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International Research Journal Of Clinical Studies And Pharma Trends

INTERNATIONAL
RESEARCH JOURNAL
OF CLINICAL
STUDIES AND
PHARMA TRENDS

NSAID Induced Ischemic Colitis: A Case Report

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ARTICLE INFO

Article history:

Received 27 September 2021

Revised 15 October 2021

Accepted 09 November 2021

Published 20 November 2021

Keywords:

NSAID

Ischemic Colitis

Peri-colonic inflammation

Early Detection.

ABSTRACT

Non-steroidal anti-inflammatory medication (NSAID)-induced Ischemic colitis is an underappreciated but potentially deadly illness that results from direct damage on the gut mucosa. Plain abdominal radiographs and multi-detector computed tomography reveal right-sided acute colitis with associated peri-colonic inflammation, progressively diminished changes along the descending and sigmoid colon, and rectal sparing, all of which are consistent with the NSAID colitis pathogenesis hypothesis. Through prevention and early detection, increased knowledge of this disorder should minimize morbidity. Consistent instrumental results, negative biochemistry, and stool studies, together with high clinical suspicion and adequate patient questioning, can assist clinicians avoid missing this critical diagnosis.

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INTRODUCTION

Ischemic colitis (IC) is a condition in which the colon's blood circulation is impaired (ischemia, from the Greek word iskhaimos, which means 'blood halting'). Mucosal inflammation occurs when the blood supply (however temporarily) is insufficient to fulfill the metabolic demands of the colon, resulting in ulceration and bleeding. The production of reactive oxygen species and inflammatory cytokines during the regeneration of healthy tissue oxygenation leads to inflammation, which may cause more harm than the immediate consequences of ischemia. Tissue injury is also caused by bacterial translocation, intestinal vasospasm, and intestinal dysbiosis (due to change of the gut micro biota) [2]. Colonic ischemia is distributed from the top down. The mucosa, which is the colon's most metabolically active layer, is the first to be damaged. Sub mucosal bleeding and (finally) transmural necrosis accompany sloughing of villous tips and mucosal edema. Gastrointestinal discomfort, diarrhea, melaena, and rectal bleeding are some of the clinical symptoms, which vary depending on the site and severity of the ischemia injury. The condition ranged from self-limiting to requiring emergency surgical excision within days.

It's important to distinguish IC from mesenteric ischemia. Acute mesenteric ischemia (AMI) is a condition in which a section of the colon loses all blood flow, resulting in fast necrosis and the need for an emergency laparotomy. Acute thromboembolic arterial blockage (typically of the superior mesenteric artery (SMA)) is the most common cause. Non-occlusive arterial AMI can also happen (usually in the context of severe illness and hemodynamic compromise); less often, it can be caused by mesenteric venous thrombosis (which can occur in the context of chronic pancreatitis or portal hypertension)

[3]. Chronic mesenteric ischemia ('mesenteric angina') involves intermittent, cramps, postprandial abdominal pain, typically within an hour of oral intake, over a period of at least 3 months. IC is a moderately frequent clinical illness (incidence of 22.9/100 000 person-years) [4], with significant clinical variation in treatment. There are significant differences in the specialties of doctors who treat individuals with IC. Surgeons handle some situations, while doctors handle others (gastroenterologists).

Over 60 years of age, atherosclerosis, smoking, chronic kidney disease (CKD), and atrial fibrillation have all been linked to an increased risk of IC [5, 6]. Non-steroidal anti-inflammatory medicines (NSAIDs) and estrogen treatment [7] are two medications that raise the risk. Of course, these risk variables interact with one another. Diabetes mellitus, anemia, and hypertension are all more common in CKD patients [6]. They also experience changes in vascular elasticity, and hemodialysis can result in the formation of micro thrombus [5]. Thromboembolic, hemodynamic insufficiency (typically in the context of a contributing condition), iatrogenic, and drug-induced IC are the four types of IC. Atrial fibrillation, prothrombotic diseases such anti-phospholipids syndrome (which causes both arterial and venous thromboembolism), and concomitant cancer are all thromboembolic causes. Hemodynamic insufficiency (a 'supply and demand' issue) occurs in heart failure, severe anemia, hypovolaemia, and septic shock; atherosclerosis (which causes vascular constriction) is a risk factor. Iatrogenic IC can occur as a result of cross-clamping of the aorta or sacrifice of the inferior mesenteric artery (IMA) due to its position in the aneurismal sac during open abdominal aortic aneurysm repair. Micro emboli caused by aortic plaque breakup during endovascular repair can also cause it [8]. Chemotherapeutic agents, vasopressors, estrogen treatment, cocaine, amphetamines, ergotamine, antipsychotics, and NSAIDs are only a few of the medicines that might induce IC [9, 10]. When obtaining a history from individuals with suspicious IC, these agents should be expressly eliminated.

