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Acute epiploic appendagitis: a case report and literature review

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Abstract
Acute epiploic appendagitis (AEA) is a rare cause of acute abdomen due to ischemic inflammation of an epiploic appendage, often from torsion or venous thrombosis. We report the case of an elderly woman with acute left lower quadrant abdominal pain, initially suspected of having an abdominal abscess. Clinical examination showed localized tenderness without fever, with mild leukocytosis and elevated C-reactive protein. Ultrasound revealed an oval hyperechoic mass, confirmed by computed tomography (CT) as a hyperdense lesion with a central fatty core, consistent with AEA. Conservative management with non-steroidal anti-inflammatory drugs led to resolution within 7 days. The literature review highlights an incidence of 0.8-2.5% in acute abdomens, with a predilection for obese adults. Diagnosis relies on clinical and imaging findings, with CT as the gold standard. Differential diagnosis includes diverticulitis, appendicitis, and abscesses. An integrated clinical-instrumental approach is essential to avoid unnecessary surgical interventions.

Introduction

Acute abdomen poses a complex diagnostic challenge, with etiologies ranging from benign conditions to potentially life-threatening diseases.[1] Among less common causes, acute epiploic appendagitis (AEA) stands out as a rare entity, often misdiagnosed due to its nonspecific presentation mimicking appendicitis, diverticulitis, or abdominal abscesses.[2] Epiploic appendages are pedunculated adipose structures along the colonic serosa, with hypothesized roles in mechanical protection and lipid metabolism.[3] AEA primarily results from spontaneous torsion of the vascular pedicle or venous thrombosis, leading to local ischemia, edema, and peritoneal inflammation.[4] Epidemiologically, AEA accounts for 0.8-2.5% of acute abdomen cases evaluated by computed tomography (CT), with a predilection for middle-aged adults and obesity as a risk factor.[5] Diagnosis relies on an integrated clinical-laboratory and instrumental approach, with ultrasound (US), CT, and, in selected cases, magnetic resonance imaging (MRI) as key tools.[6] This case report describes an AEA case in an elderly woman, with a literature review emphasizing etiology, epidemiology, diagnostics, and differential diagnosis, highlighting the critical role of clinical-instrumental diagnosis in avoiding unnecessary invasive treatment.[7]

Case Report

A 74-year-old woman with a body mass index (BMI) of 30 kg/m^2 presented to the emergency department complaining of acute left lower quadrant abdominal pain, onset 36 hours prior, without radiation. She reported no fever, nausea, vomiting, or bowel changes, and her medical history was positive only for pharmacologically controlled hypertension. On physical examination, localized tenderness was noted without signs of peritonism, with no palpable mass and preserved peristalsis. Laboratory tests showed mild leukocytosis (WBC 11.5×10^9/L) and moderately elevated C-reactive protein (CRP) (40 mg/L), with no other abnormalities.

Initially, the diagnostic suspicion was an abdominal abscess, given the patient's advanced age and pain localization. An abdominal US, performed as the first approach, revealed a 3.4 cm oval hyperechoic mass in the left lower quadrant, with a hypoechoic rim and perilesional fat stranding, suggestive of AEA. To confirm the diagnosis and rule out an abscess, a contrast-enhanced abdominal CT was performed, showing a hyperdense lesion (38 HU) with a central fatty core and mild perilesional stranding, consistent with primary AEA in the sigmoid colon, refuting the abscess suspicion due to the absence of fluid collections with capsular enhancement. MRI was not required, given the clarity of CT findings (Figure 1).

The differential diagnosis included acute diverticulitis, appendicitis, omental infarction, and abdominal abscesses, but the absence of colonic wall thickening, appendiceal dilation, or fluid collections ruled out these conditions. The patient was treated with ibuprofen (400 mg every 8 hours) and clinical observation, with complete symptom resolution within 7 days. A 14-day follow-up US was negative, and the patient reported no recurrences at 3 months.

Discussion

AEA is primarily caused by spontaneous torsion of the vascular pedicle of epiploic appendages, leading to local ischemia, edema, and peritoneal inflammation.[8] Spontaneous venous thrombosis, associated with hypercoagulability or adjacent inflammation, contributes to approximately 15% of cases.[9] Risk factors include visceral obesity (odds ratio 2.8), which increases appendage mobility, and abdominal microtrauma, reported in 20-30% of patients.[10] In our case, the patient's elevated BMI (30 kg/m^2) supports the predisposing role of visceral fat, despite her advanced age being less typical.[11] AEA incidence ranges from 0.8 to 2.5% in patients with acute abdomen evaluated by CT, with a peak between 40 and 50 years and a male predominance of 60-70%.[12] However, cases in elderly women, as in our report, are not rare, especially with obesity.[13] The sigmoid colon is the most common site (86%), as seen in our patient, followed by cecal (7%) and transverse (7%) locations.[14] Recent studies (2022-2025) report increased diagnosis due to routine CT use, with spontaneous resolution in 95% of cases within 7-10 days.[15] Table 1 summarizes epidemiological data.

AEA's clinical presentation is characterized by localized acute abdominal pain (90% of cases), often without fever (80%) or marked systemic symptoms, as observed in our patient.[1] Laboratory findings show mild-to-moderate leukocytosis (WBC 10-15×10^9/L in 40%) and elevated CRP (<50 mg/L in 60%), consistent with our patient's values (WBC 11.5×10^9/L, CRP 40 mg/L).[2] These findings, though nonspecific, are critical for guiding diagnostic suspicion but require instrumental confirmation to avoid errors, as seen with the initial abscess suspicion in our case.[3]

Abdominal US is the first-line, non-invasive diagnostic approach, with 80-90% sensitivity in detecting an oval hyperechoic mass with a hypoechoic rim, as found in our patient.[4] Contrast-enhanced CT is the gold standard (100% sensitivity, 98% specificity), showing a hyperdense lesion with a central fatty core, as confirmed in our case, ruling out an abscess due to the absence of fluid collections.[5] MRI, useful in pregnancy or complex cases, shows T2 hyperintensity with peripheral enhancement (95% sensitivity), but was not needed in our case.[6] The "central dot sign", a CT finding characterized by a central hyperdense dot representing the thrombosed vessel within the lesion, is considered pathognomonic for AEA and a key diagnostic element, though not reported in this case.[7] Table 2 summarizes imaging findings.

