SAGE Open Medical Case Reports
Volume 12: 1–4

DOI: 10.1177/2050313X241256862
journals.sagepub.com/home/sco
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Acute ischemic colitis complicating an exacerbation of chronic obstructive pulmonary disease: A case report of gut-lung crosstalk

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Abstract

Acute ischemic colitis is a pathology as frequent as it is serious and requires urgent management. It's often occurring in a context of particular thromboembolic or hypovolemic risk, but certain clinical situations are not commonly known to provide mesenteric ischemia. Herein, we report the case of a 47-year-old man who presented with a severe acute colitis occurring in the course of acute exacerbation of a chronic obstructive pulmonary diseases with maintained stability of hemodynamic state. The diagnosis of acute ischemic colitis complicating an exacerbation of chronic obstructive pulmonary diseases was made. A clinical and biological improvement quickly marked the patient's condition after the management of the respiratory problem.

Keywords

Mesenteric ischemia, ischemic colitis, exacerbation, chronic obstructive pulmonary disease, corticosteroids

Date received: 13 February 2024; accepted: 2 May 2024

Introduction

Ischemic colitis was first described in 1966 by Martson and associates[1] referring to a clinical independent entity characterized by circulatory insufficiency of the colon, resulting in varying degrees of local tissue necrosis and systemic manifestations.

Despite the frequency of etiologies it typically develops in the absence of major vasculature occlusion and in presence of viable intestine elsewhere.

Chronic obstructive pulmonary diseases (COPD) is a multisystemic disease[2] with certain link with gastrointestinal (GI) disease particularly ischemic lesions; however, there are surprisingly few research studies that investigated the nature of the cross talking and while several mechanisms have been proposed.[3]

We report the case of acute ischemic colitis occurring in the course of acute exacerbation of a COPD with maintained stability of hemodynamic state.

Case report

A 47-year-old male, a non-weaned smoker with a medical history of type 2 diabetes and COPD (centrilobular emphysema), was admitted for an acute exacerbation of his chronic respiratory disease. Treatment included oxygen therapy, nebulized beta agonists, systemic corticosteroids based on hydrocortisone at a dose of 100 mg 6 hourly, and antibiotics based on Clarithromycin 500 mg 12 hourly plus IV Amoxicillin 1 g 8 hourly. This initial management leaded to clinical and biological improvement.

On the third day of hospitalization, the patient developed diffuse abdominal pain and bloody diarrhea (14 bowel movements per day) accompanied by a low-grade fever (38.1°C).

Physical examination revealed diffuse snoring rhonchi and mild periumbilical tenderness. The patient showed no signs of dehydration, and his hemodynamic parameters were stable. An electrocardiogram showed no disturbances in heart rhythm or repolarization.

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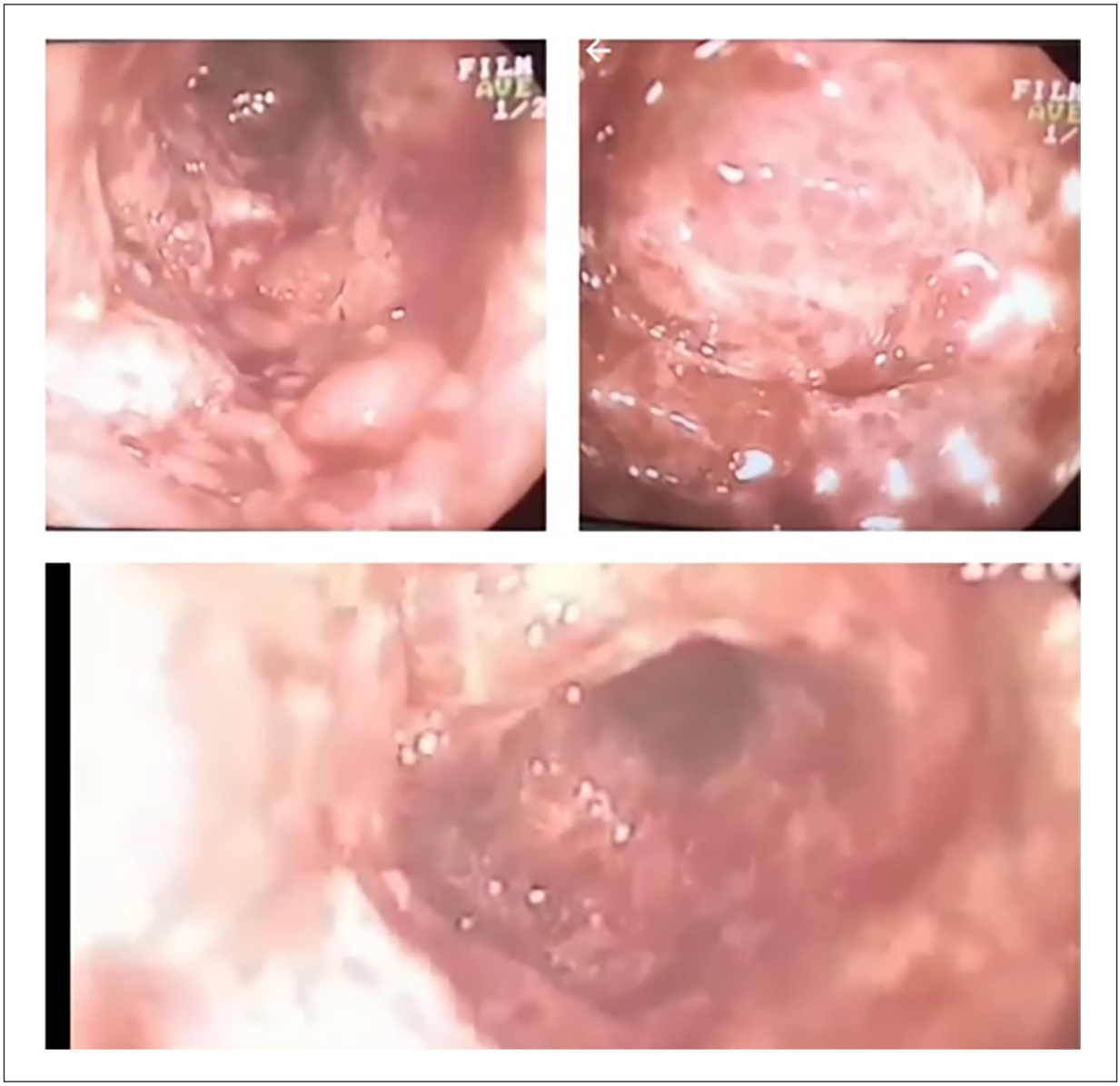

