CME SAM

Abdominal Twists and Turns

AJR FOCUS ON:

Abdominal Twists and Turns: Part 2, Solid Visceral Torsions With Pathologic Correlation

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OBJECTIVE. The solid abdominal viscera are secured in place by various suspensory ligaments. Laxity or incomplete development of these anchoring ligaments can lead to hypermobility and predispose the patient to torsion-related ischemic pathology. The clinical symptoms of solid visceral intraabdominal torsions are nonspecific. A prompt diagnosis is critical to avoid life-threatening consequences of prolonged visceral ischemia. Abdominal torsions are rarely diagnosed clinically, and it is often the responsibility of the radiologist to recognize and make the diagnosis through cross-sectional imaging. This article reviews the imaging spectrum, radiologic-pathologic correlations, and therapeutic implications of solid visceral intraabdominal torsions, including some unusual solid organ and abdominal fat torsions.

CONCLUSION. The clinical presentation of solid visceral intraabdominal torsion is nonspecific and radiologists are relied on to make this diagnosis on cross-sectional imaging studies. Recognition of the predisposing factors and imaging spectrum of intraabdominal torsions is essential to help direct timely intervention in these potentially life-threatening entities.

Keywords: abdominal viscera, torsion

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The peritoneum is a thin layer of squamous epithelium that covers most intraabdominal organs. Its various folds and reflections are subclassified based on anatomic location into named ligaments, intestinal mesentery, and omentum, which provide support to abdominal viscera, serve as a conduit for their blood supply, and compartmentalize the abdomen against the spread of infection (Fig. 1). Abnormalities of peritoneal attachments (typically ligamentous laxity or absence) can lead to hypermobility of intraabdominal viscera and predispose the patient to torsion-related ischemic pathology. Visceral hypermobility can be attributed to primary or secondary peritoneal attachment abnormalities (Fig. 2). Primary peritoneal attachment abnormalities may be congenital in origin or they may be due to an acquired laxity, whose underlying cause may be age-related, secondary to trauma, or hormonally influenced, such as in pregnancy. Alternatively, the peritoneal attachment may be normally developed but may twist on itself if it is exposed to any abnormal traction or mass effect, such as seen with an internal hernia or neoplasm.

Solid Organ Torsions

Torsion of Orthotopic or Wandering Spleen

Splenic torsion is a rare entity that predominantly occurs in patients with a hypermobile spleen. The main anchoring ligaments of the spleen include the splenogastric ligament that connects the spleen to the greater curvature of the stomach, the splenorenal ligament that helps connect the spleen to the posterior abdominal wall, and the phrenicocolic ligament that forms a supportive sling for the inferior splenic pole. Splenic hypermobility may be caused by congenital absence, maldevelopment, or acquired laxity of these suspensory ligaments [1, 2]. In certain cases, advanced hypermobility may be described as a wandering spleen, in which the spleen assumes an ectopic position inferiorly within the abdomen. Patients may present with chronic or acute abdominal pain depending on the degree of torsion. Torsion and detorsion are not rare, and the patients may have intermittent symptoms [1, 2]. Complications of splenic torsion include splenic congestion, mass effect on adjacent structures, and infarction [2]. Imaging may show an enlarged and engorged hypoenhancing orthotopic or ectopic spleen, heterogeneous sonographic echotexure with decreased

Doppler flow, and a whirled appearance of the splenic hilum used to describe twisting of the splenic pedicle with or without twisting of the pancreatic tail [1] (Fig. 3). Splenic torsion is treated with splenectomy [1, 3].

Wandering Liver

Wandering liver refers to hepatic hypermobility and is generally attributed to congenital laxity or maldevelopment of the coronary and triangular suspensory ligaments, ventral mesentery persistence, and absence of tethering of the inferior vena cava [4, 5]. Acquired disruption of hepatic ligaments (such as in liver transplantation) typically does not predispose to hepatic hypermobility or torsion because it is thought that postoperative adhesions form promptly and tether the donor liver into place [4]. Hepatic hypermobility is also associated with a history of an omphalocele in which the maldeveloped hepatic fossa and elongated hilar structures, once necessary to reach the omphalocele sac, now enable hepatic migration in the peritoneal cavity [4] (Fig. 4). Several case reports have also documented associated bowel obstruction observed in both adult and pediatric patients, although not always [5]. Despite the presumed increased risk for vascular torsion, all reported cases to date were discovered incidentally in patients without symptoms related to hepatic hypermobility [4–6].

