Review Article

Epiploic appendagitis: pathogenesis, clinical findings and imaging clues of a misdiagnosed mimicker

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Abstract: Primary epiploic appendagitis (PEA) is a rare and frequently underdiagnosed cause of acute abdominal pain. PEA most commonly affects obese, male patients in the 4th and 5th decade of life. Clinical presentation includes acute, localized, non-migrating pain without fever, nausea, vomiting or diarrhea and the laboratory workup is usually within normal limits. PEA is commonly mistaken as other more severe causes of acute abdominal pain, such as diverticulitis, acute appendicitis or cholecystitis and thus patients undergo unnecessary diagnostic and therapeutic procedures. The emergence of computerized tomography (CT) as the gold standard imaging test in diagnostic dilemmas of acute abdominal pain has resulted in increased recognition and diagnosis of PEA. Upon confirmation, PEA is considered a self-limiting disease and is managed conservatively with analgesics, occasionally combined with nonsteroidal anti-inflammatory drugs (NSAIDS). Persistence of symptoms or recurrence mandate the consideration of surgical management with laparoscopic appendage excision as the definitive treatment. We review the current literature of PEA, with a focus on clinical and imaging findings, in order to raise awareness about this frequently misdiagnosed entity.

Keywords: Epiploic appendagitis; appendicitis epiploica; acute abdomen

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Introduction

Epiploic appendagitis is a rare cause of acute abdominal pain occurring predominantly in males in the fourth and fifth decade of life with an incidence of approximately 8.8 cases/10^6 population/year (1,2). Nevertheless, cases have been reported in children, even at the age of 5, as well as in the elderly population (3,4). Primary epiploic appendagitis (PEA) is the result of torsion of the appendage or thrombosis of the draining vein that cause ischemic necrosis and subsequent inflammation of the affected epiploic appendage (5-7).

PEA has been reported to occur more frequently in obese patients (8,9). Nugent *et al.*, in a retrospective case control study involving patients with PEA and patients presenting with other causes of acute abdomen, reported that PEA subgroup had 60% greater abdominal adipose volume and 117% increased visceral adipose area (10). Other factors associated with PEA include intensive strenuous exercise and presence of hernia (8,9,11,12).

PEA is frequently misdiagnosed and commonly a mimicker of other serious causes of acute abdomen (13,14).

Previously, it was considered a surgically treated disease and was most frequently diagnosed at surgery, but currently is treated conservatively with avoidance of unnecessary surgical interventions (1,7,11,15,16). Newer diagnostic imaging modalities have played an important role in the establishment of conservative therapy as the appropriate choice in patients with PEA (14,17,18). Computerized tomography (CT) widespread use in the last years and its use as gold standard imaging test in diagnostic dilemmas of patients with acute abdominal pain has resulted in increased recognition and diagnosis of PEA (11,17). Radiologists and surgeons should be aware of the typical imaging findings of PEA in order to accurately diagnose this entity and avoid further non-indicated pharmaceutical or surgical management (1,19-21).

Anatomy

Epiploic appendages consist of pedunculated fat tissue attached to the colonic surface, most commonly located on the taeniae (taenia libera and taenia omentalis) of the cecum and sigmoid colon (22,23). They are approximately 50 to 100 and form two lines along the colonic surface with the exception of the transverse colon, where only one row exists due to attachment of the greater omentum to taenia omentalis (7,11,15-17). These outpouchings are covered by serosa, supplied by one or two arteries and one draining vein and their length varies between 0.5–5 cm (11,23,24). Epiploic appendages first develop during the fifth month of intrauterine life and their size remains small during childhood (7,15,25). They get enlarged during adulthood and this size increase is augmented in obese patients, thus PEA is more frequently diagnosed in obese adults (7,15,25). The exact functional role of epiploic appendages is not well-understood and many different physiological roles have been proposed (17,24,26). It has been theorized that these fat projections act as cushions providing mechanical protection or as a storage of blood supply during peristalsis and colonic vessels compression (24,26). Other theories include the role as a fat storage utilized in periods of decreased caloric intake—starvation or immune protection and defensive mechanisms against inflammation, a role similar to that mediated by greater omentum (17,24,26).

