ploic appendage torsion or spontaneous thrombosis of the epiploic appendage central draining vein resulting in vascular occlusion and focal inflammation [5–7, 12, 13]. In some cases of epiploic appendagitis, inflammation may be present without any vascular impairment [6, 14, 15].

The reported age range for primary epiploic appendagitis is 12–82 years, with a peak in incidence in the fifth decade. Primary epiploic appendagitis is more common in obese patients and women [3, 4]. Primary epiploic appendagitis occurs more frequently in the sigmoid colon than in the cecum or ascending colon and is uncommon in the transverse colon [3, 5, 16]. Epiploic appendages may cause incarcerated hernia; a case has been reported of an epiploic appendagitis within an incisional hernia sac [10].

Primary epiploic appendagitis usually presents as an abrupt onset of focal abdominal pain in the lateral lower quadrants, is nonmigratory, and worsens with cough and abdominal stretching [2, 3, 11, 17]. Appetite and bowel function are usually unchanged; nausea and vomiting are rare [6, 16]. On physical examination, the patient will present with localized tenderness without significant guarding or rigidity. The patient also may have a low-grade fever [3, 18]. WBC count is usually normal or slightly elevated [2]. Heavy exercise has been reported as a predisposing factor [2, 4, 6].

Primary epiploic appendagitis is difficult to diagnose clinically because of the lack of pathognomonic clinical features and can simulate a case requiring surgery [3, 4].
Right-sided primary epiploic appendagitis is often confused with acute appendicitis or right-sided diverticulitis; whereas left-sided primary epiploic appendagitis is often confused with sigmoid diverticulitis [3, 4, 18].

In the past, diagnosis of epiploic appendagitis was often the result of an unexpected finding during an exploratory laparotomy [3]. Today, this condition is usually diagnosed by ultrasound or CT, with the latter more sensitive and specific. Although ultrasound has the advantage of correlating the location of the lesion with the location of maximum tenderness, CT should be used to confirm the fatty nature of the lesion before making a definite diagnosis of primary epiploic appendagitis. With the increasing use of CT for assessing cases of acute abdominal pain, the diagnosis of epiploic appendagitis is now more common [18, 19].

Primary epiploic appendagitis is self-limited in the majority of patients and spontaneously resolves within 5–7 days. Rarely, acute epiploic appendagitis may result in adhesion, bowel obstruction, intussusception, intraperitoneal loose bodies, peritonitis, or abscess formation [3, 5]. Primary epiploic appendagitis does not require surgery, and treatment is based on the patient’s symptoms [4, 5, 7, 13, 15]. Misdiagnosis may lead to unwarranted surgery, medical treatment, and hospitalization [4].

Secondary Epiploic Appendagitis

In secondary epiploic appendagitis, the epiploic appendage is inflamed because of another process, such as diverticulitis, appendicitis, pancreatitis, or cholecystitis. Management of secondary epiploic appendagitis is based on treatment of the primary abnormality [1, 2, 4, 7].

Imaging Findings

The diagnosis of acute epiploic appendagitis primarily relies on cross-sectional CT, although ultrasound and MRI are occasionally used [11, 19].

CT

In CT of a healthy patient, the epiploic appendages blend in with the surrounding pericolic fat but become apparent when surrounded by ascites (Fig. 1) or inflammation [3, 5, 8]. An infarcted or inflamed epiploic appendage on CT appears as a 1–4 cm ovoid pericolic lesion with fat density surrounded by inflammatory changes and abuts the anterior colonic wall [1, 3, 5, 11, 15]. Primary epiploic appendagitis may have a lobular appearance because of two or more affected, contiguous epiploic appendages with hyperattenuated rings lying in proximity [19].

A 2–3 mm hyperdense rim surrounding the ovoid mass on CT (hyperattenuating ring sign) represents the inflamed visceral peritoneal covering of the epiploic appendage and is diagnostic of primary epiploic appendagitis [13, 15]. The hyperdense rim surrounding the ovoid mass on CT corresponds to the hypoechoic halo on ultrasound [13, 15, 19] (Figs. 2–6). Thickening of the parietal peritoneum secondary to the spread of inflammation may be observed [3, 5–7].

Fat stranding is more pronounced than wall thickening because the paracolonic inflammatory changes are disproportionately more severe than the mild local reactive thickening of the adjacent colonic wall (Figs. 2–6). Wall thickening of the adjacent side of the colon is asymmetric [1, 4, 8] (Figs. 2 and 3).