Torsion of an Accessory Hepatic Lobe

Accessory lobes of the liver are uncommon, usually asymptomatic, and discovered as an incidental finding [7]. Similar to wandering liver, there is an association with history of an omphalocele in which it is believed that in utero traction on liver tissue that became adherent to the omphalocele sac may have separated it from the main hepatic lobes [7]. Accessory hepatic lobes are usually located on the posterior aspect of the liver, connected to the liver by either normal hepatic parenchyma or via a narrow fibrous stalk that contains a feeding hepatic artery, portal vein, and draining bile duct [7]. This long narrow pedicle places the accessory lobe at increased risk for torsion, with subsequent secondary congestion, ischemia, and ultimately infarction [8, 9]. Clinically, patients present with nonspecific right upper abdominal quadrant pain [7]. Imaging features include a heterogeneous edematous soft-tissue attenuation mass adjacent to the liver with decreased enhancement, decreased Doppler flow, and local mass effect, which may result in lateral displacement of the gallbladder [7] (Fig. 5). The narrow accessory hepatic lobe stalk is not always visualized. Treatment involves surgical resection of the infarcted accessory lobe [7, 10]. Prophylactic resection of accessory hepatic lobes is sometimes performed at the time of omphalocele repair to prevent future torsion [11].

Gallbladder Torsion

Gallbladder torsion predominantly occurs in elderly patients and the cause is attributed to age-related laxity of mesenteric attachments that may lead to gallbladder hypermobility [12, 13]. Approximately two thirds of cases are encountered in patients without gallstones [12]. The clinical presentation includes nausea, vomiting, right upper quadrant pain, fever, and a palpable right upper quadrant mass [13]. Imaging may reveal a distended gallbladder with an aberrant orientation remote from the gallbladder fossa and tapering and twisting of the cystic duct [12] (Fig. 6). Most cases present with imaging findings similar to acalculous cholecystitis with gallbladder distention and wall thickening with or without emphysematous or ischemic changes. Definitive diagnosis is made intraoperatively at the time of cholecystectomy [13].

Epiploic Appendagitis

Epiploic appendagitis is an inflammatory process caused by torsion of an epiploic appendage of the colon or spontaneous venous thrombosis of a draining appendageal vein [14]. Mobile appendages with a bulbous configuration and long pedicles are thought to be at increased risk of torsion, particularly during changes in posture and heavy exercise [15]. Epiploic appendagitis typically presents as acute focal abdominal pain with unremarkable physical examination or laboratory findings [14, 15]. Imaging will reveal an oval-shaped fat-attenuation paracolic mass with peripheral fat stranding, and some cases may show a central increased attenuation (dot sign), representing a thrombosed draining appendageal vein [14, 16] (Fig. 7). Treatment is generally conservative and includes medical management with analgesics because spontaneous resolution usually occurs within 1 week [16, 17].

Greater Omental Infarction

Infarction of the greater omentum may be secondary to idiopathic vascular thrombosis or torsion (Fig. 8). Primary torsion is predisposed by anatomic variation of omental configuration whereas secondary torsion, which is more common, is associated with preexisting abdominal pathology, such as adhesions, hernias, cysts, and tumors [18, 19]. This condition typically occurs on the right side, but it may be seen on the left. The predilection has been attributed to a congenital variant blood supply of the right portion of the greater omentum that predisposes it to venous thrombosis [20]. Patients typically present with nonspecific abdominal complaints [19]. Complications of omental infarction include development of abscesses, adhesions, or bowel obstruction. The imaging presentation is variable, ranging from subtle heterogeneous attenuation of omental fat to whirling fatty tissue around a vascular structure, which is more suggestive of omental torsion [16, 19] (Fig. 8). Both surgical and conservative medical management approaches have been described [16, 21]. If the diagnosis is made intraoperatively, the torsed omentum will typically be resected.

