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Nonsteroidal anti-inflammatory drug-induced enteropathy, presenting with multiple small bowel strictures: A case report

Layla Lombard, Adam Boutall, Dominic Mutura, Nsizwenye Mkhwanazi

ABSTRACT

Introduction: Nonsteroidal anti-inflammatory drugs are used commonly and are widely available, they have a significant association with gastrointestinal system morbidity including small bowel enteropathy that can lead to small bowel strictures.

Case Report: We present a case of severe small bowel enteropathy in a 51-year-old woman with a history of multiple small bowel resections and a delayed diagnosis. She was admitted to our unit following a prolonged course of symptoms, multiple bowel resections, and an unclear underlying etiology. She underwent a laparotomy and on table enteroscopy revealing multiple diaphragm-like strictures. Balloon dilatation was done on amenable strictures and a bowel resection for the severe segment. A background history of nonsteroidal anti-inflammatory drugs (NSAIDs) was elicited with strong advise to discontinue, and a diagnosis of NSAID-induced small bowel enteropathy was made. She was subsequently discharged nutritionally well.

Conclusion: Nonsteroidal anti-inflammatory drugs-induced small bowel enteropathy represents a diagnostically challenging condition requiring a high index of suspicion. Advances in diagnostic modalities, including video capsule endoscopy and double-balloon enteroscopy, have significantly improved detection.

Keywords: Enteropathy, NSAIDS, Small bowel, Strictures

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INTRODUCTION

Nonsteroidal anti-inflammatory drugs (NSAIDs) have analgesic, antipyretic, and anti-inflammatory properties and are available over the counter for multiple clinical indications [1]. Their use is associated with several risks, one of which is NSAID-induced enteropathy [1]. Historically, intestinal mucosal injury was thought to affect only the proximal segments of the gastrointestinal tract (GIT). This was largely due to limitations in investigative procedures capable of accessing the mid-GIT. With the widespread use of capsule endoscopy and double-balloon enteroscopy, this entity has gained increased recognition. It is also postulated that the use of enteric-coated, sustained-release, or slow-release NSAIDs may have shifted mucosal injury from proximal regions of the GIT toward the distal small intestine, contributing to the rising incidence of small bowel enteropathy [1–3]. The mucosal injury can result in strictures frequently

leading to obstruction requiring surgical intervention. The pathophysiological mechanisms linking NSAID use to stricture formation remains unclear, though direct mucosal injury and altered intestinal motility are thought to play a role [4].

CASE REPORT

A 51-year-old female presented with a 3-year history of progressive, intermittent, and cramping abdominal pain. This was associated with non-bloody diarrhea, nausea, and intermittent bilious vomiting. She had no known chronic medical conditions. Her surgical history included a right ovarian cystectomy 16 years prior for a benign indication. She was a non-smoker with no history of alcohol or illicit drug use.

The initial management at a primary care facility was conservative; however, the patient's symptoms persisted. A gastroduodenoscopy and colonoscopy did not yield contributory findings. She later presented with severe abdominal pain and features of small bowel obstruction, necessitating emergency surgery, during which a bowel resection with primary anastomosis was performed.

Postoperatively, her symptoms persisted, and she underwent a re-look laparotomy with further bowel resection. She initially improved following this intervention; however, histological examination of resected specimens was non-diagnostic. She subsequently re-presented with similar symptoms and was referred to the intestinal failure unit, where she was assessed and optimized for surgery. Intraoperatively, the small bowel appeared macroscopically normal. Intraoperatively, the small bowel appeared macroscopically normal. Push enteroscopy demonstrated multiple diaphragm-like strictures (Figure 1). Balloon dilatation was performed on amenable strictures (Figure 2). However, the more severe and closely spaced strictures were not amenable to balloon dilatation, necessitating resection of the affected bowel segment.