A central, hyperattenuating, ill-defined round area (“central dot sign”) or a longitudinal linear area corresponds to engorged or thrombosed central vessels or central areas of hemorrhage or fibrosis (Figs. 2–4). Although the presence of a central dot or linear area is useful for diagnosis, their absence does not exclude the diagnosis of acute epiploic appendagitis [4–6, 11, 19, 20].

The central dot may have high attenuation because the infarcted tissue tends to calcify. Calcification may be eggshell in shape and may become detached and appear as a peritoneal loose body in the abdominal cavity [1, 3, 16, 21]. The calcified tissue may reattach itself to a surface, such as the lower aspect of the spleen, in which case it is called a “parasitized epiploic appendage” [3]. The smooth surface and calcified consistency of the epiploic appendage help to distinguish it from a metastatic lesion (Fig. 7).

Ultrasound

At the site of maximum tenderness, a non-compressible hyperechoic small ovoid or round solid mass of adipose tissue is seen be-
Imaging of Epiploic Appendagitis

Fig. 3—Primary epiploic appendagitis in 23-year-old man with clinical diagnosis of presumed colonic diverticulitis. Axial contrast-enhanced CT scan shows pericolic fatty lesion surrounded by hyperattenuating ring (thick arrow) containing central hyperattenuating area (thin arrow) corresponding to thrombosis and hemorrhagic changes and very mild thickening of colonic wall (open arrow).

Fig. 4—Acute epiploic appendagitis with hyperattenuating center in 31-year-old woman. Axial contrast-enhanced CT image shows lesion (arrow) that abuts sigmoid colon and has central focal area of hyperattenuation with surrounding inflammation.

Fig. 5—48-year-old man with acute epiploic appendagitis with hyperattenuating ring sign (thick arrow) adjacent to left colon without significant involvement of colonic wall. Severe and disproportionate fat stranding (thin arrow) is seen nearby.

Fig. 6—Acute epiploic appendagitis (arrow) with hyperattenuating ring sign adjacent to sigmoid colon without significant involvement of colonic wall in 52-year-old man.

Fig. 7—CT image shows small calcified body (arrow) adjacent to normal epiploic appendices, probably residual to anterior epiploic appendagitis in 59-year-old man.

Fig. 8—Sonogram in 22-year-old man shows hyperechoic small mass (asterisk) surrounded by hypoechoic border (arrow) corresponding to hyperattenuating ring on CT scans at site of maximum tenderness, located anteriorly between colon and abdominal wall.

Differential Diagnosis

In epiploic appendagitis, there is much more fat stranding than bowel wall thickening. In patients with acute abdominal pain, the finding of fat stranding that is disproportionate suggests a relatively narrow differential diagnosis: diverticulitis; omental infarction; appendicitis; and, less commonly, mesenteric panniculitis and primary tumors and metastases to the omentum.

MRI

The involved epiploic appendage is hyperintense on unenhanced T1-weighted imaging but is slightly less intense than normal peritoneal fat. Epiploic appendages show marked loss of signal on fat-suppressed T2-weighted images, confirming the fatty nature of the lesion [23].

In epiploic appendagitis, the thin peripheral rim and the perilesional inflammatory changes appear hypointense on T1-weighted imaging, appear hyperintense on T2-weighted imaging, and show marked enhancement on contrast-enhanced T1-weighted fat-suppressed images, whereas the central draining vein usually has low signal on both T1-weighted and T2-weighted imaging [23].

Differential Diagnosis

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Diverticula can be found anywhere in the colon, but the majority are located in the distal descending and sigmoid colon; hence, most cases (95%) of diverticulitis are located in the left side of the abdomen. A minority of diverticula (5%) are located on the right side of the abdomen, which for unknown reasons has a predilection for patients of Asian descent. The transverse colon is rarely affected, whereas the rectum is completely spared from diverticula formation [8].

The most common CT finding of acute diverticulitis is paracolic fat stranding. Other typical CT findings include an ill-defined or blurry diverticulum in the region where the fat stranding is most pronounced (Figs. 9 and 10); mild wall thickening (usually < 5 mm), commonly more pronounced on the side of the offending diverticulum and usually affecting a large colonic segment greater than 5 cm (Figs. 9 and 10); thickened base of the sigmoid mesocolon with fluid (“comma sign”) (Figs. 9 and 10); and engorged vessels supplying the affected segment (“centipede sign”) [8]. Other findings detected by CT include pericolic abscess, small-bowel obstruction, free intraperitoneal or extraperitoneal gas, colovesical fistula, thrombosis of the mesenteric or portal veins caused by pyophebitis, and hepatic abscess formation. In some instances, small localized collections of gas are identified adjacent to the colonic wall and indicate localized pericolic perforation [17] (Figs. 11 and 12). In cases of complicated diverticulitis, surgical management may be required.