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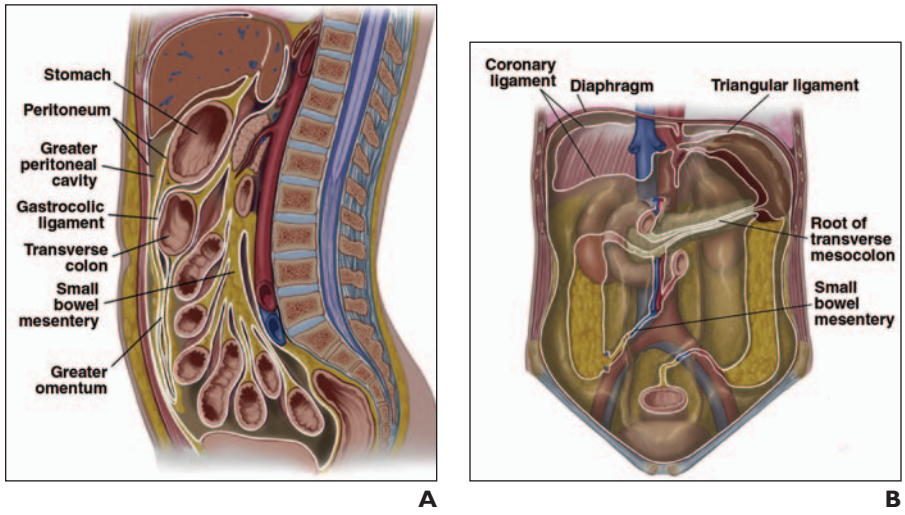


Fig. 1—Peritoneal attachments.
A and **B,** Sagittal (**A**) and coronal (**B**) section illustrations of abdominal cavity show mesenteric, ligamentous, and omental peritoneal attachments, which serve as anchors for intraabdominal viscera.

