



Clinical Communications: Adults

ABDOMINAL ANAPHYLAXIS PRESENTING AS TRAUMA: A RECIPE FOR DELAYED DIAGNOSIS

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Abstract—Background: Successful shock management requires prompt identification, classification, and treatment; however, the triage of patients with non-hemorrhagic shock to the trauma room can lead to delayed diagnosis with increased morbidity and mortality. **Objective:** Our goal is to emphasize the importance of shock identification and classification to facilitate the delivery of the appropriate and timely therapy, no matter how the patient is triaged. **Case Report:** We describe a patient triaged as a trauma patient with suspected hemorrhagic shock yet who was found to have anaphylaxis as the etiology of his condition. Abdominal anaphylaxis, a less recognized presentation of anaphylaxis, is reviewed and discussed. **Conclusions:** We hope to increase awareness of a less common presentation of anaphylaxis and discuss its management. © 2010 Elsevier Inc.

Keywords—anaphylaxis; shock; abdominal pain; abdominal anaphylaxis; emergency department

INTRODUCTION

Successful shock management requires rapid recognition, accurate classification, and definitive treatment (1–4). Atypical presentations of common shock syndromes can lead to delay in life-saving therapies that can result in increased morbidity and mortality (1,3,4). This case report describes a patient who presented in shock after a motor vehicle collision, yet, despite the obvious distrac-

tions of the trauma room, was diagnosed with abdominal anaphylaxis after the likelihood of hemorrhagic, cardiogenic, and septic shock were rapidly and systematically ruled out. Important implications for all patients who present to the emergency department (ED) in shock will be discussed.

CASE REPORT

Emergency Medical Services was called for a 60-year-old man with a past medical history of hypertension who complained of feeling weak and light-headed a few hours after being struck from behind in a low-speed motor vehicle collision (MVC). After the collision, he was ambulatory on the scene and declined transport to the hospital. He stated that he felt fine and went to a friend's house for pizza. Thirty minutes after eating pizza and salad, he began to feel nauseated and vomited once. He had two syncopal episodes at his friend's house before an ambulance was summoned. On the scene, initial vital signs assessment revealed a blood pressure of 58/38 mm Hg, pulse 87 beats/min, respiratory rate 14 breaths/min, and 97% oxygen saturation on room air. Point-of-care blood glucose was 167 mg/dL. Because he was involved in a motor vehicle collision earlier in the day, the patient was brought by ambulance to a regional Level I trauma

center and designated a category I trauma based on his hypotension. En route, the patient was given 1.8 L of normal saline through a peripheral intravenous (i.v.) line and was started on a dopamine drip, a deviation from protocol, and it was discontinued upon arrival to the trauma room.

In the trauma room, the patient was afebrile with a blood pressure of 67/41 mm Hg, pulse of 112 beats/min, respiratory rate of 14 breaths/min, and 98% oxygen saturation on room air. The patient complained of “gassy pressure” in his left lower quadrant and that he still felt weak. He was extremely diaphoretic, cold and clammy to touch but did not have pruritis, urticaria, or angioedema. The patient was sleepy but arousable. Heart sounds were distant, distinct, and tachycardic with no murmurs. Breath sounds were clear and equal. The abdomen was obese, soft, non-tender, and mildly distended with no palpable pulsatile mass. Extremities were non-tender with normal range of motion. Strength was 5/5 in the upper and lower extremities and sensation was normal. Cranial nerve examination demonstrated his left eye deviating laterally; the patient stated he had a previous history of eye deviation. The remainder of the examination was normal and there were no signs of infection.

Primary and secondary surveys did not suggest an obvious cause for the hypotension. The bedside focused assessment with sonography for trauma (FAST) examination was negative for pericardial, lung, or intraperitoneal fluid. The subxiphoid and apical four-chamber views showed normal cardiac wall motion, no pericardial effusion, and no right ventricular overload, dilatation, or hypokinesis. An initial and second electrocardiogram showed normal sinus rhythm, normal intervals, and no signs of ischemia or pericardial disease. The patient’s hypotension did not improve despite receiving 5 L of normal saline. With no evidence for hypovolemic shock, cardiogenic shock (including ischemic, pericardial, or valvular heart disease [including obstruction from pulmonary embolism]), neurogenic shock, or septic shock, and given the patient’s poor response to fluid resuscitation, the emergency medicine team leader felt that anaphylaxis should be seriously considered as the cause of the patient’s hypotension, despite the absence of skin or visible mucosal involvement. It was felt that empiric therapy for anaphylaxis was more likely to improve the patient’s condition and less likely to be harmful than continued fluid resuscitation alone. The patient was given 0.5 mg epinephrine, 1:1000 intramuscularly in his lateral thigh; 125 mg methylprednisolone i.v. push, 300 mg cimetidine i.v. push, and 50 mg diphenhydramine i.v. push to treat possible anaphylaxis. After the medications given for suspected anaphylaxis, the patient’s blood pressure rose to 101/60 mm Hg and he became much more alert. With a stable blood pressure, the patient was trans-

