

Gastrointestinal Disorders in Anaphylaxis

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Abstract

We report two rare cases complicated by gastrointestinal mucosal disorders, including peptic ulcer and ischemic colitis. Anaphylaxis was induced by cefaclor in case 1 and by pranoprofen in case 2. These two patients developed epigastric or lower abdominal pain about 10 hours after the onset of anaphylaxis. Gastroduodenoscopy revealed severe ulcers in the stomach or duodenum, while colonoscopy detected mucosal edema, erythema, and erosions, leading to the diagnosis of ischemic colitis. It is important to keep in mind that gastrointestinal mucosal lesions can occur, albeit on rare occasions, in patients with anaphylaxis.

Key words: anaphylaxis, ischemic colitis, peptic ulcer

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Introduction

Anaphylaxis is an immediate systemic reaction caused by rapid, immunoglobulin E (IgE)-mediated immune release of potent mediators from tissue mast cells and peripheral basophils (1). The most common etiologies of anaphylactic reactions include allergic responses to food, medications, *Hymenoptera* stings, and latex (2). In the classic form, anaphylaxis typically involves the cutaneous, respiratory, and cardiovascular systems, commonly presenting with urticaria, angioedema, dyspnea, wheeze, dizziness, syncope, and hypotension. Gastrointestinal symptoms, including nausea, vomiting, diarrhea, and cramping abdominal pain, can occur transiently but mucosal lesions are rarely detected. In this communication, we report on two rare cases complicated by gastrointestinal mucosal disorders, including peptic ulcer and ischemic colitis.

Case Report

Case 1: A 27-year-old man visited a local clinic complaining of fatigue and arthralgic pain in February 2003 and received medication, including acetoaminophen, cefaclor, and diclofenac. He developed generalized itching, oral dysesthesia, and dyspnea followed by hypotension and loss of consciousness 20 minutes after taking these medications. He was treated with subcutaneous epinephrine and intrave-

nous corticosteroids in the emergency department of our hospital. Fifteen hours after admission he developed epigastric pain, nausea, and vomiting. Gastroduodenoscopy revealed generalized ulcers with white exudation and petechiae from the bulbous to the second portion of the duodenum (Fig. 1, left panel) and histology confirmed duodenal ulcer with eosinophilic infiltration. Other imaging tests with regard to mucosal injury were not performed. His symptoms were resolved with a proton pump inhibitor. Total IgE was 160 IU/ml. Specific IgE antibodies were negative for 11 common allergens. Aspirin intolerance was excluded by the negative results of inhalation challenge with sodium toluometin. Since oral challenges with acetoaminophen and diclofenac were negative, it was possible that cefaclor was the cause of anaphylaxis.

Case 2: A 65-year-old man had a history of cervical spondylosis. He took a pranoprofen tablet for cervical discomfort after a 3-year interval in June 2004. Thirty minutes later, he developed dyspnea followed by generalized erythematous eruption, hypoxia, hypotension, and loss of consciousness. He was moved to our hospital and treated with epinephrine and corticosteroids, which caused these symptoms to subside within a few hours. Eight hours after the onset of anaphylaxis, however, he experienced epigastric and lower abdominal pain and hematochezia. Gastroduodenoscopy revealed gastric ulcers in the antrum and body of the stomach. Colonoscopy detected mucosal edema, erythema, and erosions all over his descending colon, leading to the diagnosis

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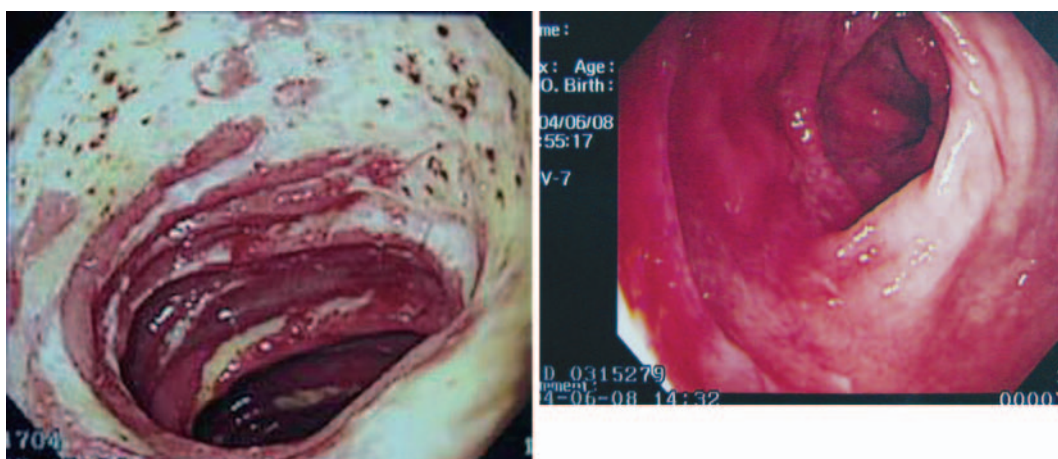


Figure 1. Endoscopic findings of the second portion of the duodenum (left panel: case 1) and descending colon (right panel: case 2).

of ischemic colitis (Fig. 1, right panel). Other imaging tests with regard to mucosal injury were not performed. The ulcers resolved with a proton pump inhibitor while the colitis resolved spontaneously. Neither gastroduodenoscopy nor colonoscopy was undertaken thereafter. Total IgE was 2900 IU/ml. Specific IgE antibodies were positive for *Candida*, *Aspergillus*, and Japanese cedar pollen. Aspirin intolerance was excluded by the negative results of oral challenge test with acetyl salicylic acid.

Discussion

Helicobacter pylori infection and nonsteroidal anti-inflammatory drug usage are the main causes of peptic ulcer (3), while the causes of acute ischemic colitis include arterial or venous occlusion and non-occlusive mesenteric ischemia, such as systemic hypotension, cardiac failure, and septic shock (4). There have been only limited numbers of reports on gastrointestinal disorders in anaphylaxis. Severe hemorrhagic gastritis following an anaphylactoid reaction due to a urographic contrast medium was reported by Rivera (5). He commented on the possible relationship between anaphylaxis and an intense but transient gastric mucosal reaction. Also, a case of acute ischemic colitis caused by IgE-

mediated allergy to amoxicillin was reported by Pérez-Carral et al (6). They speculated that the gut was damaged as a result of hypotension during anaphylaxis. In this communication, there was no evidence that the damage to the mucosa or gut was direct or induced by the immune reaction. At least in part, it is possible that systemic stress and nonsteroidal anti-inflammatory drug usage might be the cause of peptic ulcer and that hypotension during the anaphylactic episode might be the cause of ischemic colitis; however, reports on gastrointestinal mucosal lesions in anaphylaxis are very rare. It must be noted that most patients with anaphylaxis never develop peptic ulcer or ischemic colitis. Animal experimental studies suggest that mucosal anaphylaxis may cause ulceration and that the gut is the main organ of anaphylactic shock in rats (7-9). The presentation of anaphylaxis is often enigmatic, with variable target organ involvement and expression of symptoms. We speculate that some host factors might be associated with the onset of peptic ulcer and ischemic colitis in the present patients. Further, it is possible that mucosal lesions may be underdiagnosed in less severe patients complaining of abdominal symptoms. We should keep in mind that gastrointestinal mucosal lesions can occur, albeit on rare occasions, in patients with anaphylaxis.

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