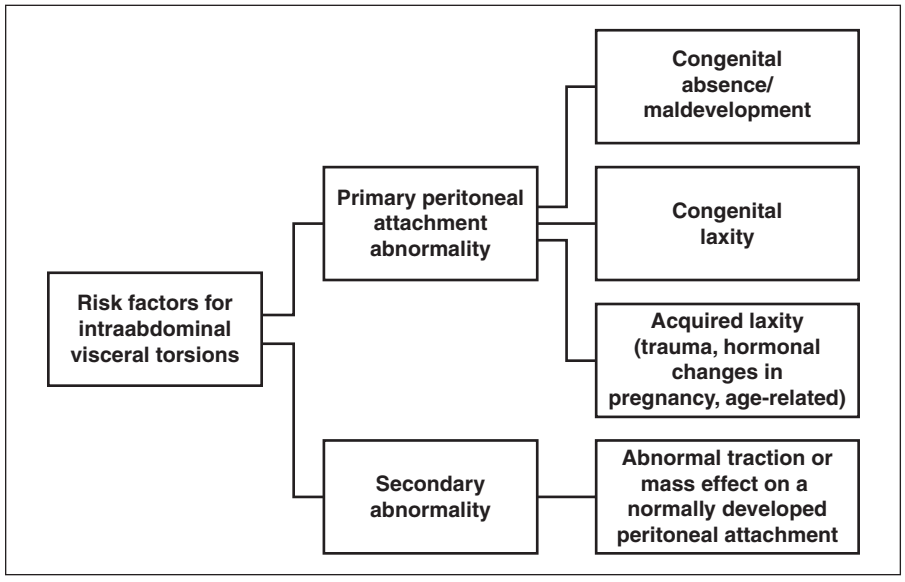
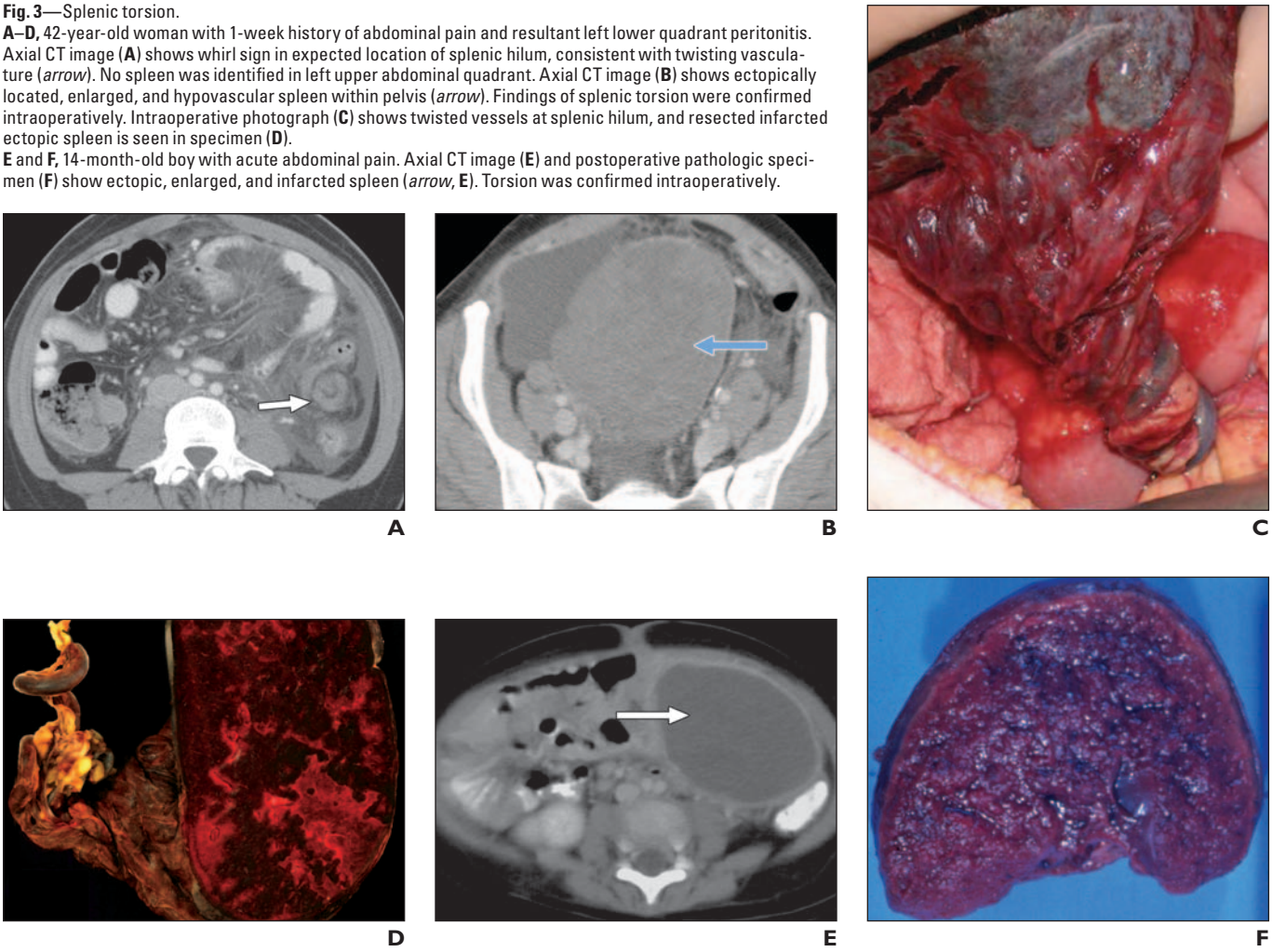


Fig. 2—Diagram shows overview of congenital and acquired peritoneal processes that increase risk for visceral torsion.

Fig. 3—Splenic torsion.
A–D, 42-year-old woman with 1-week history of abdominal pain and resultant left lower quadrant peritonitis. Axial CT image (**A**) shows whirl sign in expected location of splenic hilum, consistent with twisting vasculature (*arrow*). No spleen was identified in left upper abdominal quadrant. Axial CT image (**B**) shows ectopically located, enlarged, and hypovascular spleen within pelvis (*arrow*). Findings of splenic torsion were confirmed intraoperatively. Intraoperative photograph (**C**) shows twisted vessels at splenic hilum, and resected infarcted ectopic spleen is seen in specimen (**D**).
E and **F,** 14-month-old boy with acute abdominal pain. Axial CT image (**E**) and postoperative pathologic specimen (**F**) show ectopic, enlarged, and infarcted spleen (*arrow*, **E**). Torsion was confirmed intraoperatively.