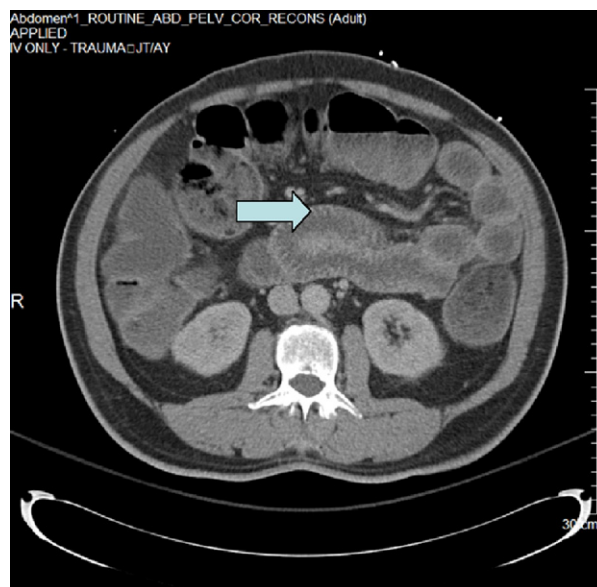


Figure 1. Computed tomography scan with intravenous contrast of the abdomen demonstrating fluid-filled dilated small bowel loops with mucosal wall thickening.

ported to the computed tomography (CT) suite. A CT scan without contrast of the head was negative for intracranial bleed. Abdominal CT with i.v. contrast showed fluid-filled dilated small bowel loops with mucosal wall thickening (Figure 1). The differential diagnosis of these findings included small bowel obstruction, enteritis, hypoproteinemia, anaphylaxis, and inflammatory bowel disease. Upon return to the trauma room from the CT scan, a nasogastric tube was placed, which drained approximately 1 L of brown, heme-occult negative fluid. The patient was admitted to the surgical intensive care unit (ICU) with suspected abdominal anaphylaxis.

DISCUSSION

Although the differential diagnosis of shock is broad after MVC-associated trauma, the most common cause of shock is hypovolemia secondary to hemorrhage (5). Yet, in this case, the mechanism of injury was not consistent with sufficient blunt force or deceleration injury to cause concealed hemorrhage. The bedside FAST examination and the abdominal CT scan did not show any free fluid. Due to the patient’s continued perfuse diaphoresis, cardiac ischemia was the next entity in our differential diagnosis. Two electrocardiograms, each performed in the trauma room before imaging, did not show any wave form abnormalities to suggest an acute coronary syndrome, and in the absence of chest pain, shortness of breath, or other anginal equivalents, a coronary syndrome seemed less likely. Possible obstructive causes

of shock in our patient included pulmonary embolism and cardiac tamponade. Of note, the patient was not tachycardic before the initiation of dopamine (and not on beta-blockers for his pre-existing hypertension), and oxygen saturations were 98–100% on room air in the ED, both findings inconsistent with massive pulmonary embolus causing hypotension. Although sepsis is always high on the differential diagnosis of shock, the patient denied recent illness or bacteremic-associated procedures, was afebrile, and did not have any source of infection on examination. With no history of a significant trauma mechanism, a normal neurologic examination, and a normal head CT scan, neurogenic shock was ruled out. Medication side effect as a cause for his shock was much less likely because there was no change in the patient's medications, no recent refill, and no herbal supplements being used by the patient. The patient was taking lisinopril for his essential hypertension. Toxic ingestion, medication side effect, and anaphylactic shock remained on the differential diagnosis. Due to the rapid resolution of hypotension, diaphoresis, and weakness after epinephrine administration, anaphylaxis became the most likely entity on our differential diagnosis. A serum tryptase level was added to the initial blood work and subsequently was found to be elevated at 17.6 (reference range: < 11.4). Tryptase values usually peak within an hour of the initial reaction. An elevated level is evidence that the patient experienced a systemic mast cell event, most likely anaphylaxis (6).

The patient was observed in the medical ICU overnight and remained normotensive for the remainder of his stay. A repeat CT scan with i.v. contrast the following day demonstrated interval resolution of the previously seen mucosal wall thickening and small bowel dilatation (Figure 2). He was discharged on hospital day 2 and advised to discontinue his lisinopril for the time being and follow-up with an allergist to investigate the cause of his anaphylaxis. Blood work as an outpatient revealed a normal serum tryptase and C1 esterase inhibitor function. Complement component, C2, and radioallergosorbent (RAST) testing to almond, apple, broccoli, cabbage, cashew, cat, celery, corn, garlic, lettuce, mustard, onion, parsley, pecan, peanut, and tomato were normal. An oral challenge totaling 162 mg of aspirin was negative. The patient's allergist at that time concluded that the most likely etiology for his anaphylactic reaction was lisinopril.

