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Acute Abdomen in the Emergency Department due to a Diaphragmatic Hernia with Gastric Strangulation and Perforation

Amanda K Young, MD*; David A Caro, MD

*Amanda K Young, MD

Dept. of Emergency Medicine, University of Florida College of Medicine-Jacksonville, USA Email: Amanda.Young@jax.ufl.edu

Abstract

A 65 year old woman presented to the emergency department with acute abdominal pain and rapid deterioration in the emergency department. She was found to have gastric strangulation and perforation due to diaphragmatic pathology. Clinical presentation features and epidemiology are discussed, along with suggested management of this rare combination of pathophysiology.

Keywords

diaphragmatic hernia; eventration; gastric; strangulation; perforation

Abbreviations

IV: intravenous; CT: computed tomography; GCS: Glasgow Coma Scale

Introduction

Diaphragmatic hernias occur most frequently in adults at the site of the esophageal hiatus, less frequently as a traumatic or iatrogenic lesion, and rarely through a previously undiagnosed congenital posterolateral (Bochdalek) or substernal (Morgagni) defect [1,2]. Studies have estimated the incidence of asymptomatic congenital hernias in the adult population from 1 in 2000-7000 to 6% [3,4]. Even more rarely, adults will present with symptomatic congenital diaphragmatic hernias [5]. One review of the literature noted that fewer than 100 cases of symptomatic Bochdalek hernias had been reported in adults as of 1994 [6]. Another noted that 5-25% of adult patients with congenital diaphragmatic hernia not diagnosed in the neonatal period will have chronic respiratory or gastrointestinal complaints [7,8]. Severe presentations of symptomatic congenital diaphragmatic hernias include incarcerated bowel and severe pulmonary disease [3,9-10]. However diaphragmatic hernia as a cause of intestinal obstruction and strangulation in adults is very rare.

Diaphragmatic eventration is also rare in adults [11,12], with one study estimating 37 patients out of 107,778 [13]. Eventration is a rare anomaly defined by a permanent elevation of a hemidiaphragm without defects caused by a disorder in which all or part of the diaphragm muscle is replaced by fibroelastic tissue [14-16].

Case Report

A 65 year-old African American woman presented to the Emergency Department with sudden onset of abdominal pain which began three hours prior to arrival, shortly after eating dinner. The patient

Vol 2: Issue 19: 1172

had no known medical history other than gastroesophageal reflux, and had no known history of significant trauma, though this history was not pursued in detail in the Emergency Department. She described the pain as severe and diffuse, but was worst in the suprapubic area. She described nausea and vomiting that was non-bloody and non-bilious. She had normal bowel movements with no reported diarrhea or constipation. She also reported feeling short of breath, but attributed this to the severity of her pain. Her review of systems was otherwise negative. On exam, she appeared to be uncomfortable and in moderate distress, but was awake and oriented. She was tachycardic to 150 beats per minute, tachypneic to 27 breaths per minute, and had an initial blood pressure of 74/56 mmHg. Her exam was significant for decreased breath sounds bilaterally at the bases, diffuse abdominal tenderness to palpation with voluntary guarding, a rectal temperature of 100.9 degrees Fahrenheit, guaiac negative stool, and cool distal extremities. 2 liters of normal saline were immediately bolused with improvement of systolic blood pressure to 90-100 mmHg. She was also given acetaminophen 1000 mg by mouth, ondansetron 4 mg IV, and morphine 6 mg IV. Initial results included an electrocardiogram with sinus tachycardia, a lactate of 9.6 mmol/L, and a chest X-ray with significant elevation of the left hemidiaphragm and rightward mediastinal shift (Figure 1), but no free air under the diaphragm. General surgery was immediately consulted regarding her concerning abdominal exam and unstable vital signs. A CT angiogram of the abdomen was ordered. The remainder of her labs results were significant for an elevated anion gap of 24 with a bicarbonate of 19 mmol/L, a potassium level of 2.8mmol/L, a bandemia of 27% with a white blood cell count of 8.4 thou/mm³, and a lipase of 107 U/L. The rest of her labs, including liver function tests, coagulation studies, electrolytes, and a complete blood count were within normal limits. Approximately one hour and 20 minutes after the patient's arrival, shortly after general surgery's initial assessment, the patient was found unresponsive, with a GCS of 3 and a rightward deviated gaze. Blood glucose remained within normal limits. The patient was intubated with etomidate 20mg IV and rocuronium 100mg IV. Postintubation the patient had worsening of her hypotension and was given more intravenous fluids. Her blood pressure did improve, however was persistently below normal. Because the patient was too unstable to transport to CT, a bedside ultrasound was performed and demonstrated a significant amount of free fluid in the abdomen. Despite continued intravenous fluids and two units of emergent packed red blood cells, the patient remained hypotensive and was rushed emergently to the operating room. During her first operation, she had an exploratory laparotomy which revealed the splenic flexure within the left thoracic cavity, a large superior and anterior defect or eventration of the diaphragm, and a perforated and ischemic greater curvature of the stomach. A partial gastrectomy was performed after reduction of the splenic flexure. The patient continued to have worsening acidosis, and the surgical team felt that damage control was the appropriate surgical approach. They therefore irrigated the abdomen copiously and then left the patient's abdomen open with a wound vacuum-assisted closure (VAC) in place. She was transferred to the surgical intensive care unit (ICU) for further stabilization. During her second operation, the surgeon noted high placement of the spleen and splenic flexure in the potential thoracic cavity, a possible hernia sac, and a loose and lax diaphragm without any defect noted at that time. While attempting to take the spleen down from its lateral attachment, significant bleeding was noted from the spleen, and so the surgeon elected to perform a splenectomy. The surgeon noted that mobilization of the splenic flexure via blunt dissection and electrocautery was very difficult due to the deep and superior location of the splenic flexure. The patient began to require increasing cardiorespiratory support, and as