AEA must be differentiated from diverticulitis (colonic wall thickening >4 mm), appendicitis (appendiceal dilation >6 mm), omental infarction (diffuse mesenteric stranding), abdominal abscesses (fluid collections with capsular enhancement), and neoplasms (irregular masses).[8] In our case, the initial suspicion of an abscess was refuted by CT, which ruled out fluid collections and confirmed AEA.[9] MRI can be decisive for distinguishing malignant processes, but was not needed.[10]

Conclusions

This AEA case in an elderly woman highlights the importance of an integrated diagnostic approach to identify rare causes of acute abdomen, which must not be overlooked in patient management.[11] The nonspecific clinical presentation, with localized pain and mild inflammatory marker changes, initially suggested an abscess, but CT confirmed AEA, avoiding unnecessary interventions.[12] The differential diagnosis, including appendicitis, diverticulitis, abscesses, and omental infarction, underscores the critical role of imaging, with US as a screening tool and CT as the gold standard.[13] Conservative management with non-steroidal anti-inflammatory drugs led to complete resolution, consistent with literature reporting favorable outcomes in 95% of cases.[14] Overlooking AEA among acute abdomen causes, even in less common populations like the elderly, must be avoided to promote standardized diagnostic protocols, reducing iatrogenic morbidity and optimizing clinical management.[15]

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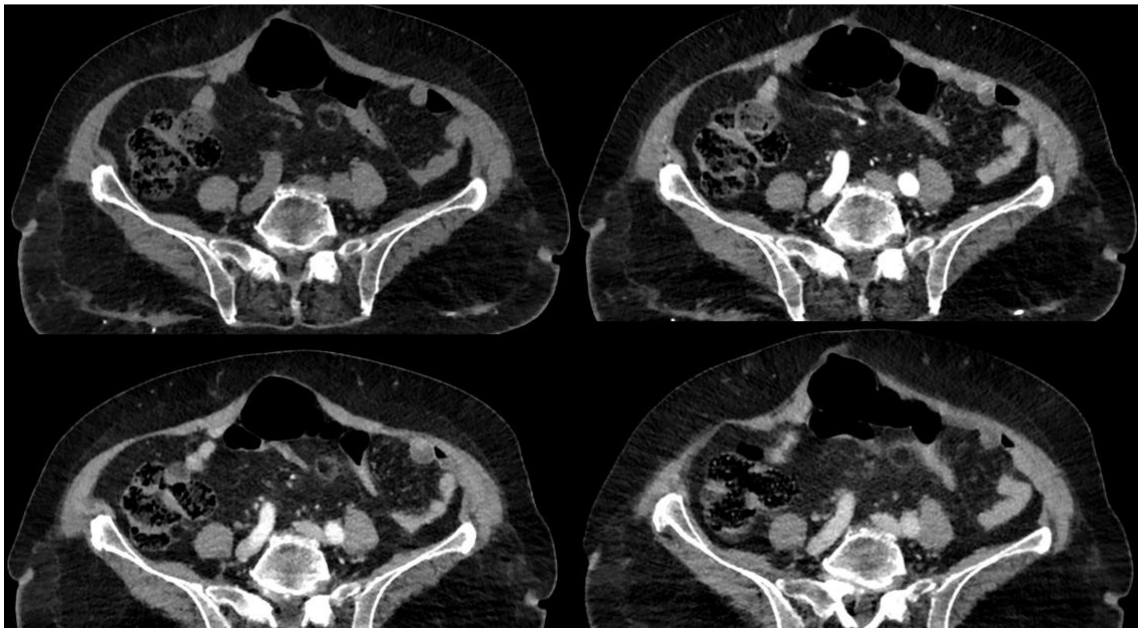

Figure 1. In the left paracolic region, adjacent to the descending colon, there is an oval-shaped lesion with well-defined margins, showing central fatty density and a thin peripheral hyperdense rim. Mild stranding of the adjacent perivisceral fat is also observed.

Table 1. Epidemiological characteristics of acute epiploic appendagitis.

Parameter	Estimated value	Notes (references)
Incidence (% acute abdomens)	0.8-2.5%	Increased with routine CT (12)
Mean age (years)	40-50	Rare in elderly (13)
M:F ratio	2.4:1	Male predominance (14)
Primary location	Sigmoid (86%)	Cecal 7 %, transverse 7% (15)
Obesity risk factor	OR 2.8 (BMI>30)	Visceral fat correlation (10)

M:F, male:female; OR, odds ratio; BMI, body mass index; CT, computed tomography.

Table 2. Instrumental diagnostic findings in acute epiploic appendagitis.

Imaging modality	Characteristic findings	Sensitivity/specificity (%)	Clinical role (references)
Ultrasound	Oval hyperechoic mass with hypoechoic rim	80-90/85	First-line, non-invasive (4)
Computed tomography	Oval lesion 2–5 cm, central fatty core	100/98	Gold standard, rules out abscesses (5)
Magnetic resonance imaging	T2 hyperintensity, rim enhancement	95/95	Selected cases, pregnancy (6)