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IC is a clinical spectrum. Depending on the anatomical location and intensity of the colitis, the clinical pattern varies. The most common symptoms (in order of prevalence) are abdominal pain (87 percent), rectal bleeding (84 percent), and diarrhea (56 percent) [11]. PR bleeding is more common in left-sided colitis and typically absent in isolated right-sided colitis, when pain predominates. The most typical sign of bleeding is fresh red blood PR, especially when it is combined with distal colitis. Melaena can be caused by more proximal colonic involvement. Around 75% of people with IC have problems with their left colon, and about 25% have problems with their spleen flexure. Isolated right colon ischemia (IRCI) affects around 10% of the population [1]. On examination, there may be mild to severe discomfort, although there is usually no extensive peritoneum. Fever is uncommon, but when it does occur, it may signal a violation [12].

CASE REPORT

The mechanisms underlying NSAID-induced colitis remain unknown. Its pathogenesis is likely multifactorial, with inhibition of cyclooxygenase and prostaglandin synthesis, as well as impairment of oxidative phosphorylation. The fact that intestinal changes are mostly limited to the right hemicolon, with typical rectal sparing, suggests a role for direct drug toxicity on the intestinal wall. Because most NSAIDs circulate via enterohepatic routes, the proximal colon is directly exposed to intact drug after bacterial breakdown in the distal ileum, and the cecum serves as a reservoir. Furthermore, the use of enteric-coated and slow release preparations allows more drugs to reach the colon [16].

She was afebrile, hemodynamically stable, and not in any distress during her initial examination in the emergency department. The physical exam, including the abdominal exam, was unremarkable. Her laboratory tests revealed no leukocytosis, but her CRP was elevated to 55 mg/L. A CT scan of the abdomen with intravenous contrast (Fig. 01) revealed focal mucosal thickening in the cecum, which raised the possibility of colonic cancer. Following that, a colonoscopy revealed a frond-like/villous, infiltrative, ulcerated, large partially obstructing mass in the ascending colon near the cecum (about 4 cm from the cecum) and a large, ulcerated mass in the cecum.

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Figure 1: Computed tomography image of the abdomen (coronal view).

DISCUSSION

NSAIDs are commonly used to treat pain and fever caused by a variety of medical conditions, including arthritis, menstrual cramps,

headaches, colds, and influenza. They are both prescription and over-the-counter medications that are generally well tolerated. Dyspepsia, nausea, and vomiting are the most common side effects. Serious side effects, such as gastrointestinal ulceration or bleeding, are rare but are more likely with high doses and prolonged use. The toxicity of NSAIDs extends beyond the gastro duodenum to the small and large intestines. Ulcerations, exacerbation of inflammatory bowel disease, NSAID-induced diverticular bleeding, and colonic strictures are common colonic mucosal injuries.

In recent years, evidence has accumulated that NSAID-related bowel injury is not limited to the gastro duodenum, and that NSAIDs may also cause or exacerbate injury in the small intestine and colon. Despite their widespread use in the general population, NSAID-induced colitis is rarely reported in medical literature and is widely underestimated by most clinicians [14].

The elderly and patients undergoing long-term treatment are most vulnerable. The most commonly reported indications for medication intake in published series of NSAID colitis include arthritis or osteoarthritis, other musculoskeletal disorders, toothache or headache, sinusitis, postoperative or postpartum pain, lumbar pain, dysmenorrhea, and prophylaxis against vascular disease. There may be oral, intramuscular, rectal, and topical preparations involved. Approximately 85 percent of cases are caused by ibuprofen, diclofenac, and aspirin. Surprisingly, the toxic effect is not dose dependent [15].

The mechanisms underlying NSAID-induced colitis remain unknown. Its pathogenesis is likely multifactorial, with inhibition of cyclooxygenase and prostaglandin synthesis, as well as impairment of oxidative phosphorylation. The fact that intestinal changes are mostly limited to the right hemicolon, with typical rectal sparing, suggests a role for direct drug toxicity on the intestinal wall. Because most NSAIDs circulate via enterohepatic routes, the proximal colon is directly exposed to intact drug after bacterial breakdown in the distal ileum, and the cecum serves as a reservoir. Furthermore, the use of enteric-coated and slow release preparations allows more drugs to reach the colon [16].

