

An Uncommon Cause of Acute Abdominal Pain: Primary Epiploic Appendagitis in the Emergency Setting

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In the emergency setting, the diagnosis of benign causes of acute abdominal pain can prevent unnecessary medical interventions. To illustrate this point, we report the case of a 28-year-old man who presented to the emergency department with symptoms suggestive of acute diverticulitis. Abdominal computed tomography (CT) established, instead, a diagnosis of primary epiploic

appendagitis (PEA), which was managed expectantly. The patient's symptoms resolved within one week of hospital discharge and he remained free of pain at a five-month phone follow-up. Increased awareness of PEA and its self-limited course can help the emergency physician avoid unnecessary imaging studies and expectantly manage this cause of acute abdominal pain.

INTRODUCTION

Epiploic appendages are pedunculated pouches of subserosal fat, 1-2 cm in thickness and 0.5-5 cm in length, that line the external surface of the large intestine (Ross, 1950). These highly mobile structures have a limited blood supply and are susceptible to ischemia due to torsion or to *de novo* venous thrombosis of the vascular stalk anchoring them to the surface of the large intestine. The inflammation resulting from this ischemia precipitates epiploic appendagitis, which can be primary or secondary (Rioux & Langis, 1994; Singh, A.K. et al., 2005; Sand et al., 2007). Primary epiploic appendagitis (PEA) occurs spontaneously, while secondary epiploic appendagitis (SEA) is the result of a pre-existing abdominal inflammatory process occurring adjacent to the affected epiploic appendage (Vriesman et al., 2003; Rioux & Langis, 1994; Vinson, 1999). Pathophysiologically, PEA or SEA are characterized by a cascade of ischemia, infarction and aseptic fat necrosis that results in clinical symptoms. Due to the presence of epiploic appendages along the entire length of the large intestine, these clinical symptoms tend to be nonspecific and closely mimic other causes of abdominal pain (Legome, 2002; Lien et al., 2004; Rioux & Langis, 1994).

CASE PRESENTATION

We report a case of a 28-year-old male who presented to the emergency department with "sharp" left lower quadrant abdominal pain that began three days prior. Although the patient reported the pain to be 8/10, it was notable that he appeared to be in no acute distress. He denied nausea and vomiting, and denied changes to his appetite and bowel habits. His past medical history was noncontributory. Collected vital signs were as follows: oral temperature 98.5°F, heart rate 81 bpm, blood pressure 158/79 mmHg, respiration rate 18 respirations/minute and oxygen saturation 97% (room air). Physical examination revealed tender-

ness and guarding in the left lower quadrant. Urinalysis, complete blood count, comprehensive metabolic panel and serum amylase/lipase values were normal, except for a mild leukocytosis ($10.9 \times 10^3 /\text{mm}^3$). A contrast-enhanced abdominal computed tomography (CT) imaging study was obtained, which revealed a normal appendix and the absence of diverticula. Instead, CT images of the sigmoid colon demonstrated an ovoid, fatty structure with a dense rim that displayed mild fat stranding consistent with a diagnosis of PEA (Figure 1A, Figure 1B). Soon thereafter, the patient was discharged with pain medication for symptom management and was told to return to the emergency department if the pain worsened. During a phone follow-up conducted five months after his presentation to the emergency department, he reported that his abdominal pain resolved completely one week following hospital discharge and had not returned.

DISCUSSION

PEA was once considered a rare surgical diagnosis, but its true incidence has recently been called into question (Almeida, 2009). Before the widespread use of abdominal CT imaging, PEA was frequently misdiagnosed as diverticulitis because of its predominance in the rectosigmoid junction. As this misdiagnosis indicated medical, rather than surgical, management, the correct diagnosis of PEA was obscured and its true incidence falsely diminished (Ghahremani, 1992; Carmichael, 1985; Molla, 1998; Almeida, 1999; Rao, 1998). Today, abdominal CT and ultrasonography have made the diagnosis of PEA more frequent with reports demonstrating PEA to be the diagnosis in two to seven percent of cases of acute abdominal pain when diverticular disease was suspected, and in one percent of cases of acute abdominal pain when appendicitis was suspected (Vriesman, 2002; Zissin, 2002).