Features that help discern the differential diagnosis—Although their clinical manifestations are similar, acute epiploic appendagitis tends to occur in younger patients, whereas acute diverticulitis frequently affects older patients (> 50 years). Patients with diverticulitis are more likely to experience nausea, vomiting, fever, rebound tenderness, and more diffuse lower abdominal pain. Only a minority (7%) of patients with acute epiploic appendagitis have leukocytosis, whereas most patients with acute diverticulitis have an elevated WBC count [5].

In cases of acute epiploic appendagitis, the involved colonic segment is short; however, in cases of acute diverticulitis, a lengthy segment of thickened colonic wall is a typical CT feature [8].

The classic complications of diverticulitis (extramural abscesses, sinus tract and fistula formation, bowel obstruction, perforation, and peritonitis) are rare in the setting of acute epiploic appendagitis [5, 17] (Figs. 11 and 12).

Features that complicate the differential diagnosis—Both epiploic appendagitis and acute diverticulitis frequently present with a sudden focal left-sided tenderness. Moreover, the two conditions can be seen simultaneously because the inflammation from an acute diverticulitis may extend to involve the epiploic appendages [4, 5, 7] (Fig. 12).

Omental Infarction
The greater omentum consists of a four-layered fold of peritoneum that covers the colon and small bowel in the peritoneal cavity, acting as a barrier to generalized spread of intraperitoneal infection or tumor. It contains fat and vascular structures (Fig. 13). Omental infarctions are rare because abundant collateral vessels perfuse the omentum [25]. The cause of omental infarction is unclear but may be similar in pathophysiology to the cause of primary epiploic appendagitis [9]. Omental infarction occurs when there is an interruption of arterial blood supply to the omentum, possibly because of omental torsion, venous insufficiency due to trauma, or spontaneous thrombosis of the omental veins [5, 26]. Right-sided epiploic vessels are involved in 90% of the cases and are thought to be caused by...
by the omentum being longer and more mobile on the right side [21]. Precipitating factors include obesity, recent abdominal surgery, strenuous activity, congestive heart failure, digitalis administration, and abdominal trauma [5, 12, 27]. However, most cases of omental infarction are idiopathic [16] (Fig. 13).

Omental infarctions are usually localized to the right upper and lower quadrants and clinically mimic cholecystitis and appendicitis, respectively [16, 26, 28]. A few cases of left-sided omental infarction have also been described [28].

Omental infarction more commonly affects elderly obese patients, with a slight predilection for men [16]. Omental infarction usually presents with acute abdominal pain with normal or mildly elevated WBC count. Other presenting symptoms include nausea, vomiting, anorexia, diarrhea, and fever [21].

CT findings of omental infarction range from a subtle, focal, hazy soft-tissue infiltration of the omentum [25, 29] to a solitary large, cakelike, nonenhancing, heterogeneous, and high-attenuating fatty mass centered in the great omentum [8, 13, 21] (Figs. 14–17). Omental infarctions are usually located in the right upper or lower quadrants, deep in relation to the abdominal wall, anterior to the transverse colon, or anteromedial to the ascending colon. Colonic involvement depends on the anatomic location of the infarcted omentum relative to the colon [8, 11, 13, 26] (Figs. 14–16). Reactive bowel wall thickening may occur, although the inflammatory process in the omentum is usually disproportionately more severe [8, 21, 26]. Omental torsion is implicated as a cause of omental infarction when a whirled pattern of concentric linear strands is seen on CT [21].

In most cases of omental infarction, the process is self-limited, but surgery may be indicated if symptoms persist or an associated abscess develops [27].

Features that help discern the differential diagnosis—Unlike acute epiploic appendagitis, which predominantly affects adults (> 20 years), omental infarction can occur in pediatric patients (15% of cases) [5].

On CT, omental infarction lacks the hyperattenuating ring and central dot seen in epiploic appendagitis [2, 5, 21, 28]. The focal lesion in acute epiploic appendagitis is often less than 5 cm long, may have a lobular appearance, and is frequently found adjacent to the sigmoid colon. The lesion in omental infarction is larger than that of epiploic appendagitis (averaging a diameter of up to 7 cm), cake-like, centered in the omentum, and commonly located medial to the cecum or the ascending colon [4–6, 19, 21].