CONCLUSION

The natural history of NSAID-induced colitis is poorly understood, as evidenced by a paucity of case reports and case series. Symptoms may include diffuse abdominal pain, diarrhea, lower gastrointestinal bleeding, and, in rare cases, ulceration or perforation. Weight loss, symptomatic iron deficiency anemia, and the long-term development of diaphragm-like fibrotic strictures are all symptoms of sub acute and chronic forms. Hospitalization is required in up to 20% of cases.

REFERENCES

1. Theodoropoulou Angeliki, Koutroubakis IE. Ischemic colitis: clinical practice in diagnosis and treatment. *World J Gastroenterol* 2008;14:7302-8. 10.3748/wjg.14.7302 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
2. Nadatani Y, Watanabe T, Shimada S, et al. . Microbiome and intestinal ischemia/reperfusion injury. *J Clin Biochem Nutr* 2018;63:26-32. 10.3164/jcbn.17-137 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
3. Schoots IG, Koffeman GI, Legemate DA, et al. . Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. *Br J Surg* 2004;91:17-27. 10.1002/bjs.4459 [PubMed] [CrossRef] [Google Scholar]
4. Yadav S, Dave M, Edakkanambeth Varayil J, et al. . A population-based study of incidence, risk factors, clinical spectrum, and outcomes of ischemic colitis. *Clin Gastroenterol Hepatol* 2015;13:731-8. quiz e41 10.1016/j.cgh.2014.07.061 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
5. Choi SR, Jee SR, Song GA, et al. . Predictive factors for severe outcomes in ischemic colitis. *Gut Liver* 2015;9:761-6. 10.5009/gnl15167 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
6. Lee TC, Wang HP, Chiu HM, et al. . Male gender and renal

- dysfunction are predictors of adverse outcome in nonpostoperative ischemic colitis patients. *J Clin Gastroenterol* 2010;44:e96-100. [PubMed] [Google Scholar]
7. Newman JR, Cooper MA. Lower gastrointestinal bleeding and ischemic colitis. *Can J Gastroenterol* 2002;16:597-600. 10.1155/2002/374682 [PubMed] [CrossRef] [Google Scholar]
 8. Lee MJ, Daniels SL, Drake TM, et al. Risk factors for ischaemic colitis after surgery for abdominal aortic aneurysm: a systematic review and observational meta-analysis. *Int J Colorectal Dis* 2016;31:1273-81. 10.1007/s00384-016-2606-6 [PubMed] [CrossRef] [Google Scholar]
 9. Hass DJ, Kozuch P, Brandt LJ. Pharmacologically mediated colon ischemia. *Am J Gastroenterol* 2007;102:1765-80. 10.1111/j.1572-0241.2007.01260.x [PubMed] [CrossRef] [Google Scholar]
 10. Longstreth GF, Yao JF. Diseases and drugs that increase risk of acute large bowel ischemia. *Clin Gastroenterol Hepatol* 2010;8:49-54. 10.1016/j.cgh.2009.09.006 [PubMed] [CrossRef] [Google Scholar]
 11. Longstreth GF, Yao JF. Epidemiology, clinical features, high-risk factors, and outcome of acute large bowel ischemia. *Clin Gastroenterol Hepatol* 2009;7:1075-80. quiz 23 10.1016/j.cgh.2009.05.026 [PubMed] [CrossRef] [Google Scholar]
 12. Baixeli J, Kiran RP, Delaney CP. Investigation and management of ischemic colitis. *Cleve Clin J Med* 2003;70:920-1. 10.3949/ccjm.70.11.920 [PubMed] [CrossRef] [Google Scholar]
 13. Geramizadeh B, Taghavi A, Banan B. Clinical, endoscopic and pathologic spectrum of non-steroidal anti-inflammatory drug-induced colitis. *Indian J Gastroenterol*. 2009 Jul-Aug;28(4):150-3.
 14. Aftab AR, Donnellan F, Zeb F, Kevans D, Cullen G, Courtney G. NSAID-induced colopathy. A case series. *J Gastrointest Liver Dis*. 2010;19:89-91. [PubMed] [Google Scholar]
 15. Püspök A, Kiener HP, Oberhuber G. Clinical, endoscopic, and histologic spectrum of nonsteroidal anti-inflammatory drug-induced lesions in the colon. *Dis Colon Rectum*. 2000;43:685-91. [PubMed] [Google Scholar]
 16. Katsinelos P, Christodoulou K, Pilpilidis I, Xiarchos P, Papagiannis A, Dimiropoulos S, et al. Colopathy associated with the systemic use of nonsteroidal antiinflammatory medications. An underestimated entity. *Hepatogastroenterology*. 2002;49:345-8. [PubMed] [Google Scholar]



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