REVIEW ARTICLE

Imaging of Mischievous Intra-abdominal Fat Presenting with Abdominal Pain: A Pictorial Review

Ranjan K Patel[1], Shruti Mittal[2], Sapna Singh[3]

Abstract

Aim: To briefly discuss the imaging features of different types of intra-abdominal fat necrosis.

Background: Trauma and ischemic insult may result in intra-abdominal fat necrosis. Fat necrosis may present with acute abdomen, clinically simulating with other etiologies, such as acute diverticulitis and acute appendicitis.

Main body: Imaging plays a crucial role in making the exact diagnosis and differentiating it from other pathologies that may require surgical intervention. Computed tomography (CT) is the most commonly used imaging modality. A small fat attenuation lesion with a hyperattenuating rim in contact with the ventral surface of the sigmoid colon indicates epiploic appendagitis while a larger fat-attenuation lesion on the right side of the abdomen in between the colon and anterior abdominal wall indicates omental infarction. Fat stranding at the root of the mesentery with fat ring sign represents inflammatory mesenteric panniculitis while retractile or sclerosing mesenteritis appears as a fibrotic spiculated mass with or without calcification, mimicking mesenteric carcinoid. In patients with acute pancreatitis, the amount of inflamed fat correlates with clinical severity and outcome.

Conclusions: Familiarity with the imaging features of different types of intraabdominal fat necrosis helps in establishing an accurate diagnosis, thus avoiding unnecessary intervention.

Keywords: Acute pancreatitis, Epiploic appendagitis, Fat necrosis, Intra-abdominal, Mesenteric panniculitis, Omental infarction, Sclerosing mesenteritis.

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Background

Adipose tissue is a metabolically active tissue that plays an important role in metabolic syndrome.[1] Although not so common, fat necrosis is an important intra-abdominal pathology that may present with abdominal pain. Common processes include epiploic appendagitis, omental infarction, and fat necrosis in pancreatitis. Owing to difficulties in clinical diagnosis, imaging plays a crucial role in diagnosing and differentiating fat necrosis from other pathologies that clinically simulate fat necrosis.[2] Fat necrosis is managed conservatively while other pathologies like acute diverticulitis, acute appendicitis, and acute cholecystitis may need surgery.[3] This review aims to briefly discuss the pathogenesis, clinical presentation, and imaging features of the various types of intra-abdominal fat necrosis.

Main Text

Epiploic Appendagitis

Epiploic appendages are peritoneal pouches along the serosal surface of the colon that are composed of adipose tissue and blood vessels with a vascular stalk. Epiploic appendages are seen along the entire colon. Most numerous and larger appendages are seen along the sigmoid colon.[3]

Torsion of the epiploic appendages or spontaneous thrombosis of their central draining veins leads to ischemic or hemorrhagic infarction, resulting in epiploic appendagitis.[4] Epiploic appendagitis often occurs in the male of fourth to fifth decades of life. Obesity, unaccustomed exercise, and hernia have been associated with a higher incidence of epiploic appendagitis. Due to frequent involvement of appendages adjacent to the sigmoid colon, it most often presents as left lower quadrant pain, simulating acute diverticulitis.[5] Involvement of appendages along the caecum or ascending colon may present with right iliac fossa pain and mimic acute appendicitis. While it is self-limited in most instances, rarely it may result in peritonitis, abscess formation, adhesion, bowel obstruction, and intussusception.[3]

Epiploic appendagitis lacks pathognomonic clinical and laboratory features. Therefore, imaging plays a vital role to establish the diagnosis and importantly to rule out any other pathology that may require hospitalization, antibiotic therapy, and even surgical intervention.[5] Although ultrasonography has a low sensitivity in the diagnosis of epiploic appendagitis, USG at the region of maximum tenderness may reveal an oval noncompressible hyperechoic mass encircled by a subtle hypoechoic rim with no blood flow on Doppler imaging.[6] CT is the imaging modality of choice. CT shows a small (<5 cm) oval fat attenuation lesion, abutting the colonic wall with perilesional inflammatory changes (Fig. 1). A central hyperattenuating focus due to venous thrombosis also called as "central dot sign" if seen provides an important clue. Inflamed visceral peritoneal

