



Clinical Case Study

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A RARE CASE OF ISCHEMIC COLITIS: CEFUROXIME-RELATED ANAPHYLACTIC SHOCK

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Ischemic colitis is the most frequent form of intestinal ischemia. It develops as a result of decreased blood flow in the small arterioles of the colon and is usually nonocclusive. The most common underlying causes are heart failure, myocardial infarction, acute hemorrhage, sepsis, cardiac arrhythmias, hypotension, or dehydration. This disease usually occurs in debilitated elderly patients. In many patients, the episode is transient. Ischemic colitis generally resolves spontaneously, but, in very few cases, it may occur with serious consequences such as peritonitis and perforation due to colonic necrosis. The classical clinical presentation of the disease consists of sudden onset abdominal pain, diarrhea, and rectal bleeding (Yngvadottir et al., 2017). Treatment ranges from parenteral support to surgical intervention depending on the severity of the disease.

Anaphylaxis is a serious allergic reaction. Its typical symptoms are itchy rash, throat or tongue swelling, shortness of breath, vomiting, lightheadedness, and low blood pressure. These symptoms typically appear within minutes to hours of exposure to an allergen (Takazawa, Oshima, & Saito, 2017). Herein, we present a unique case of ischemic colitis that developed as a result of cefuroxime-associated anaphylactic reaction.

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Case Report

A 51-year-old woman who had no illness, allergy, or smoking history was admitted to the emergency department with abdominal pain and bloody diarrhea. According to the patient's history, she was administered cefuroxime 1 g intramuscularly due to dental abscess 7 hours prior. Immediately after administration, the patient experienced a generalized erythematous reaction, respiratory distress, and syncope. Her systolic blood pressure was very low in the emergency department. The patient's blood pressure and consciousness improved after the administration of intramuscular epinephrine, intravenous corticosteroids, parenteral fluid, and H₁-antihistamines.

She was referred to our emergency department after developing severe abdominal pain and rectal bleeding. Physical examination was compatible with mild precission on the left side of abdomen. Rectal examination showed bright red blood without any stool. Colonoscopic examination showed circumferential deep ulcerations and severe mucosal erythema with friability between the splenic flexure and the descending colon (Figure 1). The mucosa and vascular structures were normal in the rectum. Endoscopic findings were highly suspicious of ischemic colitis.

Computed tomography angiography revealed no filling defect in the aorta and its branches. Blood cultures, stool cultures, and gastrointestinal panel were negative for *Clostridium difficile*. Total IgE was 500 IU/ml. A biopsy specimen reported mucosal and submucosal edema with ulcerations, necrosis, erythrocyte extravasation, focal lamina propria eosinophilic change with mild crypt attenuation, and loss of goblet cells consistent with mild ischemic changes (Figure 2). Evidence of chronic inflammation was not observed.

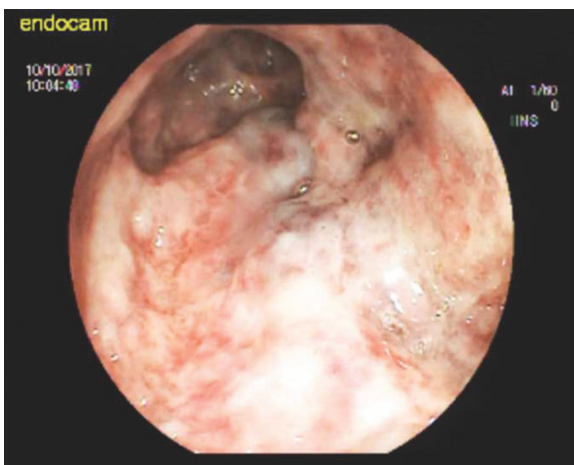


FIGURE 1. Circumferential deep ulcerations and severe mucosal erythema with friability of the colon.