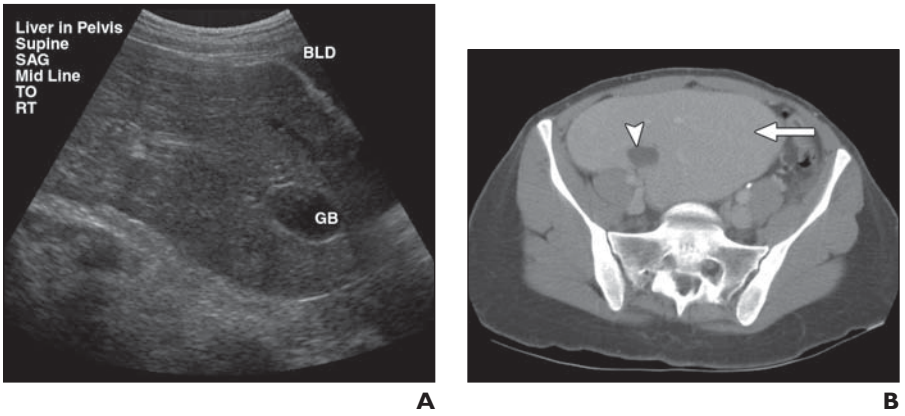


Fig. 4—22-year-old woman with history of omphalocele repair presenting with flank pain.
A and **B,** Pelvic ultrasound (**A**) and axial CT (**B**) images show ectopically located liver (*arrow*, **B**) and gallbladder (*arrowhead*, **B**) within lower pelvis. Liver conforms to shape of pelvis and was favored to represent chronic incidental finding unrelated to patient's chief complaint. GB = gallbladder, BLD = bladder.