[1–3]Department of Radiodiagnosis, Maulana Azad Medical College, New Delhi, India

Corresponding Author: Ranjan K Patel, Department of Radiodiagnosis, Maulana Azad Medical College, New Delhi, India, Phone: +91 8851228221, e-mail: ranjanair1@gmail.com

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Figs 1A to C: Acute left iliac fossa pain in a 40-year male. (A and B) Axial and coronal; (C) CECT images show a small oval shaped fatty lesion with a hyperattenuating rim abutting the ventral wall of sigmoid colon (yellow arrow) and associated surrounding fat stranding and mild reactive colonic wall thickening, suggestive of epiploic appendagitis

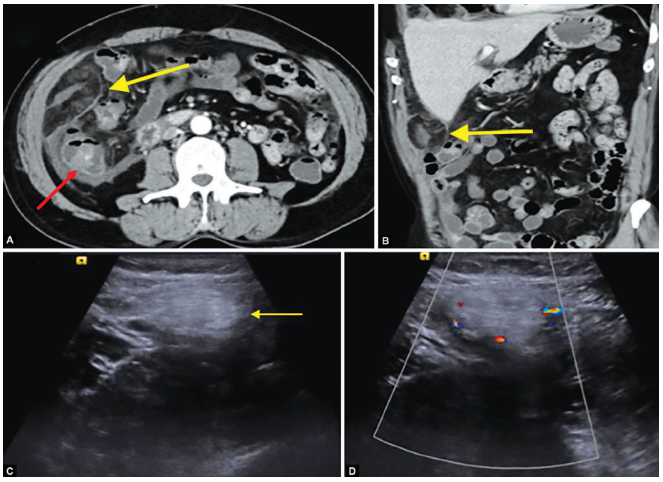
covering appears as a peripheral high attenuation rim, known as "hyperattenuating ring sign." Additional findings include thickening of the adjacent parietal peritoneum and colonic wall thickening due to reactive inflammatory changes. However, mesenteric fat stranding remains disproportionately severe as compared to adjacent bowel wall thickening. MR shows a focal lesion of fat signal intensity with an enhancing rim on postcontrast imaging.[3,5,6] In some cases, infarcted appendage detaches into the peritoneal cavity that subsequently calcifies. This calcified intraperitoneal loose seen on subsequent imaging is termed as "peritoneal mice."[5]

Omental Infarction

Omental infarction is more common in adults. However, around 15% of the omental infarction occurs in the pediatric population.[7] Venous insufficiency either due to trauma or thrombosis is the most common mechanism causing omental infarction. Different predisposing factors are obesity, strenuous exercise, abdominal trauma, and recent abdominal surgery. Omental infarction can be either due to primary or secondary omental torsion (Flowchart 1).[8] Clinically, omental infarction often presents as right-sided lower or upper quadrant pain, simulating acute appendicitis or acute cholecystitis, respectively. Higher incidence on the right side of the greater omentum is due to two reasons: (a) greater length and mobility of the right lateral free edge, making it more prone to torsion, and (b) more tenuous blood supply to the right lateral free edge.[6,8]

Imaging is crucial to exclude the mimics of omental infarction that may require surgery. At USG, omental infarction may appear as a focal area of echogenic fat at the site of focal tenderness (Fig. 2). It may turn into an abscess upon secondary infarction. At times, it may mimic a mass lesion.[9]

Omental infarction is often diagnosed on CT. Typically, it appears as a cake-like high attenuation fatty mass (usually >5 cm) located between the anterior wall of the colon and anterior abdominal wall, commonly on the right side (Fig. 2). Although reactive wall



Figs 2A to D: Omental infarction in a 50-year-old male presented to ER with a complaint of right-sided acute abdominal pain. Axial and coronal CECT images; (A and B) Show a large area of omental fat stranding on right in between colon and anterior abdominal wall (yellow arrows), associated colonic wall thickening (red arrows), and surrounding stranding noted; Transabdominal USG (C and D) images show a well-defined hyperechoic mass in the region of focal tenderness without obvious internal vascularity on color Doppler imaging

thickening may be seen on the adjacent bowel, fat stranding is disproportionately severe in omental infarction as compared to primary bowel pathologies.[3,8] Swirling of vessels may also be seen within the greater omentum.[6] The differences between epiploic appendagitis and omental infarction are summarized in Table 1.