Figure 1. Rectosigmoidoscopy showing large and deep ulcers in the rectosigmoid junction.

Laboratory results showed a white blood cell count of 16,700 cells/mL, hemoglobin level of 15 g/dL, C-reactive protein level of 86 mg/L, metabolic acidosis in arterial blood gas analysis (pH 7.32, pCO2 47 mm Hg, pO2 75 mm Hg, HCO3 18 mmol/L) and lactate levels were slightly elevated to 4 mmol/L. Stool examination did not isolate any pathogens.

Colonoscopy revealed normal rectal mucosa but deep, large ulcers at the rectosigmoid junction, prompting cessation of further exploration (Figure 1). The diagnosis of severe acute colitis was established based on Truelove and Witts criteria. Intensive first-line medical treatment with intravenous corticosteroids and venous thromboembolism prophylaxis using low molecular weight heparin was initiated.

Two days later, the patient experienced resolution of bloody diarrhea and improved inflammatory markers. A follow-up endoscopic examination revealed edematous mucosa with superficial ulcerations and narrowed colonic lumen, particularly at the colonic angles while sparing the rectum. These findings strongly suggested an ischemic etiology.

Colonic biopsy showed total or partial necrotic lesions of the mucosa, particularly in the crypt epithelium, with mucosal hyalinization. The lamina propria exhibited hemorrhagic foci and fibrin deposits, and some submucosal vessels were occluded by recent fibrinous thrombi. These histological findings confirmed acute ischemic colitis (Figure 2).

An abdominal computed tomography angiography excluded mesenteric vascular occlusion.