The pain in acute epiploic appendagitis is typically in the inferior abdomen, whereas the pain in omental infarction is more common in the right side of the abdomen.

Features that complicate the differential diagnosis—The CT findings in both epiploic appendagitis and omental infarction may overlap and the two entities cannot be differentiated. Nevertheless, the clinical relevance of such differentiation is limited because both conditions are self-limited and tend to resolve spontaneously [4, 28]. Treatment is conservative unless the infarcted omentum becomes infected [2].

Because they have the same common denominator of spontaneous fatty tissue
necrosis, epiploic appendagitis and omental infarction have recently been proposed to be regrouped under the same new term, “intraabdominal focal fat infarction” or “IFFI,” to stress that the differentiation is not clinically important and that the management of the two entities is conservative [9, 30].

**Appendicitis**

In the Western world, appendicitis is the most common cause of acute abdominal pain that requires surgical intervention [31, 32]. The cause in the majority of cases is obstruction of the lumen of the appendix secondary to fecaliths, lymphoid hyperplasia, foreign bodies, parasites, and tumors [33]. After mechanical obstruction, the continued secretion of mucus results in luminal distention, venous engorgement, arterial compromise, and tissue ischemia. Luminal bacteria multiply and invade the appendiceal wall, causing transmural inflammation. Appendiceal infarction, microperforation, and extension of inflammation to the parietal peritoneum and adjacent structures are possible sequelae of appendicitis [8, 31, 33].

Appendicitis can affect all ages, although it is relatively rare at extremes of age, with the greatest incidence in the second decade of life [32, 33]. Appendicitis usually presents as a periumbilical pain of less than 5 days in duration and migrates to the right lower quadrant, associated with peritoneal irritation, anorexia, nausea, vomiting, diarrhea, and temperature greater than 37.5°C [34]. The WBC count may be elevated, but this is not seen in all cases of appendicitis.

Acute appendicitis presents on CT images as a thick fluid-filled appendix (> 6 mm outer-to-outer wall), with intramural gas, appendiceal wall thickening (wall thickness ≥ 3 mm), and a stratified appearance that may hyperenhance after contrast material administration [31, 35]. Endoluminal appendicoliths are present in one third of patients with appendicitis; their presence increases the likelihood of appendiceal perforation [31, 33]. Occasionally, an appendicolith may be identified in an otherwise normal-appearing appendix [27].

Other associated CT signs of appendicitis are adjacent bowel wall thickening, cecal apical thickening (“cecal bar” and arrowhead signs), adjacent fat stranding, right lateral colonic thickening, the presence of an abscess, and lymphadenopathy [8, 31, 32, 34] (Figs. 18 and 19). Periappendicular fat stranding is typically mild to moderate, but the diagnosis of appendicitis is strongly implicated when severe fat stranding is found in the absence of substantial cecal or ileal thickening. A careful search for a thickened or focally perforated appendix will often confirm the diagnosis of appendicitis [8].

Features that help discern the differential diagnosis—Appendicitis affects all ages,
Imaging of Epiploic Appendagitis

unlike acute epiploic appendagitis, which predominantly affects adults (> 20 years). Appendicitis has a more typical clinical presentation with periumbilical pain migrating to the right lower quadrant and is associated with anorexia and nausea. Patients with acute epiploic appendagitis usually do not present with significant guarding or rigidity on physical examination. Most patients with acute appendicitis have an elevated WBC count, whereas only a minority of patients with acute epiploic appendagitis have leukocytosis.

A definitive CT diagnosis of appendicitis can be made with the identification of an abnormal appendix or a calcified appendicolith in association with pericecal inflammation [33]. Doppler ultrasound shows increased blood flow in contrast to absent blood flow in epiploic appendagitis [4, 12, 20].

Features that complicate the differential diagnosis—Classic presentation of acute appendicitis occurs in only 50–60% of patients, and the diagnosis may be missed or delayed in cases without the classic presentation [33]. In cases of perforated appendicitis, with peritonitis and abscess formation, the appendix may be difficult to see on imaging studies [8]. In early or mild appendicitis, the appendix may remain normal in size [35], and inflammatory stranding of the periappendicular fat may be the only finding.