Mesenteric Panniculitis

Mesenteric panniculitis represents inflammation of the mesenteric adipose tissue. The exact cause remains unknown; however, it is associated with other conditions, most commonly abdominal surgery and malignancy.[10] Depending upon the chronicity of the process, mesenteric panniculitis can be of two types: mesenteric panniculitis with only inflammation and degeneration of fat, and fibrotic form complicated by retraction of the surrounding structures, also known as "retractile or sclerosing mesenteritis."[11]

Mesenteric panniculitis is often asymptomatic but the symptomatic cases present with nonspecific symptoms. The most common symptom is abdominal pain. Other symptoms include nausea, vomiting, diarrhea, constipation, fever, and weight loss. The fibrotic or retractile form may be complicated by mesenteric ischemia, bowel obstruction, and perforation. Asymptomatic cases

Flowchart 1: Types of omental torsion according to etiopathogenesis

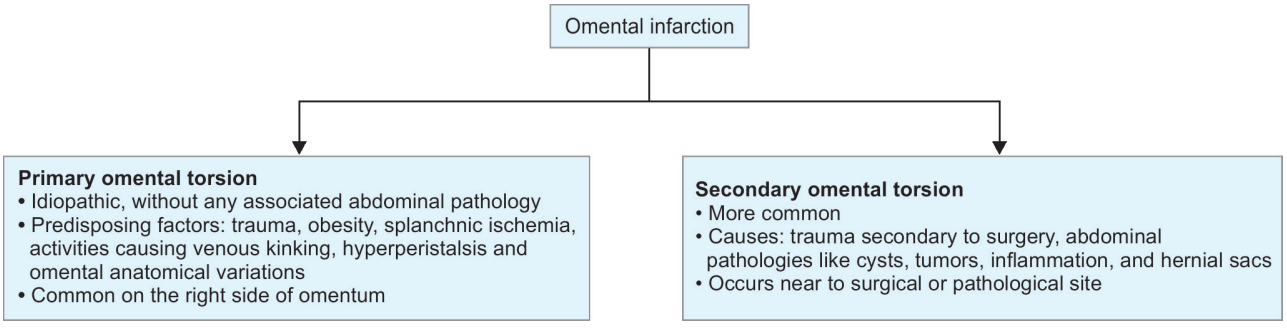
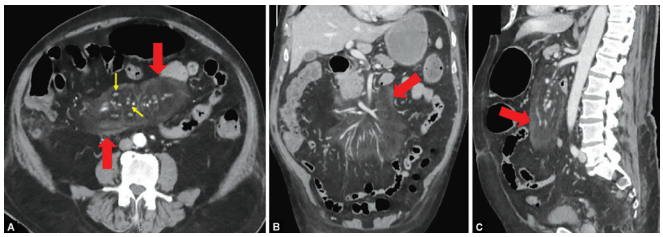


Table 1: Differences between epiploic appendagitis and omental infarction[3–8]

	Epiploic appendagitis	*Omental infarction*
Symptoms	Right lower and left lower quadrant pain	Right lower and upper quadrant pain
Classical location	Left side adjacent to sigmoid colon	Right side in between colon and anterior abdominal wall
Relation to colon	Abutting the colon surface	Epicentered in omentum, separated from colon
Size	<5 cm	>5 cm
Central dot sign	Seen	Not seen
Hyperattenuated rim	More common	Less common
Common differential diagnoses	Acute diverticulitis, acute appendicitis	Acute cholecystitis, acute appendicitis
Management	Mostly conservative	Mostly conservative

