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Figure 1
Duodenal Diaphragm Disease



Figure 2
Hepatic Flexure Diaphragm Disease

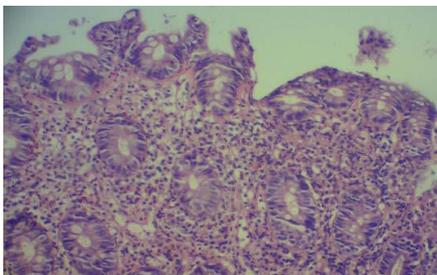


Figure 3
Nikon 10X Objective
Non specific active colitis

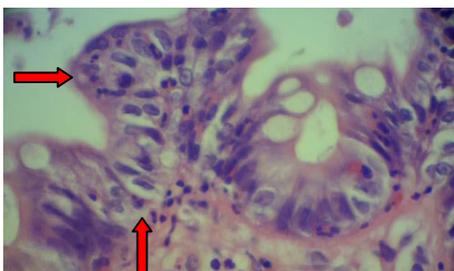


Figure 4
(Nikon 40X Objective)
Inflamed colonic epithelium (arrowheads)

Case 1; Clinical History:

80 year old male patient with hx of diabetes mellitus, HBP, and osteoarthritis who presented with a three month history of upper abdominal pain associated with occasional episodes of vomiting, especially after food intake. The patient referred daily ibuprofen usage for at least six months.

Endoscopic Findings: Upper endoscopy revealed circumferential ring-like luminal narrowing with associated edema and erythema at the duodenal sweep precluding further advancement of the endoscope. (Figure 1).

Case 2; Clinical History:

49 year old woman with hx of bronchial asthma, HBP, Major Depression, osteoarthritis and smoker of 1 pack/day who underwent a colonoscopy after an episode of enteritis/colitis.

Endoscopic Findings: Colonoscopy revealed luminal narrowing similar to a ring with associated erythema and ulcers in the hepatic flexure precluding further advance of the scope. (Figure 2)

Discussion:

“Diaphragm disease” strictures are considered uncommon but very characteristic manifestations of NSAID induced enteropathy and colopathy. Their presenting symptomatology includes abdominal pain, GI bleeding, weight loss, subacute obstruction and diarrhea.

The jejunum and ileum are the preferred sites for small intestinal diaphragm disease. Radiologic contrast and imaging studies are inaccurate and not sensitive enough for the diagnosis. The advent of video capsule endoscopy has resulted in an increased diagnostic frequency of these lesions. Some authors have predicted that the use of enteric coated and sustained release NSAID’s will probably increase the frequency of large bowel “diaphragm disease”.

“Diaphragm Disease” strictures are characterized by thin web-like septa that lead to circumferential narrowing of the lumen (Figures 1 and 2). Microscopically, the mucosa of these septa is mildly inflamed and the submucosa reveals fibrosis. The pathogenesis of these strictures is thought to be the result of prolonged exposure to NSAID’s with repeated non specific mucosal injury (either enteritis or colitis) and subsequent reparative submucosal fibrosis (“scarring”). The histopathology of these NSAID induced putative precursor inflammatory lesions include focal active superficial enteritis/colitis (Figures 3-4) and chronic nonspecific ulcers.