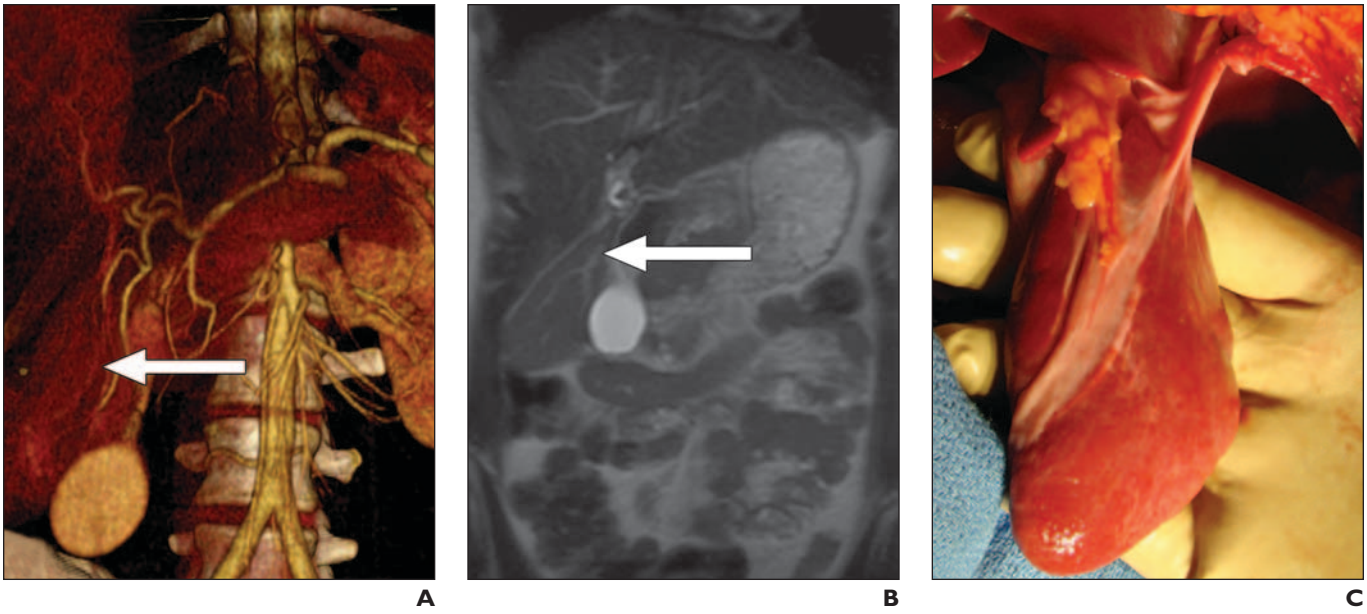


Fig. 5—Torsion of accessory hepatic lobe.
A–C, 24-year-old woman with chronic intermittent right upper quadrant abdominal pain. Volume rendered CT reconstruction (**A**) and coronal T2-weighted MR (**B**) images show hepatic appendage (*arrows*) attached to normal-appearing liver by narrow stalk. Notice subtle T2 hyperintensity of this appendage in **B** when referenced to normal-appearing liver. Accessory hepatic lobe torsion was confirmed on intraoperative photograph (**C**).
D and **E,** 12-year-old girl with 2-day history of right upper abdominal quadrant pain. Axial CT image (**D**) shows hypovascular mass (*arrow*) located adjacent to posterior margin of left hepatic lobe. Cut surface of resected gross specimen (**E**) shows hemorrhagic accessory hepatic lobe.

Fig. 6—84-year-old woman with fever and 2–3 days of right upper quadrant abdominal pain.
A and **B,** Axial (**A**) and coronal (**B**) CT images show enlarged edematous gallbladder (*arrow*) that is displaced from gallbladder fossa. Gallbladder torsion was confirmed intraoperatively.

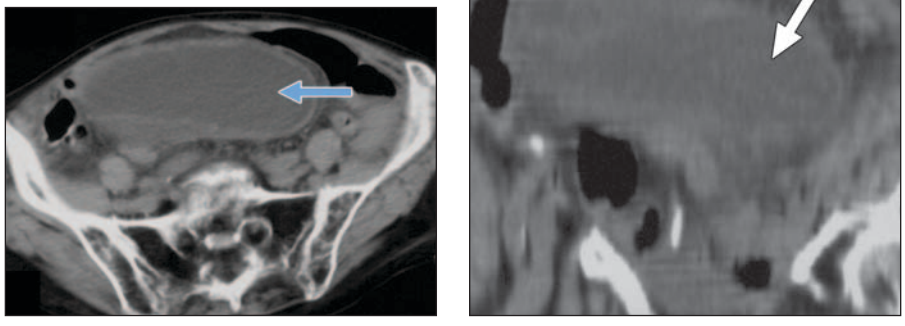
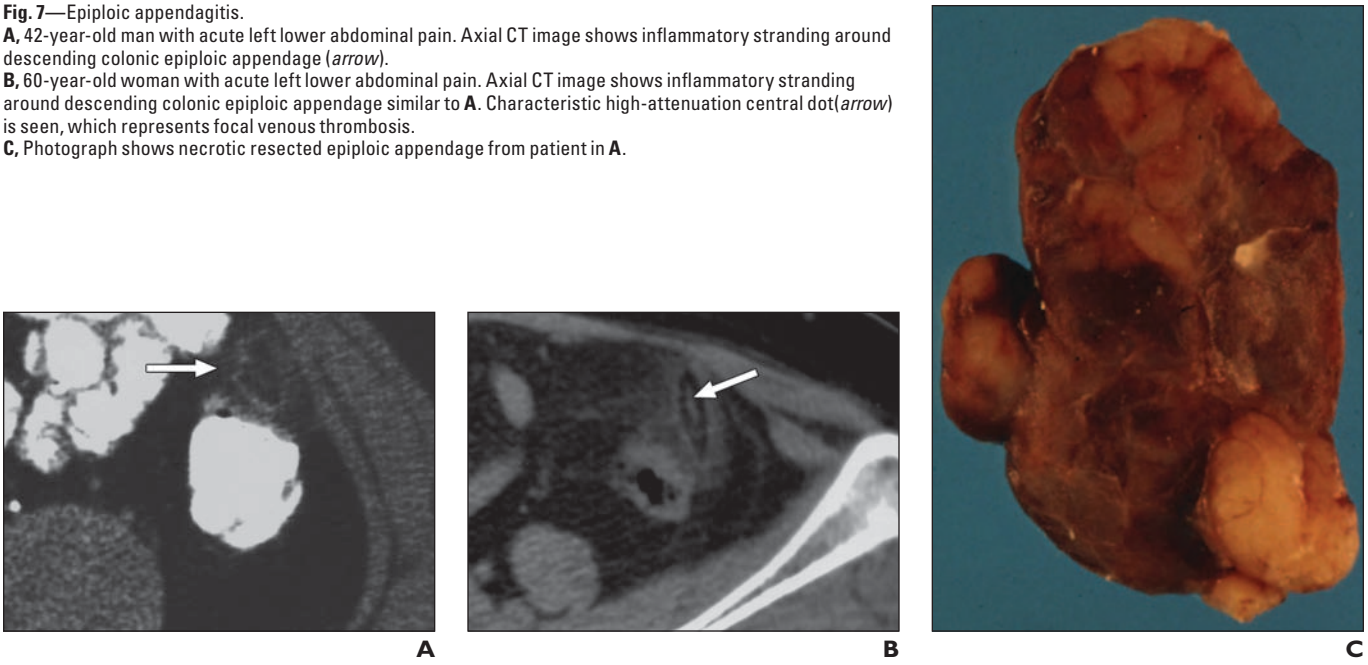