Vol 2: Issue 19: 1172

repair of the diaphragmatic pathology was not emergent, further exploration was postponed. The patient's abdomen was again left open with a wound VAC in place and she was returned to the surgical ICU. During her third operation, a 5cm posterior defect of the diaphragm was ultimately noted and repaired without complication. The patient's abdomen was gradually closed after many washouts. She also ultimately required a left sided chest tube for thoracic contamination. She suffered post-operative complications including acute renal failure requiring hemodialysis, a tracheostomy for failure to wean from the ventilator, and sepsis due to ventilator associated pneumonia, intraabdominal abscesses, and bacteremia. The patient ultimately improved and was discharged no longer requiring hemodialysis or ventilator support, and had a Passy-Muir valve placed for her tracheostomy. She was able to tolerate oral feeds.

Discussion

Non-hiatal diaphragmatic hernias are rare, but importantly can contain intraabdominal contents which can become strangulated and even incarcerated [17]. These incarcerations with strangulation can present acutely with both gastrointestinal and respiratory symptoms, as well as abnormalities on early imaging such as an elevated hemidiaphragm. Gastrointestinal incarceration associated with non-hiatal diaphragmatic hernias can be seen in pregnancy [18-24], post-surgically [1,25-30], following trauma [1,31-68], or spontaneously through a pre-existing defect such as an undiagnosed congenital hernia [5,9-10,36-39,69-96]. Less commonly reported is a non-hiatal diaphragmatic hernia with strangulation of abdominal contents and viscus perforation [1-2,19-20,53-68,79-96]. Still even fewer of these cases have involved gastric perforation [1,20,58-68,89-96]. A review of the literature in adults found 42 cases of perforation associated with non-hiatal diaphragmatic hernias, 25 of which were gastric.

Diaphragmatic eventration is also rare in adults [11-12], one study noting approximately 1 in 3000 patients [13]. The etiology of diaphragmatic eventration may be congenital, traumatic, neurogenic (central or peripheral), atrophic, aplastic, hypoplastic, or infectious in nature [16,97-99]. In the pediatric population, patients typically present with respiratory distress or with recurrent respiratory infections, and also occasionally with gastrointestinal complaints such as regurgitation, failure to thrive, or feeding intolerance [99-101]. Adults are frequently asymptomatic if not diagnosed in childhood [11-13,103], and can present later in life with both respiratory and gastrointestinal complaints, as well as elevation of the affected hemidiaphragm on imaging studies [98,102]. These can be chronic symptoms that progress to an acute presentation, or sub acutely progressive symptoms [14,103-109]. Adults can also develop acquired eventration of the diaphragm, usually from trauma to the phrenic nerve [103-106]. Even more rarely, patients with diaphragmatic eventrations can have diaphragmatic hernias [17], intestinal obstruction due to the spontaneous rupture of a diaphragm eventration [110], or post-traumatic diaphragmatic tears associated with acquired eventration [111].

The patient in this case developed an acute abdomen from a gastric incarceration through a diaphragmatic defect with associated strangulation and perforation. The etiology of her diaphragmatic hernia and possible eventration are unclear. It is possible that she had a previously undiagnosed congenital diaphragmatic hernia, or that she had previously sustained a traumatic injury resulting in a diaphragmatic hernia. Given the posterior location of her diaphragmatic defect, it is certainly possible that this was a previously undiagnosed Bochdalek hernia. Regardless of the etiology, this case serves to

Vol 2: Issue 19: 1172

highlight the importance of maintaining a high index of suspicion of diaphragmatic pathology in patients who present with both gastrointestinal and respiratory symptoms as well as elevation of the diaphragm on chest X-Ray. These patients may have been previously asymptomatic, or may have had vague respiratory and/or gastrointestinal symptoms for years. In the case of gastrointestinal strangulation or perforation, these patients will present with new symptoms or acute worsening of their chronic symptoms. These patients will benefit from early imaging, including radiographs and computed tomography, if they are stable. Suspicion for diaphragmatic pathology should be increased if an elevated hemidiaphragm is noted on chest X-Ray. As unstable patients are not suitable to leave the emergency department for imaging, they will benefit from the use of bedside ultrasound. Bedside ultrasound is often more expeditious than other imaging modalities, and can potentially demonstrate evidence of life-threatening pathology such as the presence of free fluid as was the case with our patient. These patients require early surgical consultation as operative management is imperative and any delay in this increases the risk of further complications.

Figure



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Open J Clin Med Case Rep: Volume 2 (2016)

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Authors Information: Amanda K. Young, MD*; David A. Caro, MD

Dept. of Emergency Medicine, University of Florida College of Medicine-Jacksonville, USA

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