

Diagnostic Challenges of Epiploic Appendagitis

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Summary

Introduction. Epiploic appendagitis is a rare and often misdiagnosed cause of acute abdominal pain. It can be primary or secondary. Epiploic appendagitis often mimics other acute abdominal conditions, such as appendicitis or diverticulitis, which typically require surgical intervention. Imaging techniques play a crucial role in accurately identifying epiploic appendagitis, differentiating it from more common pathologies, and preventing misdiagnosis. A precise diagnosis is essential for selecting the appropriate treatment strategy for patients.

Objective. This review aims to highlight the diagnostic challenges of epiploic appendagitis and emphasize the role

of imaging, particularly CT and ultrasound, in differentiating it from other causes of abdominal pain.

Methods. A narrative literature review was conducted. Data and information were collected from the PubMed and Google Scholar databases using the following keywords: epiploic appendagitis, abdominal pain, acute abdomen, differential diagnosis, diverticulitis, omental infarction, mesenteric panniculitis, and appendicitis. A total of 28 articles were selected that met the study objectives.

Conclusions. CT imaging is the gold standard for diagnosing epiploic appendagitis, but ultrasound also provides important information. Differential diagnosis of epiploic appendagitis most often include the diseases, that show plenty fat stranding with minimal bowel wall thickening. These diseases are: diverticulitis, appendicitis omental infarction and mesenteric panniculitis. Key CT features, that are very important in differentiation, include an ovoid fat-density lesion with a hyperattenuating ring and central dot sign, while ultrasound shows a hyperechoic, non-compressible oval mass circumscribed by a hypoechoic line with the absence of bowel wall involvement and absence of signal on color Doppler imaging.

Keywords: epiploic appendagitis, abdominal pain, acute abdomen, differential diagnosis, diverticulitis, omental infarction, mesenteric panniculitis, appendicitis.

INTRODUCTION

Acute epiploic appendagitis represents an uncommon and frequently misdiagnosed etiology of acute abdominal pain. The condition arises from either primary inflammatory or ischemic processes affecting the epiploic appendages, or secondarily from adjacent inflammatory pathologies. Accurate diagnosis carries significant clinical implications for patient management.

This entity frequently mimics other acute abdominal conditions that typically warrant surgical consultation, particularly acute appendicitis and diverticulitis. Diagnostic imaging is fundamental in correctly identifying epiploic appendagitis, enabling appropriate differential diagnosis and preventing clinically significant misdiagnosis.

The purpose of this review is to highlight the diagnostic challenges of epiploic appendagitis and emphasize the role of imaging—particularly CT and ultrasonography—in differentiating it from other causes of abdominal pain.

METHODS

A narrative literature review was conducted. Data and information were collected from the PubMed, Google Scholar databases using the following keywords: epiploic appendagitis, abdominal pain, acute abdomen, differential diagnosis, diverticulitis, omental infarction, mesenteric panniculitis, and appendicitis. A total of 28 articles were selected that met the study objectives.

LITERATURE REVIEW

Normal epiploic appendages

Epiploic appendages are small, pedunculated, fat-containing serosal outpouchings distributed along the external surface of the colon. They are typically arranged in two parallel longitudinal rows extending from the cecum to the rectosigmoid junction, corresponding to the taenia libera anteriorly and the taenia omentalis posterolaterally [1]. The

average adult possesses approximately 50–100 of these structures. These appendages generally measure 1–2 cm in thickness and 2–5 cm in length, although dimensions of up to 10 cm have been documented [2]. The appendages adjacent to the sigmoid colon are characteristically the largest and may occur in clusters [3]. The size of epiploic appendages is also positively correlated with body adiposity and may be accentuated in obese individuals or those with recent weight loss [4]. The vascular supply to each appendage consists of dual arteries with a solitary draining vein. Due to their limited blood supply, pedunculated structure and, mobility epiploic appendages are susceptible to torsion, which can lead to ischemia or hemorrhage, causing epiploic appendagitis [5]. The exact function of epiploic appendages remains unclear, but they are believed to cushion the colon for mechanical protection, store blood during peristalsis and vessel compression, and support blood supply. They may also serve as fat reserves during starvation and contribute to immune defense, similar to the greater omentum [6].