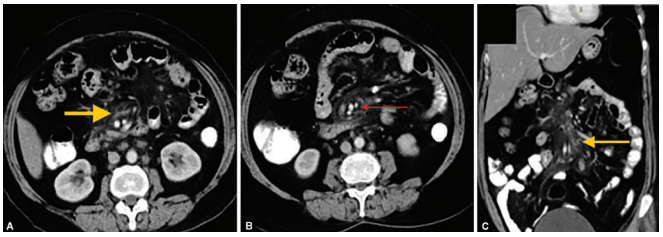


Figs 3A to C: Mesenteric panniculitis in a 42-year female who presented with acute lower abdominal pain 25 days after abdominal hysterectomy for endometrial carcinoma. CECT images (A to C) Show a well-defined area of misty mesentery at the mesenteric root surrounding the mesenteric vessels with a hyperdense peripheral pseudocapsule (red arrows) and relative sparing of fat around the vessels, giving "fat ring sign" (yellow arrows)

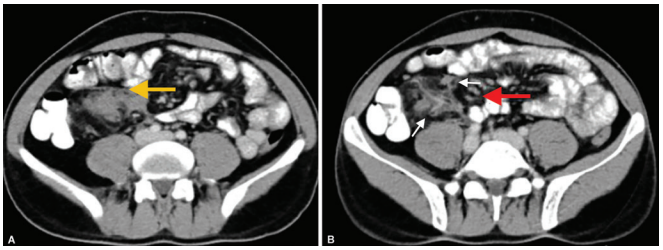
do not need any treatment while management of the symptomatic cases depends on the symptomatology.[11,12]

Imaging is the key to the diagnosis of mesenteric panniculitis. CT is the most commonly utilized for it. Classical imaging features of inflammatory mesenteric panniculitis consist of an inhomogeneous high attenuation fat (higher than the retroperitoneal fat, also called a "misty mesentery") at the mesenteric root with a thin (<3 mm) fibrotic pseudocapsule (Fig. 3). Small lymph nodes are often present in and around the involved fat. Preservation of a rim of fat around the vessels and lymph nodes inside the mass is also known as "fat ring sign" (Figs 4 and 5).[11–14] It is usually hypointense on T1 and hyperintense on T2-weighted MR sequences. Fat-saturated T2W sequence suppresses the normal fat, thereby making mesenteric panniculitis more conspicuous. The fibrous pseudocapsule appears hypointense on both T1 and T2W imaging with delayed enhancement on postcontrast images.[11]

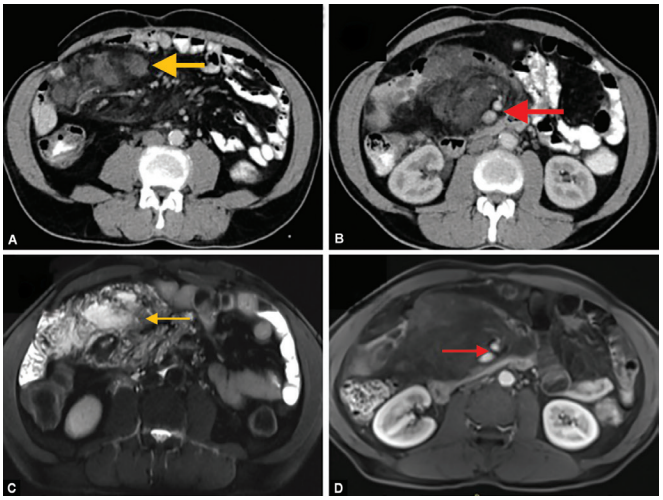
Retractile or sclerosing mesenteritis possesses a variable amount of fibrotic components. A lesion with a predominant fibrotic component appears as a solid mass with spiculation with or without retraction of the surrounding structures (Fig. 5). Punctate or coarse calcification may be seen in about 20% of cases. Fibrous pseudocapsule and fat ring sign may be absent.[11,15] On magnetic resonance imaging (MRI), the fibrotic mass exhibits hypointense



Figs 4A to C: Mesenteric panniculitis in another patient without any identifiable secondary cause. Axial CECT images (A and B) Show focal haziness of the small bowel mesentery producing "misty mesentery" sign (yellow arrows) with peripheral fat halo around mesenteric vessels giving "fat ring" sign (red arrow B)