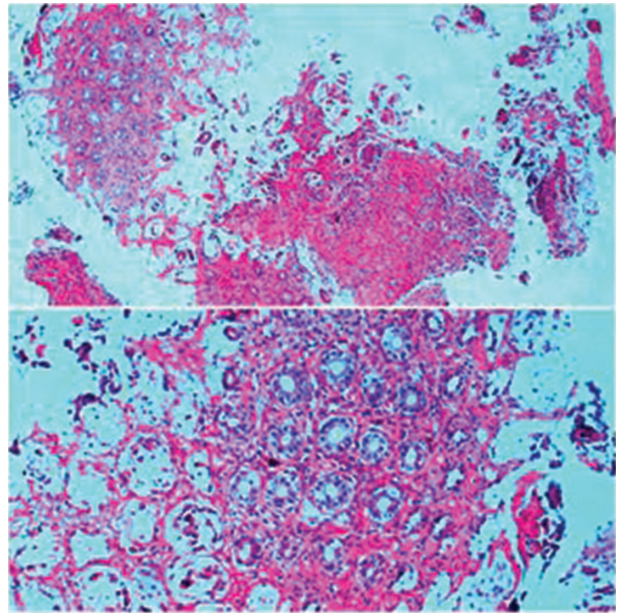


FIGURE 2. (A) Intestinal fragments showing partial necrosis and have undergone coagulative hemorrhagic infarct. Fibrin thrombi are observed in some of the capillary lumens (HE $\times$ 40). (B) Superficial epithelial lining from the crypts is extensively denuded. Residual glands are identifiable at the base of mucosa (HE $\times$ 100). HE = hematoxylin–eosin.

She was treated with intravenous fluid and bowel rest. She was observed for 7 days, and her diet was progressed slowly. Her bloody bowel movements ceased after 3 days in the hospital. Follow-up colonoscopy was performed 2 months later, and the mucosal damage was completely healed.

Discussion

Colonic ischemia is the most common form of intestinal ischemia, and it was first described in 1963 by Boley (Higgins, Davis, & Laine, 2004). Ischemic colitis is more common in debilitated elderly patients who have comorbid conditions such as surgery, vascular disease, low blood flow, colon cancer, intestinal stricture, diverticulosis, and atherosclerosis (Cruz, Abujudeh, Nazarian, & Thrall, 2015). On the contrary, this disease may rarely occur in young patients without any risk factors.

Intestinal ischemia develops as a result of three mechanisms: nonocclusive colonic ischemia; embolic-thrombotic arterial occlusion; and mesenteric venous thrombosis. The colon is the most affected region of the gastrointestinal tract as a result of hypoperfusion. In addition, the colonic wall is thicker than the small intestine wall and contains fewer microvascular plexuses. Particularly, the “watershed” regions of the colon where collateral blood flow is limited are affected.

The most affected segments are the splenic flexure and the rectosigmoid junction. The disease may be

limited to the mucosa according to the degree of ischemia; it may also involve all the layers of the colon wall. The most important risk factor is age; other risk factors are comorbid conditions such as heart failure, myocardial infarction, acute hemorrhage, sepsis, cardiac arrhythmias, aortic surgery, hypotension, or dehydration (Greenwald & Brandt, 1998). There are limited reports on the development of ischemic colitis after anaphylactic reactions.

Anaphylaxis is an emergency that is sudden and may result in death. The causes of anaphylactic reactions usually include allergic responses to food, drugs, hormones, insect bites, exercise, and latex (Takazawa et al., 2017). The most common cause of anaphylaxis after nutrients is medications, especially β -lactam antibiotics. The most common affected systems are cutaneous, respiratory, cardiovascular, and gastrointestinal systems. Symptoms typically include urticaria, angioedema, dyspnea, wheezing, dizziness, syncope, and hypotension. Although nausea, vomiting, diarrhea, and abdominal pain are common gastrointestinal symptoms, bleeding due to mucosal damage is very rare (Yngvadottir et al., 2017).

Cefuroxime is a β -lactam antibiotic. Drugs, especially β -lactam antibiotics, are among the leading factors for developing anaphylactic reactions. There are only a few cases of gastrointestinal disorders in anaphylaxis that have been reported in the literature.

One of these reports was the peptic ulcer after an anaphylactic reaction associated with a urographic contrast (Rivera, 1972). This report supported the development of gastric mucosal damage after an anaphylactic reaction. The other report was severe ischemic colitis due to IgE-mediated immune reaction following the use of amoxicillin. In that article, the authors emphasized the connection between anaphylaxis and ischemic colitis to be controversial. In addition, they indicated that gastrointestinal disorders associated with anaphylaxis might be caused by systemic stress or steroid treatment (Perez-Carral, Carreira, & Vidal, 2004).

Finally, Shirai, Mori, Uotani, and Chida (2007) suggested that anaphylaxis presentation was mysterious

and that gastrointestinal mucosal damage might develop due to anaphylactic reactions. In that article, the authors commented that the mucosal damage of patients with mild abdominal complaints might be unnoticed. Herein, we report a unique case of an anaphylactic reaction after cefuroxime administration that was complicated by acute colonic ischemia.

Conclusion

Anaphylactic reactions can affect all systems and may present with different symptoms. The mechanism of gastrointestinal mucosal damage after an anaphylactic reaction is still controversial. Gastrointestinal mucosal damage may occur either due to immunological reaction or due to hypoperfusion after anaphylactic shock. As a result, clinicians should keep in mind that anaphylactic reactions can lead to ischemic colitis and should be considered in the differential diagnosis of lower gastrointestinal bleeding even in individuals without any predisposing factors.

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