was greatly dilated, measuring 13.0 cm in circumference and 0.5 cm in thickness.

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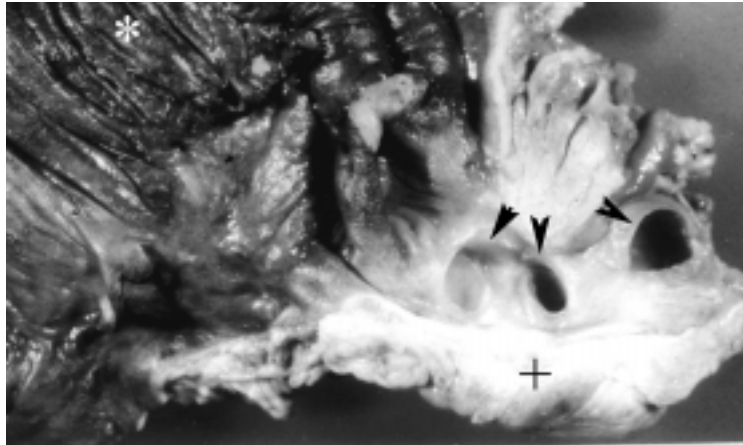


Fig. 2. Resected sigmoid colon demonstrating a markedly thickened wall (+) due to muscle hypertrophy. There is no sign of inflammatory change. Three wide-mouthed diverticula are visualized (arrowheads). The proximal colon (*) is markedly dilated.

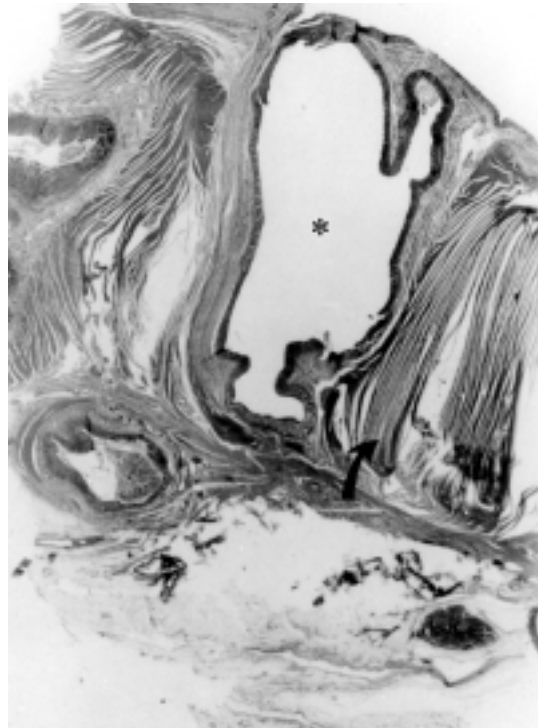


Fig. 3. Histology of sigmoid stricture: Enormous thickening of smooth muscle in the wall (curved black arrow). A large diverticulum (*) is visualized. There is no inflammation.

Microscopic examination showed no significant inflammation within or adjacent to the diverticula (**Fig. 3**). The inner circular muscle layer of the external muscularis was markedly hypertrophied, accounting for the very unusual thickening of the wall seen with the naked eye. On gross examination, the mucosa appeared atrophic. Microscopic examination of the dilated segment showed healed colitis, possibly secondary to ischemia. No fibrosis of the wall of the stenosed segment was seen and no evidence of neoplasia was documented.

Discussion

In 1952, Celio (6) described the abnormal colonic involvement in specimens of patients with diverticulitis and drew comparisons between this myopathy and that observed in infants with hypertrophic pyloric stenosis. Following these initial observations, Morson (2) reported on four specimens of sigmoid colon removed for presumed diverticulitis, which showed abnormal muscle thickness of the sigmoid wall without any detectable diverticula or inflammation. The muscle abnormality appeared strikingly similar to that seen in patients with diverticulosis. Based on these findings, he suggested that the muscle thickening may be a predisposing factor in the development of diverticula. Cassano and Torsoli (7) first reported two cases of idiopathic muscular strictures of the sigmoid colon in humans, which bore striking similarities to the pathological picture seen in patients with diverticular disease.

The muscle abnormality of diverticular disease is characteristic (5). It is not seen in other inflammatory disease of the colon, whether segmental or diffuse. The underlying abnormality is shortening of the involved segment, most often the sigmoid. This results in large part from spasm and increased thickness of the taenia coli. The shortening explains several of the histological features which have been noted. These include thickening of both circular and longitudinal muscles without evidence of hypertrophy or hyperplasia, redundant mucosal folds, excess neural and vascular elements, and increased serosa and pericolic fat (8, 9). The belief that the muscle abnormality was central to the development of symptomatic diverticulitis led a number of authors in the 1960s and 1970s to propose sigmoid myotomy as a potential solution, and a number of patients underwent this procedure with symptomatic improvement (3, 4, 10).

Morson and Whiteway (1) described the thickened taenia coli as unusually firm and almost cartilaginous in consistency. They found that the circular muscle, which possesses an accordion-like appearance, was “so thick that it appeared as if the contractions had either fused or become calcified.”

While some degree of colonic obstruction is observed in about two thirds of patients with acute diverticulitis, it is usually partial in nature and related to the inflammation, spasm and edema that commonly accompany this condition. Complete bowel obstruction due to diverticular disease is very seldom seen and rarely requires emergency surgery (5). It is usually the result of abscess formation with encroachment of the colonic lumen, or repeated episodes of diverticulitis with subsequent fibrosis and stenosis (11). In some instances, the clinical presentation is due to small bowel adherence to the inflammatory process rather than large bowel obstruction (5).

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It is rare for muscle thickening to be so severe as to lead to large bowel obstruction, a occurred in our patient, with no evidence of acute inflammation, ischemia, fibrosis or neoplasm present. It would seem likely that the patient's psychiatric illness was responsible for the delay in seeking medical treatment and permitted aggravation and progression of the muscle abnormality in the sigmoid. Then, when acute large bowel obstruction occurred, medical attention became a matter of urgency.

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