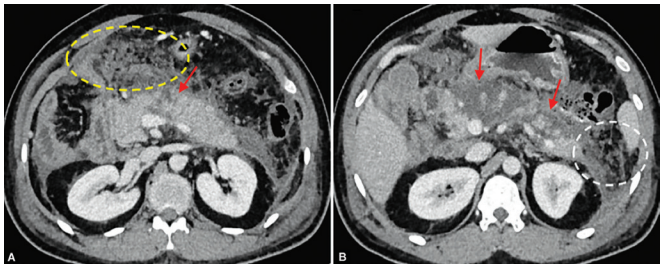
Figs 5A and B: Sclerosing or retractile mesenteritis in a 35-year female patient who present with chronic nonspecific pain in the right lumbar region since 2 months. Axial (A and B) CECT images depict a soft tissue mass involving small bowel mesentery with an irregular spiculated margin (white arrow), peripheral pseudocapsule (yellow arrow) and sparing of perivascular fat, giving fat ring sign (red arrow)



Figs 6A to D: Sclerosing mesenteritis in a 63-year-old male presented with chronic vague abdominal pain since 3 months. Axial CECT images (A and B) Show ill-defined soft tissue lesion in the small bowel mesentery, showing misty mesentery and tumoral pseudocapsule. Axial T2-weighted (T2W) FS image; (C) Shows hyperintense signal while postcontrast T1-weighted (T1W) FS image, and (D) Shows mild delayed enhancement with relative sparing of perivascular fat, giving "fat halo sign"

signal on both T1 and T2W sequences with delayed contrast enhancement (Fig. 6).[16]

It is worth mentioning that mesenteric carcinoid may closely mimic retractile mesenteritis. Fat ring sign if present directs toward the diagnosis of mesenteritis while the presence of a hypervascular bowel mass and hepatic metastasis indicate a carcinoid tumor.[11,13]



Figs 7A and B: Axial CECT images (A and B) Show acute necrotizing pancreatitis (red arrows) with peripancreatic inflammatory changes. Multiple clustered tiny nodules are seen in the mesenteric (yellow circle A) and retroperitoneal fat (white circle B) with surrounding fat stranding, suggesting fat necrosis

Table 2: Differences between encapsulated fat necrosis and liposarcoma[6,19–21]

	Encapsulated fat necrosis	*Liposarcoma*
Tenderness	Present	Absent
History of surgery	Often present	Usually absent
Temporal evolution	Gradually decreases	Gradually increases
Local invasion	Absent	Present

Fat Necrosis in Pancreatitis

Release of lipolytic enzymes in pancreatitis results in saponification of the pancreatic and peripancreatic fat. Damaged fatty tissues initiate a cascade of macrophage activation and release of inflammatory mediators which in turn perpetuate the inflammatory response. This explains the correlation between the amount of fat involved and severity and outcome in patients with acute pancreatitis.[17] A recent study proposed a fat-modified CT severity index (FMCTSI) taking into account the amount of total and visceral fat in addition to the modified CT severity index (CTSI). The study found FMCTSI a better predictor of severity and outcome compared to modified CTSI.[18]

The process of fat necrosis may result in the formation of multiple nodules scattered in the peripancreatic and mesenteric regions (Fig. 7).[6] These are usually noticed after the resolution of acute exudate and ascites in pancreatitis. A clinical history or previous imaging that demonstrates pancreatitis helps in differentiating nodular fat necrosis from the peritoneal carcinomatosis on imaging.[19,20]

Encapsulated Fat Necrosis

Traumatic or ischemic injury causes fat necrosis that becomes encapsulated by a fibrous rim. The fibrous rim may enhance. Encapsulated fat necrosis may exert a mild mass effect and mimic a liposarcoma.[6,19,21,22] Table 2 summarizes the differentiation between focal fat necrosis and liposarcoma.

