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Single Case

A Novel Case of Ischemic Colitis in the Setting of Kratom Use: A Case Report

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Keywords
Kratom · Ischemia · Colitis · Abdominal pain

Abstract
Introduction: Kratom is an herbal preparation – generally an extract, capsule, chew, or smoking substrate – made from the leaves of a tropical tree (*Mitragyna speciosa*) that is indigenous to Southeast Asia. Its physiological effects are complex and dose dependent: low doses mimic stimulants, while higher doses have sedative and analgesic effects. Although injury and failure of various organs have been reported with kratom use across multiple case study analyses, gastrointestinal involvement has been rarely documented. ***Case Presentation:*** We present a 26-year-old female with no past medical history, no risk factors for intestinal ischemia, and an addiction to the commercial substance, kratom, who presented for 1 day of bright-red bloody diarrhea and bilious vomiting, and several weeks of intermittent sharp abdominal pain. Colonoscopy showed decreased mucosa vascular pattern in watershed areas of the colon and a splenic flexure that was so ulcerated it could not be safely traversed. ***Conclusion:*** Given the lack of concomitant risk factors and comorbidities, as well as the timing of her symptoms beginning well before she started any medications, our most likely patient developed ischemic colitis due to her kratom use. This is further supported by her repeat colonoscopy 2 months after quitting kratom, which showed complete resolution. We would advise vigilance for possible bowel ischemia in those using kratom, a substance on which there is currently limited knowledge.

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Introduction

Kratom is an herbal preparation, generally an extract, capsule, chew, or smoking substrate, made from the leaves of a tropical tree (*Mitragyna speciosa*) that is indigenous to Southeast Asia. Mitragynine, a mu-opioid receptor partial agonist, is the main pharmacologically active component of kratom [1]. The physiological effects of kratom are complex and dose dependent; low doses mimic stimulants, while higher doses have sedative and analgesic effects [2]. An estimated 1.7 million people aged 12 or older used kratom in 2021 and the lifetime prevalence rate of kratom use in the USA is estimated at 1.3% [3, 4]. While the Food and Drug Administration has not approved kratom for any clinical indication [5], multiple mice studies and a cross-sectional study in humans have shown kratom can significantly decrease withdrawal symptoms in chronic opioid users [6–8]. Notable adverse effects associated with kratom use include agitation, tachycardia, seizures, hallucinations, and a wide spectrum of organ injuries [2, 9–11]. Although injury and failure of various organs have been reported with kratom use across multiple case study analyses, reports of GI involvement are rare [11–13]. We present a case of a patient in withdrawal from 1 year of daily kratom use who went on to be diagnosed with ischemic colitis.

Case Report

The patient is a 26-year-old female with no known past medical history, who presented to the emergency department from a rehabilitation center for 1 day of at least 8 episodes of bloody, foul-smelling diarrhea, nausea, and bilious vomiting. She denied any previous history of bloody diarrhea but did note intermittent severe, cramping abdominal pain, which was not associated with her menstrual cycle for several weeks leading up to her hospitalization. Due to this pain, the patient's gynecologist restarted her previously taken oral contraceptives 5 days prior to admission. She denied recent travel, sick contacts, family history of bowel disease, new diet, or any other lifestyle changes. The patient denied smoking tobacco, vaping, marijuana and noted social drinking. Of note, she endorsed daily kratom use, taking at least one 8.8 mL tincture kratom shot per day for the last 1 year. The patient reported she unsuccessfully tried to stop using kratom several times, which prompted her to check into a rehabilitation center 2 days prior to admission. The patient was transferred from the rehabilitation facility to the hospital after the bloody diarrhea started. She last used kratom 2 days prior to hospital admission.