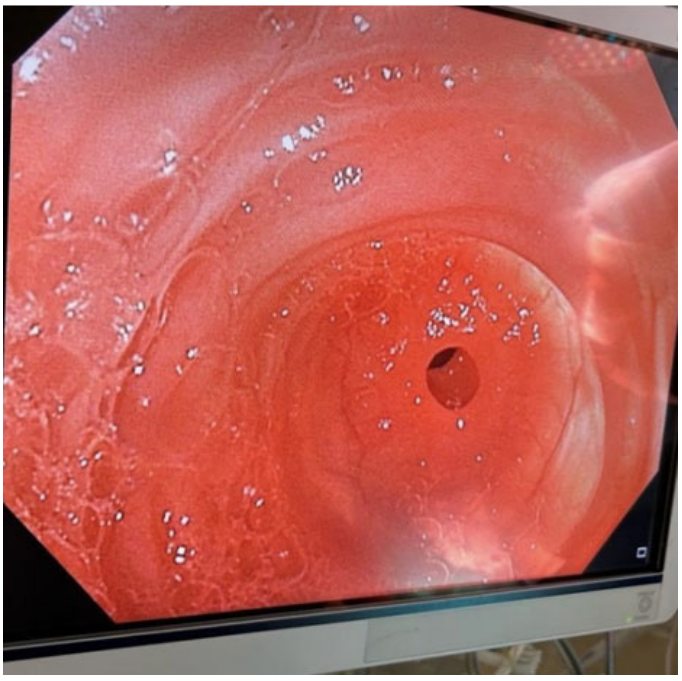


Figure 1: Endoscopic picture of small bowel stricture.

Histological analysis revealed >20 strictures, with no evidence of Crohn's disease or intestinal tuberculosis. A further probe into her history revealed a significant intake of NSAIDS, we made a diagnosis of stricturing NSAID-associated small bowel enteropathy, and the patient was advised to permanently discontinue NSAID use. She was subsequently discharged in good health and remained nutritionally independent.

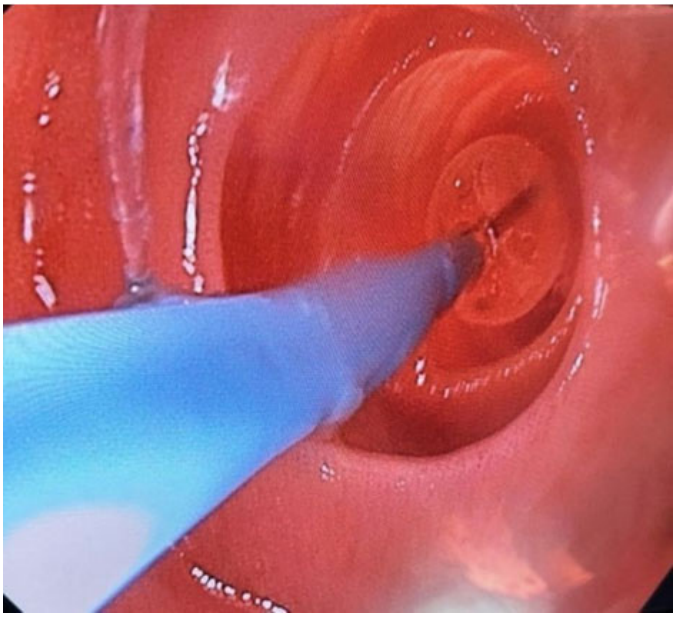


Figure 2: Balloon dilatation of strictures is demonstrated.

DISCUSSION

This case demonstrates a severe and diffuse manifestation of NSAID-induced enteropathy, with an exceptionally high density of strictures. While this condition is increasingly recognized, this report is notable for both the severity and the number of strictures within a confined segment. The delay in diagnosis is attributed to an overlap in presentation with other small bowel conditions causing strictures. Cross-sectional imaging was repeatedly inconclusive, with definitive diagnosis only achieved following surgical resection.

Small bowel ulcers have been reported in 8.4% of NSAID users compared with 0.6% of non-NSAID users [3]. Short-term use of diclofenac and low-dose aspirin over a two-week period demonstrated macroscopic small bowel injury in 68% and 80% of participants, respectively, predominantly affecting the distal small bowel and colon, most commonly the ileocecal region [3]. Increased intestinal permeability and small bowel inflammation has been reported to develop within 12 hours and 10 days of NSAID use, respectively [3].

The precise mechanisms underlying NSAID-associated small bowel injury remain incompletely understood. Injury is thought to result from direct damage to cell membrane phospholipids, leading to mitochondrial dysfunction, reduced adenosine triphosphate (ATP) production, increased intracellular calcium release, and free radical formation [3]. These processes impair intercellular integrity and increase mucosal permeability, allowing luminal contents to penetrate the epithelial barrier and trigger secondary inflammation via neutrophil activation [3].