Mesenteric Panniculitis

Mesenteric panniculitis is a subgroup of sclerosing mesenteritis, consisting of nonspecific chronic inflammation and fibrosis of the fatty tissue of the bowel mesentery. Depending on the predominant tissue type in the mesenteric lesion, sclerosing mesenteritis can be categorized into three subgroups: mesenter-
ic panniculitis if inflammation predominates over fibrosis, mesenteric lipodystrophy if fat necrosis is the predominant process, and retractile mesenteritis if fibrosis and retraction predominate [5, 36, 37]. Retractile mesenteritis is considered the final, more invasive stage of mesenteric panniculitis complicated by fibrosis and retraction [36]. Progression from mesenteric panniculitis to retractile mesenteritis is difficult to predict but fortunately is rare [29]. In most patients, the condition consists of a mixture of chronic inflammation, fat necrosis, and fibrosis [8].

Most cases of mesenteric panniculitis occur in middle or late adulthood (mean age, ~60 years), with a slight male predominance [5, 37]. Mesenteric panniculitis may be entirely asymptomatic, but clinical manifestations may be related to the inflammation or its mass effect and include acute abdominal pain, fever, nausea, vomiting, diarrhea, and weight loss [5, 36, 37]. Mesenteric panniculitis can be found in 0.6% of all patients undergoing abdominal CT for various indications. The pathogenesis is uncertain but can be associated with autoimmune disease, a paraneoplastic process, trauma, previous surgery, drug-induced disease, infection, and thrombosis of mesenteric vessels [37].

Mesenteric panniculitis mainly involves the mesentery of the small bowel, especially at its root [25, 37, 38]. The CT findings of mesenteric panniculitis include a focal area of increased attenuation within the mesenteric fat surrounded by a thin pseudocapsule that is usually oriented to the left side [5, 37, 38]. Mesenteric panniculitis surrounds the mesenteric vessels and shows some regional mass effect by local displacement of small-bowel loops. The small soft-tissue nodules associated with mesenteric panniculitis are thought to correspond to lymph nodes scattered within the mesenteric mass, usually less than 5 mm in diameter [8, 26]. The “fat-ring sign” appears as low-density fat that surrounds vessels and nodules within the mesenteric mass and represents preservation of normal fat density because of unaffected noninflamed fat [25, 26, 37, 38] (Fig. 20). The fat-ring sign is suggestive of mesenteric panniculitis but is nonspecific because it can be found in other entities such as lymphoma [26]. Areas of fibrosis within the inflammation appear as linear bands of soft-tissue attenuation, resulting in spiculation that may be mistaken for a neoplastic process [5]. Calcification is uncommon and may be related to the fat necrosis [36]. The major complications of mesenteric panniculitis are related to the progressive fibrosis that may lead to shortening of the mesentery, compression of the mesenteric vessels, and bowel-loop narrowing [5, 37].

Some features can help discern the differential diagnosis. Mesenteric panniculitis is not an acute abdominal condition and appears as a larger lesion. It is most commonly located in the root of the small-bowel mesentery that does not abut the colonic wall. Acute epiploic appendagitis, as the name im-
plies, is an acute disease seen as a small focal lesion anterior or anteromedial to the colon, abuts the colon wall, and does not involve the small-bowel mesentry [5].

**Primary Tumors and Metastases**

There are many other possible causes for a CT finding of a fatty mass or masslike lesion in the abdomen, such as liposarcoma (Fig. 21), dermoid and carcinoid tumor, lipoma, and omental metastases (Fig. 22).

Omental metastases can present as soft-tissue implants on peritoneal surfaces. “Omental cake” is the replacement of the omental fat by tumor infiltration and on CT appears as a thick, confluent soft-tissue mass closely adherent to the ventral surface of the transverse colon in the mid abdomen [25]. The presence of ill-defined lesion margins, numerous lesions, a lesion centered in the omentum, and a history of primary neoplasm are useful for diagnosing omental metastasis [5] (Fig. 22). In patients with known malignancy, the diagnosis of acute epiploic appendagitis should only be made if there is a presentation with acute abdominal pain and no CT evidence of peritoneal metastatic disease elsewhere [2].

**Conclusion**

Epiploic appendagitis is self-limiting, and the appropriate management is conservative. In our experience, many clinicians are not familiar with this entity, and the radiologist can provide guidance for supportive management. Therefore, a lesion centered in the omentum, and a history of primary neoplasm are useful for diagnosing omental metastasis [5] (Fig. 22). In patients with known malignancy, the diagnosis of acute epiploic appendagitis should only be made if there is a presentation with acute abdominal pain and no CT evidence of peritoneal metastatic disease elsewhere [2].

**References**