Pathogenesis

Epiploic appendages, in the context of their anatomic structure of a bulbous protrusion connected to a narrow peduncle, undergo torsion with consequent vascular supply impairment, initially affecting the venous component (11,15,27). Ischemia of the epiploic appendages may also occur as a result of draining vein thrombosis (5,6). Both conditions lead to edema, ischemic necrosis, aseptic inflammation of the affected appendage and eventually absorption by the peritoneal cavity (5-7,15). Nevertheless, Virchow was the first to suggest that detached loose intraperitoneal bodies represent detached epiploic appendages (28). In the era of laparoscopic surgery and radiographic diagnosis, calcified detached epiploic appendages can be identified as peritoneal loose bodies, also known as 'peritoneal mice' (29,30).

PEA should always be differentiated from secondary epiploic appendagitis (SEA), an entity that results from a different pathophysiologic mechanism. SEA involves inflammation of a normal epiploic appendage located in proximity to an inflamed organ, such as colon (diverticulitis), appendix (appendicitis) or gallbladder (cholecystitis) (15,31,32). The most frequent source of inflammation in SEA is diverticulitis and pathognomonic signs of the adjacent organ disease are evident in diagnostic imaging modalities, such as CT (31,32).

Clinical and laboratory findings

The clinical presentation of PEA is vague and similar to those caused by other acute conditions such as acute appendicitis (right lower quadrant abdominal pain), acute diverticulitis (left lower quadrant abdominal pain) and acute cholecystitis (right upper quadrant abdominal pain) (*Tables 1*,*2*) (6,9,11,22,33,34). PEA most commonly manifests with acute onset, non-migrating lower abdominal pain, most commonly on the left, with localized tenderness on abdominal palpation and rebound tenderness in some occasions (2,11,24,35). Fever, nausea, vomiting, diarrhea and constipation are sometimes associated with PEA, but are usually absent (2,8,35). Mollà *et al.* demonstrated that approximately 7% of patients undergoing investigation to exclude the initial presumptive clinical diagnosis of acute diverticulitis of the sigmoid colon showed imaging findings of PEA (23). Choi *et al.*, in their retrospective case series study of 31 patients with PEA, reported abdominal tenderness (100%) and right or left lower quadrant abdominal pain (41.9% and 41.9%, respectively) as the most common presenting symptoms in their cohort, whereas muscle rigidity and fever were absent in all patients (22). They also reported that patients with sigmoid

Table 1 Summary of PEA demographic, clinical and laboratory findings

Demographics
Obese, male, in 4th or 5th decade
Clinical findings
Pain: acute, non-migrating, most commonly located in LLQ (other locations: RLQ, RUQ)
Rebound tenderness (occasional)
Other symptoms (fever, nausea, vomiting, bowel habit changes) usually absent
Laboratory findings
Routine workup usually within normal limits
WBC elevation
Increased CRP

LLQ, left lower quadrant; RLQ, right lower quadrant; RUQ, right upper quadrant; WBC, white blood cell count.

Table 2 PEA differential diagnosis from mimicking conditions

Acute diverticulitis (LLQ)
Acute appendicitis (RLQ)
Acute cholecystitis (RUQ)
Pelvic inflammatory disease (Lower abdomen/Pelvis)
Ovarian torsion (RLQ or LLQ)
Ectopic pregnancy (RLQ or LLQ)
Ureter colic (RLA or LLA pain associated with flank pain)
Mesenteric lymphadenitis (RLQ)
Acute omental infarction (RLQ or RUQ)
Mesenteric panniculitis (Variable)

LLQ, left lower quadrant; RLQ, right lower quadrant; RUQ, right upper quadrant; RLA, right lateral abdomen; LLA, left lateral abdomen.