On arrival, the patient was tachycardic, afebrile, and normotensive. Her abdomen was diffusely tender, a digital rectal exam showed bright red blood but was otherwise unremarkable, and nursing witnessed several episodes of bloody diarrhea in person. Laboratories were significant for white blood cell count of 25 k, hemoglobin within normal limits and normocytic, fecal calprotectin of 925 μg/g (reference <120), and high-sensitivity C-reactive protein of 116 mg/L (reference 0.10–2.80). Urine drug screen was positive only for opiates, which she was given upon arrival. Laboratories were otherwise unremarkable including lactate within normal limits and stool studies (*C. difficile* toxin, *Salmonella*, *Campylobacter*, Shiga toxin, *Giardia*, ova and parasite, and fecal leukocytes), urine pregnancy test, hepatitis panel, and HIV were all negative. Initial computed tomography (CT) scan showed diffuse inflammatory changes in the colon and inflammatory changes of the terminal ileum potentially representing backwash ileitis. She was given analgesics, antiemetics, and IV fluids and kept NPO.

Colonoscopy on day 2 of hospitalization demonstrated diffusely decreased mucosa vascular pattern in the sigmoid colon, in the descending colon, and at the splenic flexure along with a continuous area of congested, edematous, and nonbleeding ulcerated mucosa at

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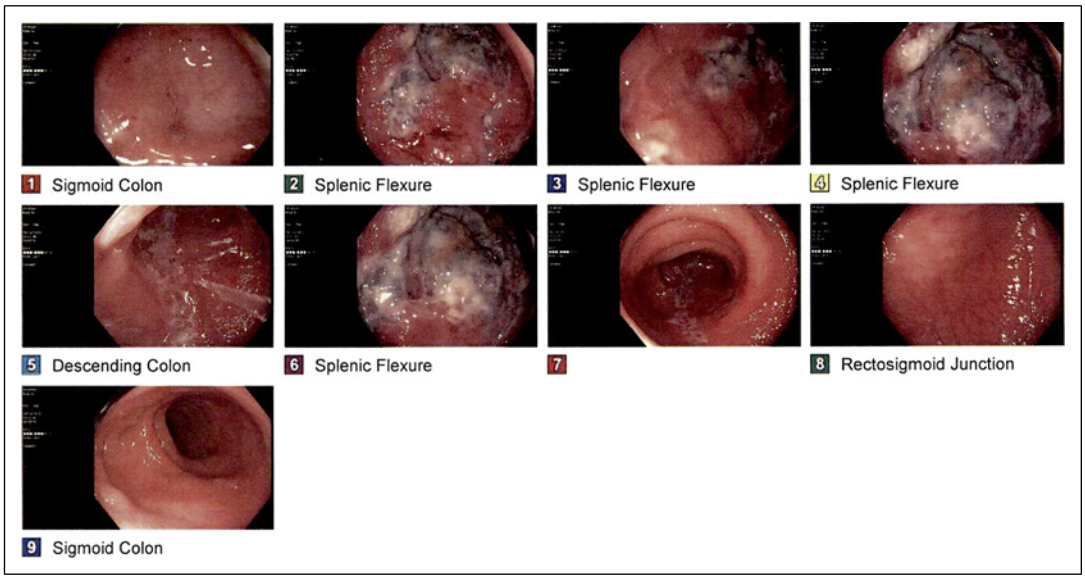

Fig. 1. Colonoscopy on day 2 of hospitalization demonstrating diffusely decreased mucosa vascular pattern in the sigmoid colon, in the descending colon, and at the splenic flexure along with a continuous area of congested, edematous, and nonbleeding ulcerated mucosa at the splenic flexure.

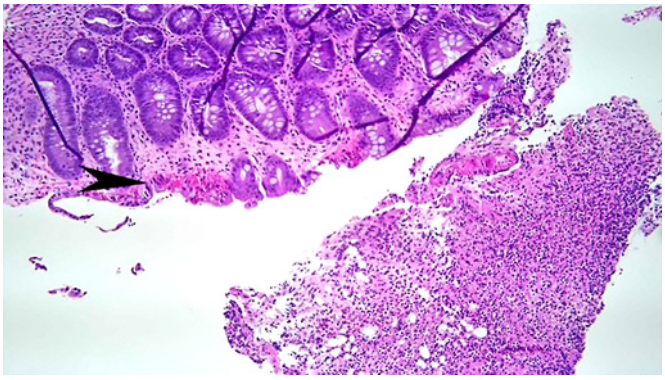

Fig. 2. Initial splenic flexure biopsy with H&E stain under ×10 view demonstrating colitis with mucosal erosion (arrow) and focal hyalinization of the lamina propria.