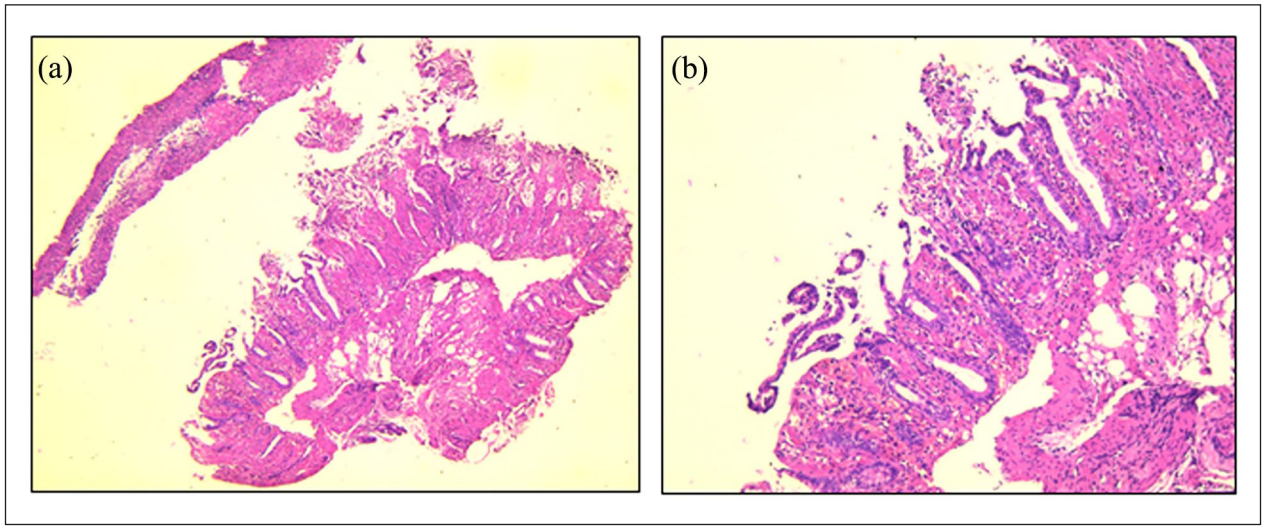

Figure 2. Results of the histological study of colonic specimen. (a) Marked exudate on the surface of the mucosa (HE stain × 40); (b) Mucosal hyalinization, denudation and vascular congestion with atypical reactive change of crypts (HE stain × 100).

The patient's condition improved rapidly, and corticosteroids were tapered off.

The acute exacerbation of COPD was deemed the probable etiology of ischemic colitis.

Following hospital discharge, the patient was monitored in an outpatient clinic for 1 year. He experienced complete resolution of GI symptoms and stable COPD but was subsequently lost to follow-up.

Discussion

COPD is primarily known as a respiratory disorder, but secondary organ manifestations, particularly in the GI tract, are becoming increasingly recognized. GI disorders are more prevalent in patients with COPD compared to healthy individuals.[4,5]

Research indicates a link between COPD and intestinal dysfunction, with several studies demonstrating compromised intestinal perfusion during acute exacerbations of COPD (AE-COPD). The increased metabolic demands associated with physical activities, such as acute exacerbations, may lead to reduced intestinal blood flow, resulting in ischemia and enterocyte damage.[6,7]

In resting conditions, both small and large intestines exhibit higher permeability in COPD patients compared to age-, BMI-, and sex-matched control subjects. This has been assessed using the excretion of orally ingested sugar probes. The increased permeability is associated with acute enterocyte damage, which occurs rapidly during physical exertion and persists after activity cessation. Notably, standardized household activities significantly exacerbate the difference in intestinal permeability between patients with COPD and control subjects.[6]

At rest, there is no evidence of increased enterocyte damage in COPD patients, suggesting that physical activity precipitates epithelial damage. Metabolic load, measured by serum lactic acid, correlates with intestinal permeability in these patients, indicating that physical exertion places a higher metabolic demand on COPD patients.

The pathophysiological and metabolic similarities between intense physical effort and AE-COPD suggest a potential association between COPD exacerbations and intestinal ischemia. This hypothesis posits that COPD patients, struggling to meet the metabolic demands of daily activities, may experience intestinal ischemia and related enterocyte damage.[7,8]

Interestingly, while chronic intestinal ischemia can lead to increased intestinal permeability, the intestinal mucosa—particularly epithelial cells—possess adaptive mechanisms that may mitigate acute ischemic damage.[9,10]

Rutten et al.[6] observed that only a minority of COPD patients in their study were current smokers. This suggests that COPD progression can continue even after smoking cessation. Their findings demonstrate that epithelial damage in COPD patients may occur independently or as a consequence of smoking.

Impaired lung function and increased metabolic demands associated with COPD may promote intestinal ischemia and have significant implications for other extrapulmonary manifestations of the disease.[6,11]

In addition, increased intestinal permeability and altered colonic bacterial biofilm have been observed in COPD patients, with moderate activities leading to splanchnic hypoperfusion.[12]

Hypoxemia is a common feature in patients with severe AE-COPD, and oxygen therapy is standard treatment in hospitalized COPD patients. However, the systemic consequences of hypoxemia, particularly its effects on the GI tract, are often overlooked in clinical practice. This warrants extending monitoring protocols to include the digestive system for patients with severe AE-COPD.[13]

Conclusion

Chronic mesenteric ischemia in patients with COPD must be highlighted, thus requiring the management of all ischemical risk factors associated with COPD.

In the context of AE-COPD any acute digestive manifestation should raise the suspicion of the ischemic etiology.

Acknowledgements

None.

Author contributions

Z.M. contributed to conception; N.A. contributed to writing the manuscript; B.A. contributed to data collection; L.M.H. contributed to literature review; G.A. contributed to data collection; J.I. contributed to design; N.B.C. contributed to critical review; Z.A. contributed to supervision; S.L. contributed to supervision.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethics approval

Our institution does not require ethical approval for reporting individual cases.

Informed consent

Written informed consent was obtained from the patient for his anonymized information to be published in this article.

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