diverticulitis were older than patients with PEA affecting sigmoid colon appendages (69.7 *vs.* 41.4 respectively, P<0.001) (22). Other conditions, presenting with symptoms similar to PEA, include pelvic inflammatory disease, ovarian torsion, ectopic pregnancy, mesenteric lymphadenitis, acute omental infarction, mesenteric panniculitis and ureter stone (19,22,33). Laboratory evaluation in patients with PEA is usually within normal limits and the findings, if present, are mostly non-specific (*Table 1*) (8). In most cases erythrocyte sedimentation rate, liver transaminases, pancreatic amylase and lipase and urinalysis are within normal limits (5,15,18). Infrequently, a slightly elevated white blood cell count (WBC) and C-reactive protein, as a result of ischemic fat necrosis induced inflammatory response, may be observed (6,24,35).

Imaging studies

The lack of specific pathognomonic clinical and laboratory findings as well as absence of awareness among physicians renders PEA diagnosis difficult without the use of imaging modalities (2,33). Prior to establishment of newer diagnostic techniques, PEA was a diagnosis considered after the exclusion of more common diseases and was identified during diagnostic laparotomy or laparoscopy (5,15,36).

Currently, in the era of widespread use of CT scans in the differentiation of acute abdominal pain, PEA is easily identified in the presence of pathognomonic imaging findings and its reported incidence is increasing (*Table 3*) (22). The hallmark of PEA in CT consists of an ovoid mass measuring between 1.5 and 3.5 cm in maximal

Table 3 PEA most common imaging findings in CT, U/S and MRI

CT
Ovoid mass with hyperdense ring
Thickened parietal peritoneum
Hyperdense thrombosed draining vein (“central dot” sign)
Bowel wall thickening
Mass location anterior to the colonic wall
U/S
Hyperechoic, non-compressible mass
Hypoechoic peripheral rim (inflamed serosal surface)
Absence of mass blood flow in Doppler
Hyperechoic adjacent fat tissue
Mass located adjacent to the colonic surface
Mass attachment to the anterior abdominal wall
MRI
Oval mass with fat tissue signal intensity (T1 and T2)
Ring enhancement with gadolinium (T1)

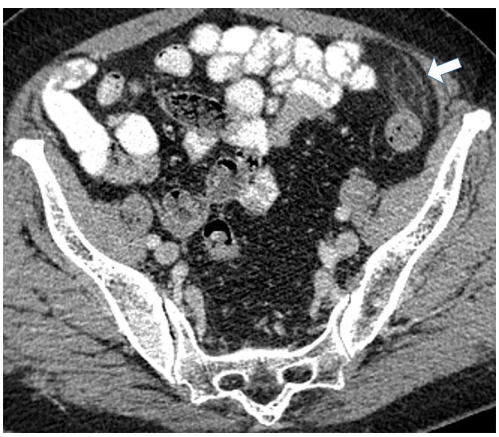

Figure 1 Axial MDCT depicts an ovoid mass (white arrow) with maximal diameter of 3.4 cm with fat attenuation, surrounded by a hyperdense ring corresponding to the inflammatory reaction of the overlying visceral peritoneum anterior to the distal part of the descending colon, indicative of epiploic appendagitis.

diameter with fat attenuation, surrounded by a hyperdense ring corresponding to the inflammatory reaction of the overlying serosa (visceral peritoneum) (*Figure 1*) (6,11). Frequently, the serosal surface spreads the inflammation through attachment to the parietal peritoneum, which may have a thickened appearance (*Figure 2*) (11,37). The occasional presence of a centrally located hyperdense area corresponds to the draining vein thrombosis (‘central dot sign’) (*Figure 3*) (6,8,11,37). Additionally, the anterior relationship of the inflamed epiploic appendage to the colonic wall is a useful anatomic imaging clue in order to confirm PEA diagnosis (*Figure 4*) (38). Nugent *et al.* reported that the ovoid mass with hyperattenuating ring (100%), the central hyperdense dot sign (79%), peritoneal thickening (76%), bowel wall thickening (*Figure 5*) (47%), presence of diverticula (28%) and free fluid were the most common imaging findings of PEA (10).