The normal epiploic appendages remain radiographically occult on computed tomography (CT) due to isodensity with adjacent peritoneal and omental adipose tissue. These structures become radiographically apparent only in pathologic states characterized by inflammation or when delineated by surrounding ascitic fluid [2].

Definition and types of epiploic appendagitis

Epiploic appendagitis is an uncommon cause of acute abdominal pain, resulting from a benign, self-limiting inflammatory or ischemic condition affecting the epiploic appendages [2]. There are two types of epiploic appendagitis – primary and secondary. The primary epiploic appendagitis 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, manifesting as epiploic appendagitis on imaging. In comparison, secondary epiploic appendagitis develops in response to inflammation in nearby organs, particularly in cases of diverticulitis, appendicitis, pancreatitis, or cholecystitis [7]. Secondary epiploic appendagitis most commonly results from contiguous spread of inflammation from diverticulitis. Characteristic imaging features of the primary pathology are readily identifiable on diagnostic imaging modalities, such as CT, facilitating diagnostic differentiation from primary epiploic inflammation [6].

Epidemiology, clinical presentation and management of epiploic appendagitis

The precise incidence of epiploic appendagitis remains unknown and is likely underestimated due to limited clinical recognition and diagnostic challenges in the absence of CT and self-limiting nature of this disease. However, published data suggest an incidence of 2–7% in patients with initial clinical suspicion of acute diverticulitis or appendicitis [2]. Anatomical distribution data from surgical literature demonstrate a predilection for the rectosigmoid junction (57% of cases), corresponding to the region with the largest and most numerous appendages. Additional sites of

involvement include the ileocecal region (26%), ascending colon (9%), transverse colon (6%), and descending colon (2%) [1]. The condition predominantly affects individuals between the second and fifth decades, with a mean age at diagnosis of 44 years (range: 12–82 years) [6]. A marked gender disparity exists, with males affected approximately four times more frequently than females [2]. Obesity, rapid weight loss, hernia, excessive stretching or heavy exercise are potential risk factors for the development of epiploic appendagitis [3, 4].

The diagnosis of epiploic appendagitis presents significant clinical challenges due to the absence of pathognomonic clinical findings or specific laboratory abnormalities. The condition frequently mimics more common etiologies of abdominal pain, particularly diverticulitis and appendicitis [6]. Physical examination typically reveals focal, non-migratory abdominal pain, most commonly localized to the left lower quadrant, with the right lower quadrant representing the second most frequent site of involvement. This distribution corresponds to the anatomical predilection of epiploic appendagitis. The pain characteristically remains unchanged with movement and is not associated with significant guarding or rigidity. No palpable mass is typically detected and the pain is typically localized, with patients often able to indicate the exact site of pathology with point localization. Associated symptoms also may include postprandial fullness, abdominal distension, vomiting, early satiety, diarrhea, and, infrequently, mild fever [8].

Laboratory findings in patients with primary epiploic appendagitis is usually within normal limits and the findings, if present, are mostly non-specific. Erythrocyte sedimentation rate, hepatic transaminases, pancreatic enzymes (amylase and lipase), and urinalysis parameters generally remain within normal ranges. Occasionally, mild leukocytosis and modest elevations in C-reactive protein may be observed, reflecting the inflammatory response to ischemic fat necrosis [9].

Primary epiploic appendagitis typically follows a self-limiting clinical course with spontaneous resolution occurring within 2 weeks from the onset of symptoms [10]. Complications are infrequent but may include adhesion formation, bowel obstruction, intussusception, peritonitis, or abscess formation [8]. Management is conservative and symptom-directed, without requirement for surgical intervention [7]. Despite its benign nature, accurate diagnosis remains clinically significant, as misdiagnosis may result in unnecessary hospitalization, dietary restrictions, medical treatment, and potentially unwarranted surgical procedures [11].

Radiologic imaging features of epiploic appendagitis

Diagnosing epiploic appendagitis based solely on clinical symptoms is highly challenging; therefore, radiological assessment is essential. The primary imaging modalities used for diagnosis are computed tomography (CT) and ultrasonography. Although abdominal ultrasonography offers the advantage of targeted examination guided by the patient's point of maximal tenderness, it could show no abnormalities in some cases [12]. Consequently, CT has emerged as the gold standard imaging technique for

definitive confirmation of primary epiploic appendagitis [2]. In the pediatric population, magnetic resonance imaging (MRI) is also considered the modality of choice [13]. However, in adults, MRI is not a key diagnostic tool for evaluating acute abdominal pain and for instance epiploic appendagitis and will therefore not be discussed further in this article.