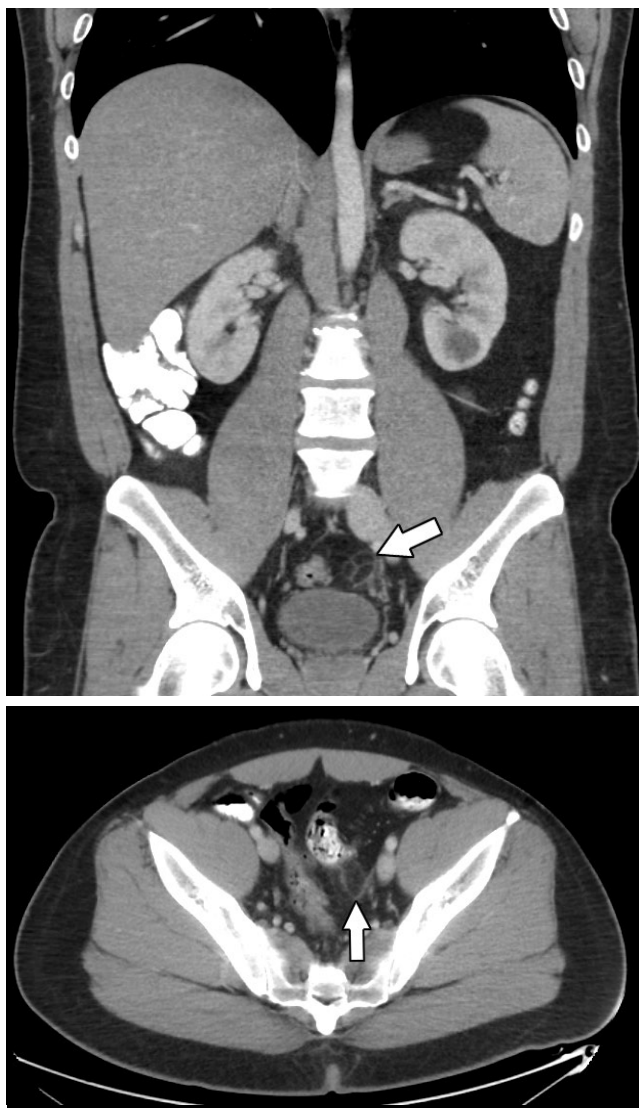


Figure 1 | (A) Axial CT image with hyperattenuating ring sign (arrow). (B) Frontal CT image with hyperattenuating ring sign (arrow).

The mean age of diagnosis of PEA is 40 years of age, and men may be more often affected by PEA than women (Sand et al., 2007; Jain et al., 2008; Rioux & Langis, 1994; Ozdemir et al., 2010; Hollerweger et al., 2002). Physical exertion or an extreme stretching movement of the abdomen has been reported to be a predisposing factor in the development of PEA (Ghahremani et al., 1992; Jennings & Collins, 1987; Ross, 1950; Rioux & Langis, 1994). Although epiploic appendages are present along the entire length of the large intestine, published reports suggest that 57% of PEA cases affect the rectosigmoid junction and 26% of cases affect the ileocecal region (Legome et al., 1999; Sangha et al., 2004; Hiller et al., 2000). An inflamed epiploic appendage in these locations produces a sharp, focal, non-migratory abdominal pain that can easily be mistaken for diverticulitis, appendicitis or acute cholecystitis (Boulanger et al., 2002; Schnedl et al., 2011). Patients are predominantly afebrile and do

not usually have gastrointestinal symptoms such as nausea, vomiting, anorexia or changes in bowel habits (Legome et al., 2002; Sangha, 2004). Physical examination is usually notable for focal tenderness localized over the site of inflammation (Rioux & Langis, 1994). Laboratory values are usually within normal limits, but a slight leukocytosis and a mildly elevated serum C-reactive protein may be seen (Rioux & Langis, 1994; Ozdemir et al., 2010; Sand et al., 2007; Ozkurt et al., 2007; Sandrasegaran, 2004).

The diagnosis of PEA relies on abdominal CT or ultrasonography—diagnosis based on symptoms alone is essentially impossible (Schnedl et al., 2011). PEA can be diagnosed on abdominal CT images by the “hyperattenuating ring sign” (Vriesman, 1999; Rioux & Langis, 1994). This sign, considered diagnostic for PEA, consists of an approximately 3 cm fatty, ovoid mass bound by a thick ring of hyperattenuation located near the colon (Figure 1A, Figure 1B). The ring may also contain a centralized hyperattenuating dot, presumably representing the thrombosed and necrotic vessel that once supplied the appendage. Evidence of fat stranding may also be seen in the vicinity of the lesion (Vriesman, 2003; Danielson et al., 1986).

The diagnostic finding of PEA on abdominal ultrasonography is the visualization of an ovoid, paracolic mass that is hyperechogenic and noncompressible, and is usually attached to the anterior parietal peritoneum (Rioux & Langis, 1994; Jennings et al., 1987; Hollerweger et al., 2002). The mass is often surrounded by a hypoechoic border, which corresponds to the hyperattenuating ring sign found on CT imaging (Vriesman, 2003; Rioux & Langis, 1994). Ultrasonography is particularly useful in the diagnosis of PEA because the sonographic findings can be directly correlated to the patient’s point of maximal tenderness and because the inflamed appendage’s adherence to the peritoneum can be easily demonstrated upon deep inspiration and expiration by the patient (Hollerweger et al., 2002).

PEA is usually a self-limited condition that can be managed expectantly with anti-inflammatory medications, or surgically by laparotomy (Apakama et al., 2011). Managed expectantly, symptoms generally resolve between three and 14 days, although future recurrences are possible (Fraser et al., 2009; Apakama et al., 2011). Patients should be counseled to return for surgical excision of the affected appendage if their symptoms persist, as this is considered the only definitive cure (Apakama et al., 2011; Rioux et al., 1994; Schwartz et al., 1994). Although rare, adverse outcomes of expectantly managed PEA have been reported and include abscess formation, bowel obstruction, intussusception, peritonitis and death (Romaniuk et al., 1993; Shamblin et al., 1986; Puppala et al., 1981; Murdie, 1953; Ghahremani, 1992; Apakama, 2011). Our patient presented with characteristic symptoms of PEA: a sharp, well-localized non-migratory pain in the left lower quadrant without additional gastrointestinal symptoms. Although initial imaging with ultrasonography would have been preferable, the discordance between this patient’s clinical presentation and his age raised our suspicion of a more insidious etiology,

and so an abdominal CT study was ordered. His symptoms resolved completely with expectant management within the time range reported in the literature and had not returned at a five-month phone follow-up.

While once thought of as a rare diagnosis, PEA should be part of the emergency physician's differential diagnosis in patients presenting with acute abdominal pain. The use of ultrasonography as an initial imaging method for these patients can help physicians rapidly diagnose PEA, avoid unnecessary further imaging and expectantly manage this benign condition successfully.

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