Three months later, the patient had another episode of severe hypotension. Immediately after eating, he went for a walk and started to experience diffuse urticaria 30 min into the walk. With this presentation, he did have a diffuse, red, urticarial rash but lost consciousness before administering his epinephrine auto-injector. Upon awakening, he informed the emergency medical technicians of his previous anaphylactic episode and was treated appropriately for anaphylaxis. Further RAST testing was done and did not reveal



Figure 2. The delayed images demonstrate interval resolution of the previously seen mucosal wall thickening and small bowel dilatation.

a definite cause for the anaphylaxis. Currently, the etiology for these episodes remains unclear. The patient carries an epinephrine auto-injector and does not drive within an hour of eating.

A recent case report described allergic angioedema of the bowel resulting in shock due to angiotensin-converting enzyme (ACE) inhibitors presenting in a similar manner as our patient (7). Angioedema and anaphylactoid reactions due to ACE inhibitors are known adverse effects (8).

Anaphylaxis (Greek, “away from protection”), is reported to occur at a rate of 50 to 2000 episodes per 100,000 people in the United States (9). It has been estimated to cause approximately 1500 deaths annually (10). The actual incidence is unknown. It is underreported due to inconsistencies in diagnosis and the lack of a standardized definition (11). Even though anaphylaxis is one of the most alarming disorders encountered in clinical medicine, first described approximately a century ago, there is no universal agreement on its definition or criteria for diagnosis (12). In addition, this lack of specific diagnostic criteria has greatly hampered management of this disorder; led to confusion on the part of the first responder, emergency physicians, and patients; and resulted in a failure to diagnose and treat anaphylaxis in a consistent manner. The most current consensus statement from the National Institute of Allergy and Infectious Disease and Food Allergy and Anaphylaxis Network defined anaphylaxis as “a serious allergic reaction that is rapid in onset and may cause death” and proposed diagnostic criteria to capture 95% of such patients, despite not being prospectively studied (Table 1) (12). The diagnostic criteria were developed with the knowledge that approximately 10–20% of the time, anaphylaxis presents in the

Table 1. Clinical Criteria for Diagnosing Anaphylaxis*

Anaphylaxis is highly likely when any one of the following three criteria is fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g., generalized hives, pruritus or flushing, swollen lips-tongue-uvula)

And at least one of the following:

- a. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
 - b. Reduced BP or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence)
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):
 - a. Involvement of the skin-mucosal tissue (e.g., generalized hives, itch-flush, swollen lips-tongue-uvula)
 - b. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
 - c. Reduced BP or associated symptoms (e.g., hypotonia [collapse], syncope, incontinence)
 - d. Persistent gastrointestinal symptoms (e.g., crampy abdominal pain, vomiting)
 3. Reduced BP after exposure to known allergen for that patient (minutes to several hours):
 - a. Infants and children: low systolic BP (age specific) or > 30% decrease in systolic BP*
 - b. Adults: systolic BP of < 90 mm Hg or > 30% decrease from that person's baseline

* Low systolic blood pressure for children is defined as < 70 mm Hg from 1 month to 1 year, < (70 mm Hg + [2 × age]) from 1 to 10 years, and < 90 mm Hg from 11 to 17 years.

PEF = peak expiratory flow; BP = blood pressure.

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absence of cutaneous symptoms (12–14). Gastrointestinal symptoms have been associated with severe outcomes in various anaphylactic reactions (15); rare patients present with an acute hypotensive episode after exposure to a known allergen (16).

We believe that our patient falls into the second proposed criterion for anaphylaxis, which describes exposure to a likely allergen with hypotension and persistent gastrointestinal symptoms. One possible explanation for our patient having profound hypotension without an obvious source is the phenomenon of food-dependent exercise-induced anaphylaxis (FDEIA). FDEIA occurs with exercise after ingestion of a specific allergen (17). Exercise lowers the threshold for mast cell degranulation (17). The exact mechanism for this is unclear. Our patient did not exercise the day of admission, but the trauma that occurred earlier in the day could have caused a similar release of catecholamines, mimicking the FDEIA syndrome. During his second episode of anaphylaxis, the patient was 30 min into a walk with his wife.

CONCLUSION

Successful shock management requires rapid recognition, accurate classification, and definitive treatment. Atypical presentations of common shock syndromes can lead to delays in life-saving therapy, resulting in increased morbidity and mortality.

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