On non-contrast CT images, epiploic appendagitis manifests as an ovoid fat-density lesion measuring 1–5 cm in axial diameter, contiguous with the colonic wall. While classically located anteriorly along the sigmoid or descending colon, it may occur anywhere along the circumference of the colon [6,11]. Another key finding is the characteristic “hyperattenuating ring sign” representing a 2–3 mm thick peripheral rim of increased attenuation surrounding the lesion, reflecting inflammation of the visceral peritoneal covering. The inflammatory process may extend to involve the parietal peritoneum in 30–40% of cases, causing its thickening and hyperattenuation. Peri-lesional fat stranding constitutes the third key radiographic feature, indicating inflammation of adjacent mesenteric adipose tissue [7]. Notably, colonic wall thickening is disproportionately minimal compared to the surrounding fat stranding, confirming the pericolonic appendage as the primary inflammatory focus rather than the bowel wall [2]. Furthermore, a central high-attenuation focus, referred to as the “central dot sign,” strongly suggests central venous pedicle thrombosis within an inflamed epiploic appendage. Although this is a characteristic imaging feature, its prevalence varies, being reported in 30–78% of cases [2]. Therefore, the absence of this sign does not rule out a diagnosis of epiploic appendagitis. Post-contrast CT images show no enhancement of the inflamed epiploic appendage or the surrounding peritoneum [2].

On ultrasound images, epiploic appendagitis has been described as hyperechoic, non-compressible oval mass circumscribed by a hypoechoic line located at the point of maximum tenderness [2]. Using color Doppler ultrasound, in the mass flow is not detected, as a result of hypovascularity caused by torsion and vein thrombosis [7]. Occasionally, an echogenic focus, known as the “central dot sign,” may be observed, representing a thrombosed central vein within the torsed epiploic appendage [7].

Differential diagnosis

The differential diagnosis of epiploic appendagitis should include other conditions that present with acute abdominal pain, particularly in the lower quadrants. On CT, epiploic appendagitis is part of a group of diseases characterized by fat stranding that is disproportionately greater than the degree of bowel wall thickening. This imaging feature is a useful clue in narrowing the differential diagnosis to four main conditions: diverticulitis, omental infarction, mesenteric panniculitis, and appendicitis. Conversely, conditions primarily affecting the bowel wall, such as ischemic colitis, inflammatory bowel diseases (e.g., Crohn's disease and ulcerative colitis), infectious colitis and other enteropathies, are less likely in this context [14]. Radiological imaging results together with the clinical presentation and physical examination results lead to correct diagnosis.

Acute colonic diverticulitis

Patients with acute diverticulitis often present with left lower abdominal pain, similar to those with epiploic appendagitis. However, diverticulitis is more frequently associated with systemic inflammatory symptoms, including diffuse abdominal tenderness, rebound tenderness, and fever, which are less common in epiploic appendagitis. Additionally, laboratory findings in patients with diverticulitis typically reveal a significantly elevated white blood cell (WBC) count and higher levels of inflammatory markers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), further distinguishing it from epiploic appendagitis, which often presents with normal or only mildly elevated inflammatory markers [15].

Acute diverticulitis is typically characterized on CT imaging by colonic wall thickening and pericolonic fat stranding in patients with a known history of diverticulosis [15]. On post-contrast CT, enhancement of both the diverticulum and colonic wall is commonly observed. In some cases, the colonic wall exhibits a distinct 'target sign' appearance, characterized by inner and outer high-attenuation layers surrounding a thick middle layer of low attenuation, reflecting mural edema and inflammation [7]. Notably, colonic wall thickening in acute diverticulitis is significantly more pronounced than in epiploic appendagitis. Furthermore, diverticulitis is often associated with complications such as perforation, abscess formation, and fistulization, none of which are characteristic of epiploic appendagitis. Importantly, acute diverticulitis is the most common underlying cause of secondary epiploic appendagitis. The presence of multiple epiploic lesions, larger lesion size, and diffuse colonic wall thickening may suggest secondary rather than primary epiploic appendagitis [2]. However, diverticulosis without active inflammation can coexist in patients with epiploic appendagitis and should not preclude its diagnosis when typical imaging features are present. Therefore, careful evaluation of both diverticular and epiploic appendage inflammation is crucial in cases where these entities coexist.

