Nonsteroidal Antiinflammatory Drug-Induced Colonic Stricture
An Unusual Cause of Large Bowel Obstruction and Perforation

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Over 100 million prescriptions for nonsteroidal anti-inflammatory drugs (NSAIDs) are written annually in the United States (1) and more than 20 million in the U.K. (2). Gastroduodenal mucosal injury is a widely recognized side effect, up to 22% of patients on NSAID treatment having peptic ulceration (2). More recently, inflammation and ulceration affecting both small and large intestine have been recognized as adverse effects (3–5). Of particular interest have been the reports of strictures, both broad and "diaphragmlike," involving jejunum and ileum associated with the long-term usage of these drugs (6–8). A similar NSAID-induced colonic stricture was first reported in 1989 (9). Subsequent reports of colonic diaphragms have usually been in patients being investigated for iron-deficiency anemia (10–15). Emergency presentation with large bowel obstruction and perforation secondary to an NSAID-induced colonic stricture is previously unreported. We present such a case and review the literature.

CASE REPORT

A 53-year old white female with a seven-year history of rheumatoid arthritis was admitted as an emergency. She complained of a three-day history of lower abdominal pain, suddenly becoming severe 3 hr before admission with radiation to the right shoulder. She was taking diclofenac (slow release), aspirin, ranitidine, and coproxamol. Diclofenac had been regularly prescribed for the last five years.

She was distressed, pale, and sweaty. Pulse was 78 beats per minute, blood pressure 78/50, and temperature 36.8°C. Her abdomen was rigid and bowel sounds scanty. Serum amylase was normal and erect chest x-ray revealed free gas under the right hemidiaphragm. Following fluid resuscitation, surgical exploration was undertaken.

At laparotomy, she was found to have a 1-cm perforation in a grossly distended and thinned cecum. The cause of obstruction was a tight stricture of the ascending colon, but the remaining small and large bowel appeared normal. A right hemicolectomy was performed. Pathologic evaluation of the resected specimen showed two separate similar areas of circumferential mucosal ulceration with stenosis secondary to extensive submucosal fibrosis and hypertrophy of the muscularis propria; prominent lymphoid follicles were present through the bowel wall (Figures 1a,b and 2). One stricture was located in the ascending colon and the other at the ileocaecal valve involving the root of the appendix. Between these strictures the cecum was distended and thinned, leading to perforation. The remaining resected small and large bowel was grossly and microscopically normal. There was not evidence of Crohn’s disease and the mesenteric blood vessels were unremarkable without evidence of occlusion, vasculitis or amyloid.

The patient made an uneventful recovery.

DISCUSSION

The most widely recognized complications of NSAID therapy are the consequences of gastroduodenal inflammation and ulceration—pain, bleeding, and perforation. The first report of NSAID damage in the more distal gastrointestinal tract was in 1966, cecal ulceration occurring in association with oxyphenbutazone and indomethacin therapy (3). In a comprehensive review of the literature, Bjarnason et al (5) summarize a number of adverse effects of
ingested NSAIDs on the small and large intestine, but do not include colonic stricture and obstruction. Small intestinal stricture associated with NSAID ingestion was described in 1973 (6), but obstruction due to "diaphragm disease" (multiple, 2- to 4-mm-thick concentric diaphragmlike septa that narrow the lumen to a pinhole) was not recognized until 1987 (7). This unusual condition was identified in five separate cases by Lang et al (8) in a retrospective review of 576 small bowel resections for obstruction over a 16-year period. In this series, they also recognized broad-based stenoses associated with NSAID ingestion. They hypothesized an evolution of pathological changes in which tall, thin

Fig 1. (a) Photograph of the opened resected specimen demonstrating obstructing stenoses proximal and distal to the perforated caecum, and (b) line drawing.