Fig. 7—Epiploic appendagitis.
A, 42-year-old man with acute left lower abdominal pain. Axial CT image shows inflammatory stranding around descending colonic epiploic appendage (*arrow*).
B, 60-year-old woman with acute left lower abdominal pain. Axial CT image shows inflammatory stranding around descending colonic epiploic appendage similar to **A**. Characteristic high-attenuation central dot(*arrow*) is seen, which represents focal venous thrombosis.
C, Photograph shows necrotic resected epiploic appendage from patient in **A**.



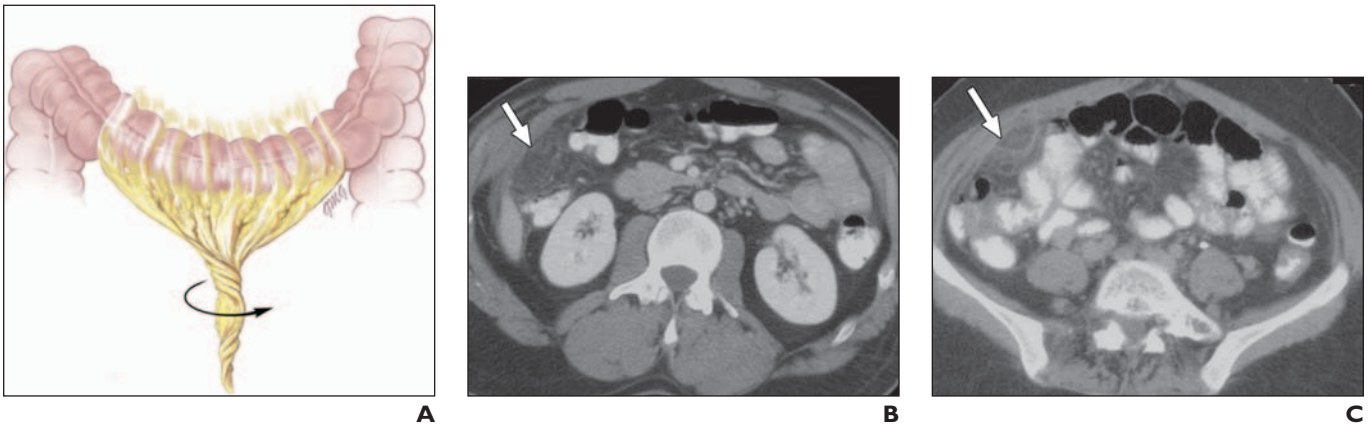


Fig. 8—Omental infarction.
A, Illustration shows torsion (*arrow*) of greater omentum.
B and **C,** Axial CT images from 30-year-old man with 3-day history of right lower quadrant abdominal pain. Heterogeneous increased attenuation of omental fat (*arrow*) is seen anterior to normal-appearing colon, findings that are consistent with omental infarction.