On ultrasound, acute diverticulitis also demonstrates distinct features that differentiate it from epiploic appendagitis. One of the hallmark findings is the “Dome Sign,” characterized by a hemispheric outpouching with a hypoechoic wall and a central echogenic area, typically protruding from a thickened colonic segment [16]. This echogenic focus may produce acoustic shadowing, often attributed to the presence of gas or inspissated fecal material within the diverticulum. Another key sonographic feature is pericolic fat inflammation, seen as echogenic, non-compressible fat surrounding the affected diverticulum. Bowel wall thickening (>4 mm) further supports the diagnosis, indicating edematous changes and inflammation within the colonic wall [17]. Furthermore, on Color Doppler acute diverticulitis shows increased signal, which helps to differentiate it from epiploic appendagitis, which shows absence of Doppler signal because of a lack of blood flow [7]. In more severe cases, ultrasound may reveal organized fluid collections, suggestive of abscess formation [17].

Acute Appendicitis

Clinically, acute appendicitis follows a characteristic course, initially presenting as periumbilical pain lasting less than 5 days, which subsequently migrates to the right lower quadrant. This is often accompanied by peritoneal irritation, anorexia, nausea, vomiting, diarrhea, and fever $>37.5^{\circ}\text{C}$. However, classic pain migration to the right iliac fossa occurs in only about 50% of cases, making the diagnosis more challenging [18]. Laboratory findings in acute diverticulitis, similar to those in acute appendicitis, typically show an elevated white blood cell (WBC) count and increased inflammatory markers. However, normal laboratory results do not exclude the diagnosis of acute appendicitis [7].

On CT imaging, acute appendicitis typically appears as a fluid-filled, thickened appendix measuring $>8\text{--}9\text{ mm}$ (outer-to-outer wall) [19, 20], with wall thickening ($\geq 3\text{ mm}$) and a stratified appearance that may exhibit hyperenhancement following contrast administration [21]. On the other hand, in gangrenous appendicitis, focal wall non-enhancement and intraluminal gas may be present, indicating an elevated risk of perforation. Additionally, endoluminal appendicoliths are seen in approximately one-third of appendicitis cases, and their presence further increases the likelihood of perforation [21]. However, an appendicolith may occasionally be found in an otherwise normal-appearing appendix. Other CT signs associated with appendicitis include adjacent bowel wall thickening, cecal apical thickening (evident as the “cecal bar” and “arrowhead” signs), periappendiceal fat stranding, mesoappendiceal thickening, abscess formation, and lymphadenopathy [2, 3, 21]. Periappendicular fat stranding is typically mild to moderate, but when it is severe in the absence of significant cecal or ileal thickening, appendicitis is strongly suspected [7]. Diagnostic challenges arise in both the early and late stages of appendicitis. In early or mild appendicitis, the appendix may retain a normal diameter, with inflammatory fat stranding as the only detectable abnormality. In contrast, advanced cases can be difficult to diagnose due to peritonitis and abscess formation, which may obscure visualization of the appendix on imaging. [21]. In rare cases, epiploic appendages may be located near the vermiform appendix, where they are smaller than those along the colonic wall. Differentiating appendiceal epiploic appendagitis from tip appendicitis is essential to prevent unnecessary surgical intervention [2].

On ultrasound (US), appendicitis appears as an aperistaltic, non-compressible, fluid-filled, blind-ending tube with an outer diameter $>6\text{ mm}$ (noting that US measurements are typically 1–2 mm smaller than those on CT) [22]. It is characterized by wall thickening ($\geq 3\text{ mm}$) and a layered stratified appearance. On transverse imaging, the inflamed appendix exhibits a “target” (bull’s eye) sign, reflecting bowel wall thickening due to acute inflammation [23]. An appendicolith if present appears as a hyperechoic intraluminal mass with posterior acoustic shadowing. On color Doppler imaging, appendicitis—like acute diverticulitis—demonstrates mural or extramural hyperemia, which

helps differentiate it from epiploic appendagitis, where Doppler signal is absent. However, in cases of necrotic appendicitis, vascular flow may also be absent in the affected segment. Additional US findings include periappendiceal hyperechoic indurated fat, complex fluid collections, and reactive lymphadenopathy, which further support the diagnosis [7, 21, 24].

