

Images in Allergy

Isolated Abdominal Angioedema from an Angiotensin-Converting Enzyme-Inhibitor

Maximiliano Diaz-Menendez^a, Ismael Carrillo-Martin, MD^a, Monia E. Werlang, MD^b, William C. Palmer, MD^b, and Alexei Gonzalez-Estrada, MD^a Jacksonville, Fla

Angiotensin-converting enzyme-inhibitors (ACE-Is) are a frequently prescribed medication as treatment of several conditions including but not limited to hypertension. Angioedema is the second most common (prevalence 0.1%-3%) adverse effect of ACE-Is,¹ preceded only by cough (prevalence 10%-20%). ACE-I-associated angioedema can be life-threatening and typically occurs on the lips, tongue, pharynx, and larynx; however, some cases have a gastrointestinal presentation. This is a case of ACE-I-induced abdominal angioedema.

A 38-year-old woman with a history of hypertension was evaluated for episodic diarrhea, vomiting, nausea, abdominal pain, and 20-lb weight loss during the preceding 8 months. The patient had undergone multiple relatively invasive studies including biopsies, colonoscopy, endoscopy, Pill Cam, as well as imaging studies (abdomen and pelvis computed tomography, magnetic resonance imaging, and enterography). These studies failed to clearly orient toward a diagnosis and found only small-bowel thickening and enteritis (Figure 1). Furthermore, she required multiple hospitalizations and antibiotic courses, which failed to resolve or ameliorate her symptoms. During a comprehensive evaluation and discussion, the onset of symptoms was found to coincide with the start of lisinopril for the treatment of hypertension. The patient was advised to suspend treatment with ACE-I and was switched to amlodipine. Her symptoms resolved a day after and she remains without episodes of angioedema since her consultation in the allergy department 9 months prior. No further imaging studies were performed.

ACE-Is are commonly prescribed for the treatment of high blood pressure, heart failure, proteinuria, and coronary artery disease, among others. It is estimated that 40 million patients in the United States use these medications, and studies have shown that 20% to 40% of every 100,000 emergency department visits in the country are due to ACE-I-associated angioedema.² In the

literature, no prevalence rate of abdominal angioedema from an ACE-I is given; it is only known that "typical angioedema" can occur in 0.1% to 3% of cases. However, there are 34 reported cases of ACE-I-induced isolated abdominal angioedema during the past 40 years. Information regarding the frequency of airway angioedema along with abdominal angioedema is limited. The prevalence of ACE-I-associated angioedema may be underestimated because it is a diagnosis based on the exclusion of other probable causes of angioedema, which may lead to a delay in diagnosis when the clinical presentation is atypical or timing from start of ACE-I to symptom onset is unclear. As mentioned above, ACE-I-induced angioedema normally affects the upper airway; however, the presentation may vary. Abdominal presentation of ACE-I-induced angioedema might produce symptoms such as diarrhea, abdominal pain, nausea, and vomiting. This condition should be considered and addressed in an attempt to reduce delay in diagnosis and treatment. There is no evidence regarding an effective drug therapy to prevent ACE-I-induced angioedema, and treatment of this condition is limited to discontinuation of treatment with ACE-I in favor of alternative medications.¹ Recurrent angioedema may occur in around 50% of patients who have discontinued an ACE-I and usually within the first month. Finally, ACE-I-induced angioedema has been shown to occur up to 5 years after starting therapy with ACE-I;² therefore, it is important to consider these medications as a possible cause for angioedema to discontinue ACE-I use as early as possible and resolve patients' symptoms.²

REFERENCES

1. Baram M, Kommuri A, Sellers SA, Cohn JR. ACE inhibitor-induced angioedema. *J Allergy Clin Immunol Pract* 2013;1:442-5.
2. Banerji A, Blumenthal K, Lai K, Zhou L. Epidemiology of ACE inhibitor angioedema utilizing a large electronic health record. *J Allergy Clin Immunol Pract* 2017;5:744-9.

^aDivision of Pulmonary, Allergy and Sleep Medicine, Mayo Clinic, Jacksonville, Fla

^bDivision of Gastroenterology and Hepatology, Mayo Clinic, Jacksonville, Fla

Conflicts of interest: The authors declare that they have no relevant conflicts of interest.

Received for publication August 22, 2019; revised October 24, 2019; accepted for publication October 27, 2019.

Available online ■ ■ ■

Corresponding author: Alexei Gonzalez-Estrada, MD, Division of Pulmonary, Allergy, and Sleep Medicine, Department of Medicine, Mayo Clinic, 4500 San Pablo Rd, Jacksonville, FL 32224. E-mail: gonzalez.alexei@mayo.edu.

J Allergy Clin Immunol Pract 2019;■:■-■.
2213-2198

© 2019 American Academy of Allergy, Asthma & Immunology

<https://doi.org/10.1016/j.jaip.2019.10.035>

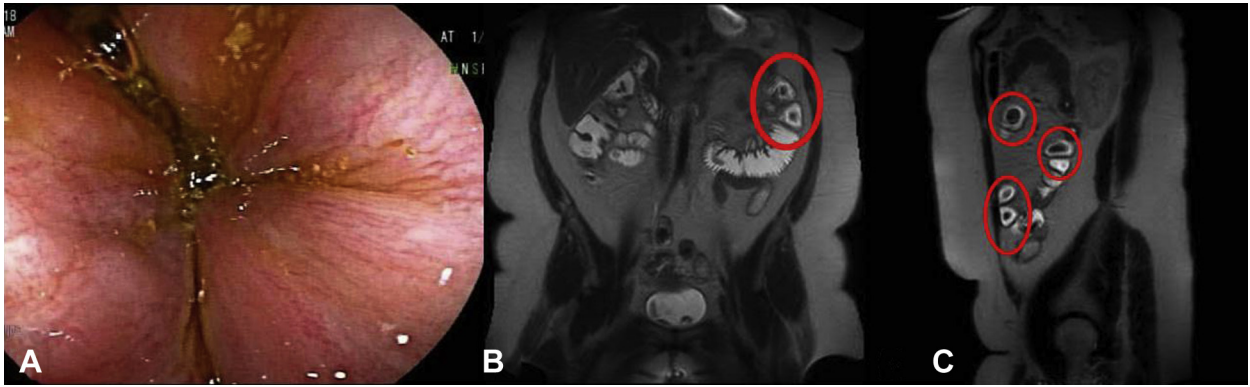


FIGURE 1. (A) Lower endoscopy with lumen reduction secondary to wall thickening. (B) MRI (coronal) showing wall thickening of descending colon. (C) MRI (sagittal) showing wall thickening of ascending, transverse, and descending colon. *MRI*, Magnetic resonance imaging.