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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.** © 2017 Elsevier Inc. All rights reserved.

☐ **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 non-bloody 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

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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, chlorthalidone, 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 to the current visit, where he presented with abdominal pain, hypotension (blood pressure 70/36 mm Hg), mild hypoxemia (oxygen saturation 95% on 2 L oxygen), leukocytosis (white blood cells [WBC] 28,500 cells/uL), and an elevated hemoglobin (21 g/dL). These all corrected to his baseline within 48 h: blood pressure 129/68 mm Hg, oxygen saturation 95% on room air, WBC 9300 cells/uL, and hemoglobin 15.6 g/dL. Discharge diagnoses were dehydration and gastroenteritis. His lisinopril was continued. After discharge, the patient underwent endoscopy of his esophagus, stomach, and colon, but these were all normal. The patient had celiac studies performed by his primary care physician, but these were also normal.

On physical examination, he was alert, oriented, did not look toxic or in acute distress, but complained of mild abdominal discomfort. His mucous membranes were minimally dry. His lung sounds were clear to auscultation bilaterally. Cardiac examination revealed regular rate and rhythm without murmurs, rubs, or gallop. Peripheral pulses were strong and equal. His skin was warm and dry. His abdomen was mildly and diffusely tender without guarding, rebound, or organomegaly. Bowel sounds were diminished, but present. There was no leg tenderness, no peripheral edema, no jugular venous distention, and no rash. There were no focal neurologic deficits. Fecal occult test for blood was negative. The remainder of his physical examination was unremarkable.

A complete blood count showed a WBC count of 21,500 cells/uL (76% neutrophils), hemoglobin 20.4 g/dL, and platelets of 269,000/uL. His complete metabolic panel showed sodium 141 mmol/L, potassium 3.7 mmol/L, and bicarbonate 29 mmol/L, with an anion gap of 12 mmol/L, chloride 100 mmol/L, glucose 9.83 mmol/L (177 mg/dL), blood urea nitrogen 6.07 mmol/L (17 mg/dL), creatinine 0.12 mmol/L (1.4 mg/dL), total protein 7.1 g/dL, albumin 3.9 g/dL, aspartate transaminase 37 U/L, alanine transaminase 57 U/L, and lipase 186 U/L. Urinalysis showed a specific gravity of 1.017, and was negative for protein, glucose, ketones, bilirubin, blood, nitrite, or leukocyte esterase. Urine microscopy showed 1 WBC, no red blood cells and no bacteria. Kidney, ureter, and bladder x-ray study showed: "nonobstructive bowel gas pattern; gaseous distention of the stomach." Due to his initial hypotension, an abdominal computed tomography (CT) scan was ordered, which showed "distention of small bowel loops in the upper and mid abdomen with bowel wall thickening and surrounding inflammatory sign suggestive of infectious or inflammatory enteritis, no convincing evidence of bowel obstruction, and small volume of ascites."

After 2 h in the ED, the patient had received 2000 cc of normal saline, and his blood pressure was 94/37 mm Hg. His pulse was 65 beats/min and pulse oximetry had improved to 99% on 2 L/min oxygen by nasal cannula. He received ondansetron, which relieved his nausea, and he had no further episodes of vomiting or loose stools.

The patient was admitted with a presumptive diagnosis of ACEI-induced visceral angioedema. The patient underwent intravenous fluid hydration, cessation of his lisinopril, 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, 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