Omental infarction

The clinical presentation of omental infarction is very similar to that of epiploic appendagitis, as both result from torsion-induced aseptic inflammation. This contrasts with the septic inflammation seen in appendicitis or acute diverticulitis. As a result, patients with omental infarction typically exhibit only mild elevations in WBC count, ESR, and CRP, or may even have values within the normal range. [25]. Other gastrointestinal symptoms such as nausea, vomiting, anorexia, fever, or bowel dysfunction are also usually absent [14]. The key clinical distinction is pain location, with omental infarction more commonly affecting the right side of abdomen (most often lower quadrant). This predisposition is due to the omental anatomy, including its greater length, mobility, and weaker blood supply, making it more prone to torsion and infarction [7].

On CT imaging, omental infarction appears as a heterogeneous, ill-defined fat-density lesion [7]. It is typically larger than epiploic appendagitis, ranging from 3 to 15 cm in diameter, and usually lacks both the hyperattenuating ring sign and the central dot sign observed in epiploic appendagitis [2, 3]. Omental infarction also tends to occur in atypical locations, most commonly within the omentum or the right iliac fossa, rather than directly abutting the colonic surface as seen in epiploic appendagitis. However, in some cases, it may be adjacent to the colonic wall, leading to mild colonic wall inflammation, which is significantly less pronounced than the associated fat stranding [14]. CT findings in epiploic appendagitis and omental infarction may overlap, making differentiation challenging. However, this distinction is often of limited clinical significance, as both conditions are self-limiting and typically resolve spontaneously [7].

On ultrasound (US), omental infarction closely resembles epiploic appendagitis. It typically appears at the site of maximal tenderness as a triangular or oval-shaped hyperechoic mass, ranging in size from 3.5 to 15.5 cm, with no internal blood flow on color or power Doppler imaging. On dynamic US compression, omental infarction is rigid, fixed to the abdominal wall, and remains immobile during respiration, unlike normal omental fat, which moves with breathing. This finding, known as the “tethered fat sign,” along with the absence of a surrounding hypoechoic rim, helps distinguish omental infarction from epiploic appendagitis [26].

Mesenteric panniculitis

The clinical presentation of mesenteric panniculitis varies widely. In most cases, mesenteric panniculitis is asymptomatic, however in some cases it presents with nonspecific symptoms like abdominal pain (30%–72%), fever, malaise, weight loss

(20–23%), and altered bowel habits with either constipation or diarrhea (20–26%). A palpable, poorly defined abdominal mass is reported in 35–50% of cases. Rare symptoms include rectal bleeding, jaundice, and gastric outlet obstruction [27]. Laboratory inflammatory markers, such as C-reactive protein, leukocytosis is nonspecific and elevated in 14–88%, often secondary to underlying inflammation [27].

On CT imaging, mesenteric panniculitis typically presents as a heterogeneous mass characterized by hyperdense fat streaks and attenuation values higher than those of retroperitoneal fat. This mass is often surrounded by a thin pseudocapsule, which is usually oriented toward the left side. Another imaging features of mesenteric panniculitis is low attenuation fatty “halo sign” surrounds lymph nodes or mesenteric vessels and its ability to cause mass effect that results in the displacement of adjacent small-bowel loops. Additionally, the presence of small lymph nodes, generally measuring less than 5 mm in diameter, within the fat-stranding mass is considered a characteristic finding [7]. Distinguishing mesenteric panniculitis from epiploic appendagitis can be aided by its central mesenteric location, its relatively larger extent, and the presence of a cluster of enlarged lymph nodes, particularly in the absence of an identifiable acute inflammatory process [14].

Ultrasound imaging commonly reveals thickening and distortion of the mesenteric root, accompanied by a mild increase in echogenicity. A mass effect may also be observed. Additionally, a hypoechoic fat region surrounding the mesenteric vessels, indicative of a halo of sparing, can be visualized. Color Doppler sonography further demonstrates the presence of mesenteric vessels within the mass without significant deviation [28].