the splenic flexure (Fig. 1). The endoscope was not able to be advanced past the splenic flexure due to the degree of friability. Biopsy taken from the splenic flexure revealed colonic mucosa with erosions, injured crypts, focal hyalinization of the lamina propria, and neutrophilic inflammatory exudate, with no overt features of chronic colitis (Fig. 2, 3).

The patient continued to report frequent, severe, stabbing abdominal pain along with bloody diarrhea, until day 4 of hospitalization, at which point the diarrhea was no longer bloody, and the abdominal pain decreased in frequency, noted to occur 20 min after eating with advancement of diet. Given the colonoscopy suspicious for ischemia in the watershed areas, CT angiography of the abdomen and pelvis was done on day 5, which demonstrated the previously seen diffuse edematous mural thickening and surrounding fat stranding of the ascending, transverse, and descending colon, most likely representing inflammatory or infectious colitis. The scan noted that the inflammation was not in a particular vascular distribution. Diet was

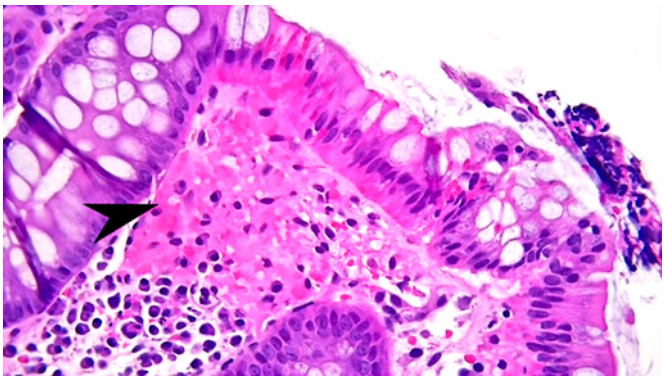

Fig. 3. Initial splenic flexure biopsy with H&E stain under ×40 view demonstrating colitis with focal hyalinization of lamina propria (arrow).

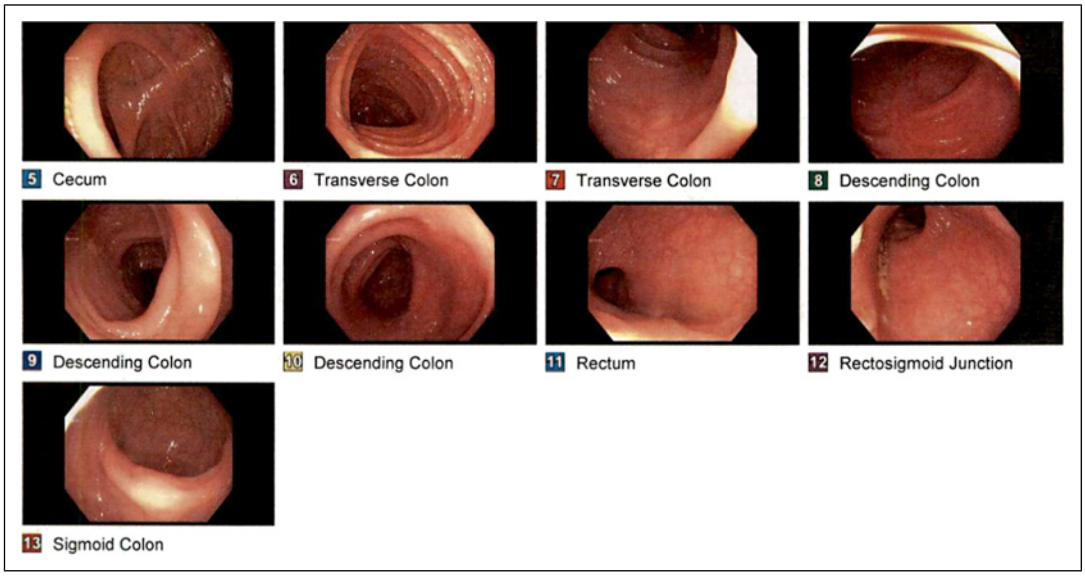

Fig. 4. Colonoscopy 2 months after admission showing an unremarkable examined colon with complete resolution of previously seen pathology.