Ultrasound (U/S) at the site of maximal tenderness reveals an oval hyperechoic, non-compressible mass adjacent to the colonic surface (8,11,18,23,39). Doppler images reveal absence of central blood flow and the mass may be surrounded by a hypoechoic peripheral rim corresponding to thickened—inflamed serosal surface (7,8,12,18,24). The adjacent fat tissue may present with increased echogenicity and color Doppler signal in the context of inflammation induced increase in blood flow (7,15). In addition, U/S easily identifies the fixed attachment of inflamed appendages to the anterior abdominal wall during breathing movements (7,23,40). U/S is a rapid noninvasive imaging diagnostic test, usually helpful in the diagnosis of PEA in

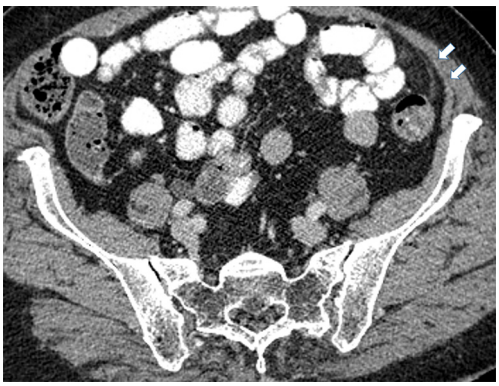

Figure 2 Axial MDCT reveals epiploic appendagitis with minimal thickening of the adjacent parietal peritoneum (white arrows).

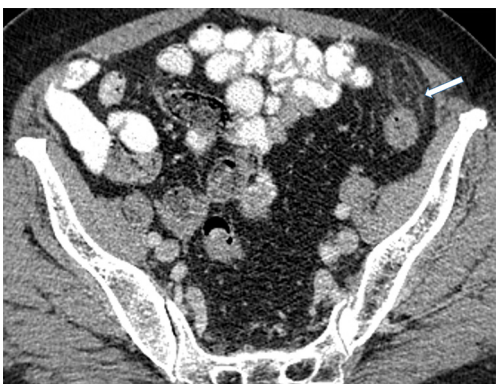

Figure 3 The 'central dot sign' of primary epiploic appendagitis (PEA). Axial CT (prior to intravenous contrast administration) images a small hyperdense dot (white arrow) within the inflamed epiploic appendage corresponding to draining vein thrombosis ("central dot" sign).

non-obese patients or those who have contraindications to CT radiation exposure, such as pregnant women (18).

Magnetic resonance imaging (MRI) is not a routinely used imaging test in the diagnosis of PEA, but can be used in pediatric patients and pregnants (41-43). Advantages of MRI include absence of ionizing radiation and higher soft tissue resolution in contrast to CT. Imaging findings include the presence of an oval mass with fat tissue signal intensity in T1 and T2 weighted MRI images and ring enhancement in contrast agent (gadolinium) enhanced T1 weighted imaging (11,15,41).