CONCLUSIONS

Epiploic appendagitis remains an often-overlooked yet clinically significant cause of acute abdominal pain. Its non-specific symptoms and overlap with more common conditions, such as diverticulitis and appendicitis, contribute to frequent misdiagnosis. Accurate recognition through imaging, particularly CT and ultrasound, is crucial in distinguishing epiploic appendagitis from other intra-abdominal pathologies to prevent unnecessary hospitalizations, antibiotic therapy, and surgical interventions.

CT remains the gold standard for diagnosing epiploic appendagitis, offering superior accuracy compared to ultrasonography, which is limited by variable sensitivity. On CT imaging, epiploic appendagitis belongs to a group of pathologies characterized by disproportionately greater fat stranding relative to bowel wall thickening. This key feature aids in narrowing the differential diagnosis to diverticulitis, omental infarction, mesenteric panniculitis, and appendicitis.

Distinct imaging characteristics further help differentiate epiploic appendagitis from these conditions. On CT, it appears as an ovoid fat-density lesion with a hyperattenuating ring sign and a central dot sign, while ultrasound findings include hyperechoic, non-compressible oval mass circumscribed by a hypoechoic line with the absence of bowel

wall involvement and absence of signal on color Doppler imaging. Recognizing these features is crucial for accurate diagnosis, preventing unnecessary interventions, and ensuring appropriate patient management.

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Santrauka

EPIPLOINIO APENDIGITO DIAGNOSTINIAI IŠŠŪKIAI

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Išvadas. Epiploinis apendigitas yra reta ir dažnai klaidingai diagnozuojama ūmaus pilvo skausmo priežastis. Jis gali būti pirminis ir antrinis. Epiploinis apendigitas dažnai imituoja kitas ūmines pilvo ligas, tokias kaip apendicitas ar divertikulitas, kuriems paprastai reikia chirurginės intervencijos. Diagnostiniai vaizdinimo metodai padeda teisingai atpažinti epiploinį apendigitą, jį diferencijuoti nuo kitų dažnesnių patologijų ir išvengti klaidingos diagnozės. Tiksliai diagnozė yra labai svarbi tinkamai pacientų gydymo taktikai pasirinkti.

Darbo tikslas. Atkreipti dėmesį į epiploinio apendigito diagnostinius iššūkius ir pabrėžti vaizdinimo metodus, ypač KT ir ultragarsinio tyrimo, vaidmenį diferencijuojant jį nuo kitų pilvo skausmo priežasčių.

Tyrimo metodai. Literatūros apžvalga atlikta naudojantis *PubMed* ir *Google Scholar* duomenų bazėmis, įvedus šiuos reikšminius žodžius: epiploinis apendigitas, pilvo skausmas, ūmus pilvas, diferencinė diagnostika, divertikulitas, taukinės infarktas, mezenterinis panikulitas, apendicitas. Atrinkti 28 straipsniai, tinkami tyrimo tikslo sąlygoms patenkinti.

Išvados. KT tyrimas yra aukso vertės standartas diagnozuojant epiploinį apendigitą, tačiau ultragarsinis tyrimas taip pat suteikia svarbios informacijos. Diferencinė epiploinio apendigito diagnostika dažniausiai apima ligas, kurioms būdingas ryškus riebalinio audinio uždegimas su minimaliais žarnos sienelės pokyčiais. Šioms ligoms priklauso divertikulitas, apendicitas, taukinės infarktas ir mezenterinis panikulitas. Svarbiausi KT požymiai, padedantys diferencijuoti epiploinį apendigitą, yra ovalus riebalinio audinio tankio darinys su padidėjusio tankio žiedu ir centrinio taško ženklai. Ultragarsu matomas hiperechogeniškas, nesuspaudžiamas ovalus darinys, apsuptas hipoechogeninės linijos, be žarnų sienelės pažeidimo ir neturintis spalvinio doplerio signalo.

Reikšminiai žodžiai: epiploinis apendigitas, pilvo skausmas, ūmus pilvas, diferencinė diagnostika, divertikulitas, taukinės infarktas, mezenterinis panikulitas, apendicitas.

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