Nonsteroidal-Induced Benign Strictures of the Caecum: A Case Report And Review Of The Literature

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Citation

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Abstract

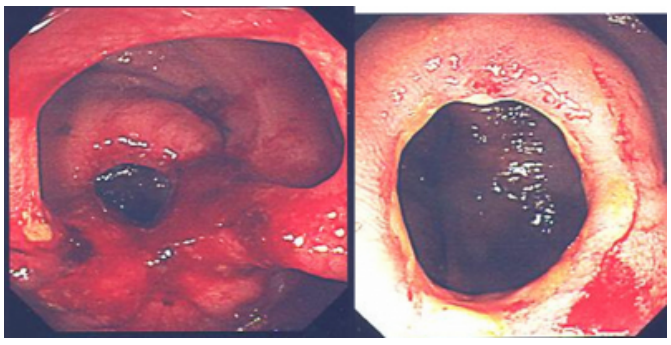
A rare complication seen with chronic use of sustained-release non-steroidal anti-inflammatory drugs (NSAIDs) is formation of bowel strictures and diaphragm-like disease with characteristic histological appearance of submucosal fibrosis. They are thought to result from alterations in enterocyte homeostasis. The strictures generally occur in the terminal ileum and right side of the colon with the symptoms of occult blood loss, change in bowel habits, weight loss, obstruction and rarely perforation. We report the case of a women diagnosed with caecal strictures who was taking slow-release diclofenac tablets for osteoarthritis.

CASE REPORT

A 70-year-old female referred to the outpatient clinic with 5 month history of change in bowel habits, weight loss, lethargy, iron deficiency anaemia and possible mass in the right iliac fossa. She had been taking diclofenac retard tablets for many years for her osteoarthritis. She was admitted for blood transfusion and started on iron therapy, and diclofenac was stopped. Gastroscopy was normal and CT colonoscopy showed possible Crohn's disease or malignant stricture in the caecum. Colonoscopy showed two strictures in the caecum (fig.) and the biopsies showed active chronic colitis.

Figure 1

Figure 1: Caecal strictures seen in colonoscopy.



In view of the uncertainty, she underwent right hemicolectomy with end to end anastamosis. She had uneventful recovery and remains well. Histology shows a circular stenosis in the caecal lumen measuring approximately 10 mm, the mucosa is otherwise normal. But there is thickening of the muscularis mucosa and submucosal fibrosis consistent with diaphragm disease due to NSAIDs with no evidence of malignancy identified.

DISCUSSION

Side effects of NSAIDs on the upper gastrointestinal tract are well recognised. Less well known are the lesions of the lower gastrointestinal tract associated with their chronic use. Strictures and diaphragm-like disease in the proximal colon have been identified as rare complication of chronic use of some NSAIDs [1] . Diaphragm disease of the colon was first reported in 1989 [2] . There are only few case reports of diaphragm-like disease of colon in comparison to a large number of reported cases affecting the small bowel [4] . The exact pathophysiology of colonic stricture is not clear. Nearly all documented cases have had iron deficiency anaemia. Patients presenting with these stricture have typically been elderly with chronic rheumatoid or osteo-arthritis and symptoms of non-specific abdominal pain or change in bowel habits [1,6] . One case initially presented as perforation [7] . The strictures can be difficult to differentiate macroscopically from cancer, diverticular disease and Crohn's disease [16] .

The effects on the small and large bowel are thought to result from local phenomena related to prolonged contact of NSAIDs with bowel mucosa [11] . Localisation of inflammation in the distal ileum and proximal colon may be attributable to the hold-up of gastrointestinal contents there by the ileocaecal valve [6] . However, Whittle [8] showed

mucosal effects in the small bowel with rectally administered preparations supporting a systemic postulate. NSAIDs provide analgesia by inhibiting cyclo-oxygenase (COX) 2. However, conventional NSAIDs also inhibit COX-1 which forms the cytoprotective prostaglandins (PGE, PGI). Another report proposed that enterocyte damage results from an uncoupling of oxidative phosphorylation in mucosal mitochondria with resulting depletion of adenosine triphosphate stores [3], leading to increased mucosal permeability and efflux of calcium ions. Injured enterocytes are also prevented from converting arachidonic acid into prostaglandins due to inhibited cyclooxygenase, making them susceptible to further damage by bile acids, enzymes and bacteria [5]. Mucosal invasion by resident bacteria release chemotactic factors that recruit neutrophils into the submucosa with inflammatory sequelae. Progression of submucosal fibrotic lesions has been proposed to proceed from mucosal diaphragms resembling normal plicae circulares with fibrosis at their luminal margins to broad based stenoses that become dome-shaped and grossly fibrotic over time [12].

Colonic strictures have been described in children with cystic fibrosis taking methacrylic acid copolymer-coated pancreatic enzyme supplements [9], patients taking potassium supplementation [3] and also in those with secondary vasculitis in SLE and rheumatoid arthritis [10].

Treatment of colonic stricture includes endoscopic balloon dilatation with adjuvant prednisolone to promote healing and prevent restructuring [16]. Resistant cases need segmental resection and anastamosis. Discontinuation of all NSAIDs is warranted, but may be problematic in chronic arthritis patients. Studies on a limited number of patients have demonstrated the efficacy of sulfasalazine [13], metronidazole [15] and misoprostol [14] in mitigating or preventing NSAID-induced enteropathy. It will be interesting to review the effect of selective COX-2 inhibitors on the incidence of colonic diaphragm disease with the passage of time.

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