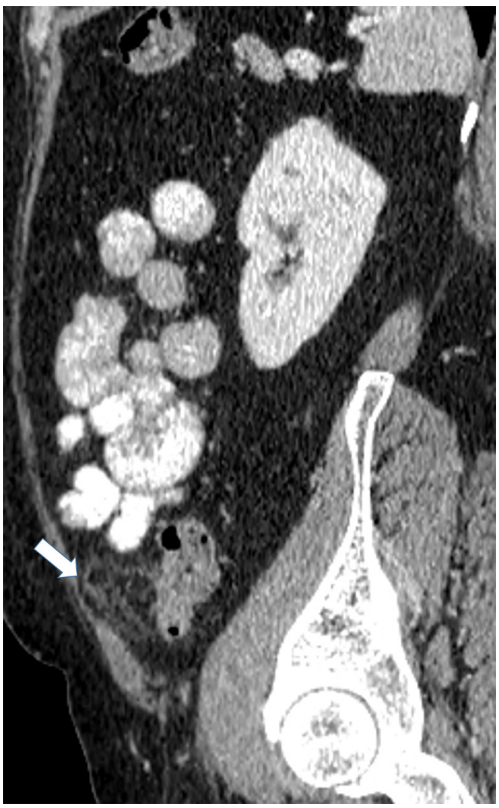

Figure 4 Sagittal maximum intensity projection (MIP)-MDCT reconstruction shows the anterior relationship of the inflamed epiploic appendage to the colonic wall (white arrow).

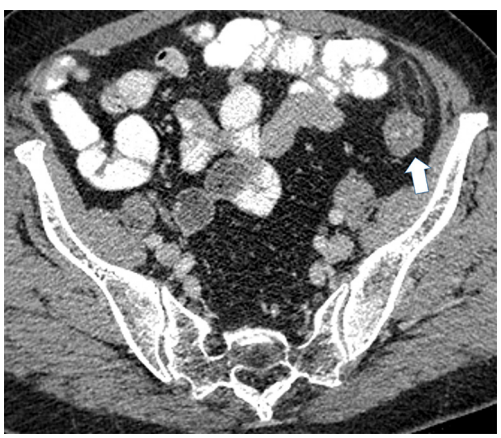

Figure 5 Bowel wall thickening in primary epiploic appendagitis (PEA). Axial MDCT demonstrates a small degree of anterior descending colonic wall thickening (white arrow), adjacent to the inflamed epiploic appendage.

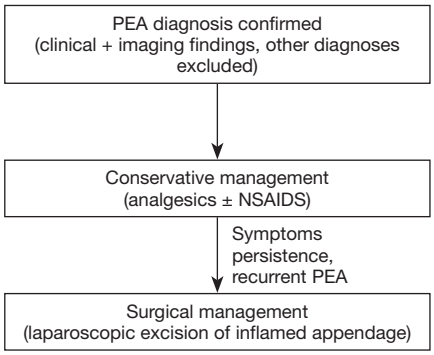

Figure 6 PEA proposed treatment algorithm. PEA, primary epiploic appendagitis; NSAIDS, non-steroidal anti-inflammatory drugs.

Treatment

Prior to the widespread use of newer imaging diagnostic modalities, PEA was considered a surgical disease and was usually diagnosed and treated during operations performed to exclude more severe cause of acute abdomen (1,7,15). Currently, PEA is generally considered a self-limiting disease and conservative management with or without NSAIDS is the first choice of treatment, resulting in disappearance of symptoms within several days, but is associated with a high rate of recurrence (2,8,12,24,33,40). Antibiotic usage has been proposed as an adjunct to anti-inflammatory medications, but their therapeutic benefit is not established (11,15,16,28). Nevertheless, if conservative approach fails to alleviate symptomatology, laparoscopic excision of the affected appendage may be required (8,15,24). Most patients treated conservatively show resolution of symptoms within 1–2 weeks, but the CT findings may persist and subside in a slower fashion (7,15,44). Furthermore, CT pathognomonic changes may persist for up to 6 months and physicians should be aware of the long-term imaging residual findings, in order to avoid misdiagnosis of patients presenting with acute abdominal pain due to other causes (38). PEA complications include abscess development and gastrointestinal obstruction; thus patients are suggested to immediately seek medical evaluation upon worsening of post-discharge clinical status (35).

Conclusions

PEA is an uncommon and frequently misdiagnosed cause of acute abdominal pain in patients presenting in the emergency department. Widespread CT usage has resulted in increased recognition and diagnosis of PEA. Upon establishment of diagnosis, PEA patients are treated with analgesics. Persistence of symptoms or recurrent episodes are treated with laparoscopic appendage excision. Surgeons and emergency department physicians should be aware of this frequently underdiagnosed entity.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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