Such luminal factors include gram-negative bacilli within the normal GIT flora, which contain lipopolysaccharide (LPS) [3]. Lipopolysaccharide activates toll-like receptors, amplifying the inflammatory cascade and contributing to small bowel injury [3]. Re-exposure of the small bowel to NSAIDs via enterohepatic circulation further exacerbates mucosal damage [3]. Nonsteroidal anti-inflammatory drugs also inhibit cyclooxygenase (COX) enzymes 1 and 2 [1, 3]. Cyclooxygenase enzyme 1 inhibition results in GIT injury through reduced mucosal blood flow and increased intestinal permeability [1, 3]. Cyclooxygenase enzyme 2, which is induced during inflammation, plays a role in mucosal healing and immunomodulation; thus, its inhibition may also contribute to gastrointestinal injury [1, 3]. Most NSAIDs are non-selective inhibitors of both COX-1 and COX-2 [1]. Selective COX-2 inhibitors, however, provide anti-inflammatory effects with a reduced risk of gastrointestinal complications, thereby preserving GIT integrity [1].

The diagnosis of NSAID-induced small bowel enteropathy is challenging and frequently delayed, as illustrated by this case. A high index of suspicion and targeted questioning regarding NSAID use are essential. Clinical manifestations vary widely and include gastrointestinal bleeding, protein-losing enteropathy, and stricture formation [4]. Obstruction occurs in approximately 17% of affected patients [3]. Our patient presented with nonspecific abdominal pains with the diagnosis only cliched after complications necessitating surgery.

Diagnostic evaluation incorporates clinical assessment and adjunctive investigations. Non-invasive tests, such as faecal calprotectin and Chromium-51-labeled ethylenediaminetetraacetic acid (51Cr-EDTA) permeability assays, may detect early mucosal inflammation and barrier dysfunction but lack disease specificity [3]. In none complicated cases capsule endoscopy remains the diagnostic modality of choice identifying mucosal injury in up to 70% of long-term NSAID users and visualizing lesions ranging from petechiae and erosions to circumferential ulcers and characteristic diaphragm-like strictures [3]. Computed tomography and magnetic resonance (MR) enterography allow for multiplanar, multiphase imaging and stricture characteristics can be better defined [5]. When histological confirmation or therapeutic intervention is required, double-balloon enteroscopy offers both diagnostic and therapeutic advantages [3]. The management of diaphragm stricturing disease is dependent on severity and the potential for small bowel length loss. Limited strictures causing obstruction are best managed with resection. In patients with multiple strictures at risk for short bowel with massive resections, balloon dilation when feasible is an option as demonstrated in our case [6]. Recurrence of stricture when NSAIDS are stopped is rare [7], correct diagnosis is therefore important because continued use is a key factor for continued mucosal injury. Our patient made a good recovery and has not required further resections.

Important mimickers include multi-focal Crohn's disease, radiation induced-strictures and adhesions and require exclusion [5].

Similarly, cryptogenic multifocal ulcerative stenosing enteritis (CMUSE) is a rare small bowel disorder characterized by recurrent ulceration and multifocal strictures [8]. Diagnosis can be challenging due to overlapping clinical and radiological features with Crohn's disease and nonsteroidal anti-inflammatory drug-induced enteropathy [8]. In the absence of a history of NSAID use, findings consistent with this pattern may suggest CMUSE.

CONCLUSION

Nonsteroidal anti-inflammatory drug-induced small bowel enteropathy represents a diagnostically challenging condition requiring a high index of suspicion. Advances in diagnostic modalities, including video capsule endoscopy and double-balloon enteroscopy, have significantly improved detection. This case contributes to the existing literature by demonstrating an unusually severe presentation characterized by multiple strictures not amenable to endoscopic balloon dilatation.

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Author Contributions

Layla Lombard – Conception of the work, Design of the work, Acquisition of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Adam Boutall – Conception of the work, Design of the work, Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Dominic Mutura – Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Nsizwenye Mkhwanazi – Design of the work, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission
The corresponding author is the guarantor of submission.

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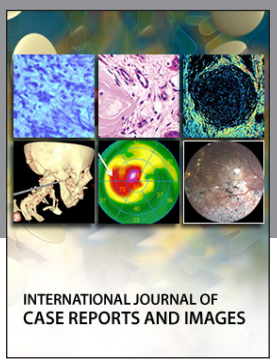
Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.

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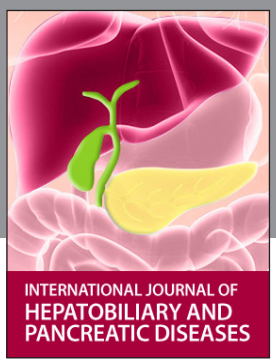
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