

# An Infrequent Cause of Intestinal Subocclusion in a Patient With Rheumatoid Arthritis

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### Clinical Case

A 76 year-old female presenting recurrent intestinal subocclusion for 2 months was consulted. She was being treated with non-steroidal anti-inflammatory drugs (NSAID) due to long-term rheumatoid arthritis. The abdomen was diffusely painful, with diminished peristalsis. Laboratory analysis and a complete digestive study with an abdominal x-ray, fibrogastroscopy, fibrocolonoscopy, echography, and computerized tomography were normal. The esophagogastrointestinal transit study showed a small bowel stenosis (Figure 1).

### Evolution

Exploratory laparotomy confirmed a distal jejunal stenosis and a partial resection was carried out. The anatomopathological study showed a luminal stenosis, mucosal ulceration, concentric fibrosis of the submucosa, an important inflammatory infiltrate, and abundant fibrin, all of which was characteristic of a diaphragm stenosis (Figure 2). No amyloid was found.

### Diagnosis

Diaphragm stenosis of the distal jejunum secondary to NSAID therapy.

**Figure 1.** *sophagogastrointestinal barium transit: distal jejunal stenosis (arrow).*

### Discussion

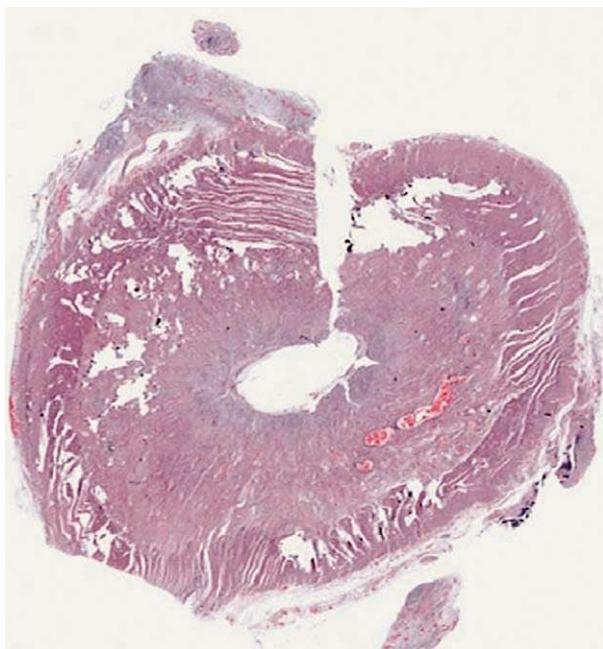
The prevalence of gastropathy due to NSAID is elevated, but this enteropathy can affect any portion of the gastrointestinal tract.<sup>1</sup> The complications described have been anemia due to intestinal hemorrhage malabsorption syndromes, ileal dysfunction and diaphragm luminal stenosis, a lesion that is pathognomonic of NSAID therapy. The elevated intraluminal concentration of the drug increases the intestinal mucosa permeability and after a neutrophilic infiltrate, an ulcer is formed and ultimately

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**Figure 2.** Transverse section of the distal jejunum seen by optical microscopy; partial concentric (diaphragm) occlusion of the intestinal lumen.

perforated, with its healing originating a diaphragm stenosis.<sup>2</sup> NSAID drugs with enterohepatic circulation, especially diclofenac, have more intestinal toxicity, which is not the case of aspirin because its absorption is mainly gastroduodenal. Coxibs could have less toxicity due to the

protective function of the prostaglandins.<sup>3</sup> A 60%-70% of patients undergoing chronic treatment with NSAID develop asymptomatic enteropathy<sup>4</sup> as well as 70% of healthy volunteers after 2 weeks of treatment with diclofenac.<sup>5</sup> This complication must be suspected in every patient treated with NSAID in a chronic manner that presents with diarrhea, abdominal pain with sub occlusive symptoms, weight loss, iron deficit anemia or malabsorption symptoms. The best diagnostic test is esophagogastrointestinal transit, though recently videocapsule technique has been employed.<sup>6</sup> Treatment consists in intestinal resection. NSAID enteropathy can pass undetected by the clinician because it can have an asymptomatic course.

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