HYPOVOLEMIC SHOCK CAUSED BY ANGIOTENSIN-CONVERTING ENZYME INHIBITOR-INDUCED VISCERAL ANGIOEDEMA: A CASE SERIES AND A SIMPLE METHOD TO DIAGNOSE THIS COMPLICATION IN THE EMERGENCY DEPARTMENT

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Abstract—Background: Visceral angioedema is a rarely reported side effect of angiotensin-converting-enzyme inhibitors (ACEI). Because signs and symptoms tend to be nonspecific, the diagnosis is difficult to make, especially in the emergency department (ED). Case Report: We describe 2 patients presenting with signs of hypovolemic shock, in which the diagnosis of ACEI-induced visceral angioedema was made in the ED. We surmise that patients with abdominal pain, who present with hypovolemic shock and are taking medications that can predispose to angioedema, may have this complication if their hemoglobin level is elevated compared with their previous levels. An abdominal computed tomography scan, if it does not identify any other significant etiology, will increase the probability that ACEI-induced visceral angioedema is the diagnosis when there is nonspecific bowel wall thickening or edema. Why Should an Emergency Physician Be Aware of This?: Identification of ACEI-induced visceral angioedema in the ED will avoid prolonged admissions, unnecessary procedures, and future recurrences.

Keywords—visceral angioedema; shock; hypotension; tachycardia

INTRODUCTION

Angiotensin-converting enzyme inhibitors (ACEI) are commonly used medications for chronic congestive heart failure and hypertension treatment (1). The life-threatening complication of oral angioedema is one known side effect of ACEI. However, we suspect that few emergency physicians are aware that ACEIs can also cause another major side effect, edema of the intestines or visceral angioedema. A 2013 literature review noted only 27 cases of ACEI-induced visceral angioedema (2).

In this article, we discuss 2 patients presenting with mild gastrointestinal symptoms and cardiovascular compromise, who were ultimately diagnosed with visceral angioedema. To our knowledge, these are the first cases in the literature attributing hypovolemic shock to ACEI-induced visceral angioedema (AIVA).

CASE REPORTS

Case One

A 62-year old obese man (189 kg) presented by ambulance with abdominal pain, vomiting, and diarrhea. His symptoms began 1 h prior to presentation while eating dinner and he attributed his symptoms to something he ate. He first noticed diffuse and cramping abdominal pain, which was then rapidly followed by five nonbloody watery stools. When he became lightheaded and dizzy, he called an ambulance. Initial blood pressure in the ambulance was 80 mm Hg systolic and he
received 500 cc of intravenous normal saline prior to arrival.

In the emergency department (ED), his blood pressure had improved to 97/80 mm Hg after his fluid bolus. His other vital signs were: pulse 77 beats/min, temperature 36.7°C (98.1°F), respiratory rate 18 breaths/min, and pulse oximetry 94% on 2 L oxygen by nasal cannula. He denied any chest pain or dyspnea, but did admit to diaphoresis, which had resolved after receiving the fluid bolus. His abdominal pain was still diffuse, but improved. His lightheadedness and dizziness had resolved by the time he reached the ED, and he stated that he felt much better. While undergoing his evaluation in the ED, the patient had two episodes of nonbilious, nonbloody emesis.

His review of systems was negative for fever, chills, cough, headache, oropharyngeal symptoms, change in voice, pruritus, back pain, rash, or flushing. He denied any allergies. He also denied any alcohol, tobacco, or illicit drug use. Medications included amlodipine, chlorothalidone, lisinopril, atorvastatin, allopurinol, aspirin, and omeprazole, and he stated compliance with all of them.

His past medical history was significant for cardiac arrest due to presumed ventricular tachycardia 3 years prior, and subsequent defibrillator placement. During that hospitalization, he underwent coronary catheterization, which revealed normal coronary arteries. An echocardiogram showed an ejection fraction of 65% with no valvular abnormalities, but did show grade II diastolic dysfunction. Other past medical history was hypertension, gout, and sleep apnea, for which he used a continuous positive airway pressure machine at night.

Of note, the patient had presented to the ED at least seven times over the past 3 years with abdominal pain, vomiting, and diarrhea. Each time he was admitted to the hospital, responded well to intravenous fluids, and was discharged within 72 h. No infectious etiology could be identified. The most recent episode was 3 months prior. Stool cultures were obtained, as the patient did not complain of chest pain or dyspnea, and had resolution of his abdominal pain within 24 h. Electrocardiogram (ECG) and troponin levels were not obtained, as the patient did not complain of chest pain or dyspnea, and had a normal coronary catheterization 3 years prior, and had a normal ECG and troponin during the most recent hospital admission 3 months prior. Stool cultures were ordered, but later cancelled, as he had no further episodes of diarrhea while in the hospital. C4
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