slowly advanced until she was able to tolerate small solid snacks. The patient was discharged on day 7 with outpatient gastroenterology follow-up and repeat colonoscopy scheduled.

Two months after admission and discontinuation of kratom, the patient underwent outpatient colonoscopy showing a normal examined colon as well as a normal examined ileum (Fig. 4). One biopsy taken of the left colon showed colonic mucosa with no significant pathologic changes (Fig. 5).

Discussion

Our patient had a unique presentation of nonocclusive ischemic colitis, without the presence of typical risk factors including advanced age, history of thrombi, smoking, cerebrovascular disease, heart disease, or peripheral artery disease, nor common inciting

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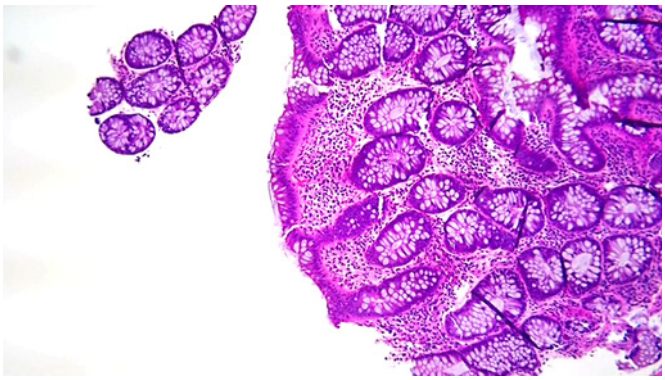

Fig. 5. Biopsy from descending colon 2 months after admission with H&E stain under ×10 view with no significant pathological changes.

factors like hypovolemia, sepsis, arrhythmia, valvopathy, or traumatic injury. The patient did restart estrogen-progesterone oral contraception, which has a known risk of hypercoagulability, 5 days prior to admission. However, it is less likely that this contributed considering she had taken that type of oral contraceptive from age 15–19 without incident, CT angiography was negative for thrombus, there was a watershed distribution of ulceration seen on colonoscopy, and the patient had endorsed crampy intermittent abdominal pain for several weeks prior to starting the medication.

While there is no conclusive test to prove that the patient's kratom use contributed to her nonocclusive ischemic colitis, it is the most likely explanation given the lack of alternate etiologies and resolution of her condition after discontinuation of the substance. Additionally, the patient's symptoms of nausea, vomiting, and pancolitis align with limited previous literature in GI side effects of kratom [11–13]. Of note, concomitant small bowel ischemia could not be ruled out as friability prevented imaging beyond the splenic flexure.

This case highlights the importance of obtaining a detailed history of substance use including specific questions about kratom, as well as vigilance for possible bowel ischemia in those actively using the substance. There is minimal literature on kratom and further research is warranted to delineate mechanisms of kratom-induced cellular injury.

Statement of Ethics

Written informed consent was obtained from the participant for publication of the details of their medical case and any accompanying images. In accordance with national guidelines, Institutional Review Board was not required for this case report. The CARE Checklist has been completed by the authors for this case report and is attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000546558).

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

L.S.B. and Y.S. aided in conception and creation of manuscript, drafting of the manuscript, final approval, and agreement of accountability for all aspects of the work. C.M., A.A., R.M., P.S., and M.P.G. aided in conception of manuscript, drafting of the manuscript, final approval, and agreement of accountability for all aspects of the work. L.S.B. is the article guarantor.

Data Availability Statement

Further details from the case are not available publicly due to ethical concerns; however, they may be requested from the authors by editors and reviewers as necessary while remaining compliant with HIPAA.

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