Conclusions

Imaging plays a crucial role in the evaluation of intra-abdominal fat necrosis, more importantly, to differentiate from other causes of abdominal pain that warrant surgical intervention. Among available modalities, CT is the most commonly used. Detailed knowledge about the imaging features helps in making the exact diagnosis.

Clinical Significance

Basic idea about the imaging findings of different types of intra-abdominal fat necrosis helps in reaching a specific diagnosis and to avoid unnecessary surgical intervention.

Declarations

Ethical Approval and Consent to Participate

This is an educational pictorial review article and ethical approval was waived; consent to participate was not applicable.

Consent for Publication

Being an educational review, consent for publication was not required (not applicable).

Ethical Considerations

This article followed all ethical standards for research.

Orcid

Ranjan K Patel https://orcid.org/0000-0003-4780-5810

References

1. Grundy SM. Adipose tissue and metabolic syndrome: too much, too little or neither. Eur J Clin Invest 2015;45(11):1209–1217. DOI: 10.1111/eci.12519.
2. Aguilar-García JJ, Alcaide-León P, Vargas-Serrano B. Necrosis grasa intraabdominal [Intraabdominal fat necrosis]. Radiologia 2012;54(5):449–456. DOI: 10.1016/j.rx.2011.07.006.
3. Singh AK, Gervais DA, Hahn PF, et al. Acute epiploic appendagitis and its mimics. Radiographic 2005;25(6):1521–1534. DOI: 10.1148/rg.256055030.
4. Schnedl WJ, Krause R, Tafeit E, et al. Insights into epiploic appendagitis. Nat Rev Gastroenterol Hepatol 2011;8(1):45–49. DOI: 10.1038/nrgastro.2010.189.
5. Giannis D, Matenoglou E, Sidiropoulou MS, et al. Epiploic appendagitis: pathogenesis, clinical findings and imaging clues of a misdiagnosed mimicker. Ann Transl Med 2019;7(24):814. DOI: 10.21037/atm.2019.12.74.
6. Kamaya A, Federle MP, Desser TS. Imaging manifestations of abdominal fat necrosis and its mimics. Radiographics 2011;31(7):2021–2034. DOI: 10.1148/rg.317115046.
7. Esposito F, Di Serafino M, Mauro A, et al. Not only fat: omental infarction and its mimics in children. Clinical and ultrasound findings: a pictorial review. J Ultrasound 2020;23(4):621–629. DOI: 10.1007/s40477-020-00492-5.
8. Tonerini M, Calcagni F, Lorenzi S, et al. Omental infarction and its mimics: imaging features of acute abdominal conditions presenting with fat stranding greater than the degree of bowel wall thickening. Emerg Radiol 2015;22(4):431–436. DOI: 10.1007/s10140-015-1302-0.
9. Baldisserotto M, Maffazzoni DR, Dora MD. Omental infarction in children: color Doppler sonography correlated with surgery and pathology findings. AJR Am J Roentgenol 2005;184(1):156–162. DOI: 10.2214/ajr.184.1.01840156.
10. Issa I, Baydoun H. Mesenteric panniculitis: various presentations and treatment regimens. World J Gastroenterol 2009;15(30):3827–3830. DOI: 10.3748/wjg.15.3827.

11. Buragina G, Magenta Biasina A, Carrafiello G. Clinical and radiological features of mesenteric panniculitis: a critical overview. Acta Biomed 2019;90(4):411–422. DOI: 10.23750/abm.v90i4.7696.
12. Hussein MR, Abdelwahed SR. Mesenteric panniculitis: an update. Expert Rev Gastroenterol Hepatol 2015;9(1):67–78. DOI: 10.1586/17474124.2014.939632.
13. Horton KM, Lawler LP, Fishman EK. CT findings in sclerosing mesenteritis (panniculitis): spectrum of disease. Radiographics 2003;23(6):1561–1567. DOI: 10.1148/rg.1103035010.
14. McLaughlin PD, Filippone A, Maher MM. The "misty mesentery": mesenteric panniculitis and its mimics. AJR Am J Roentgenol 2013;200(2):W116–W123. DOI: 10.2214/AJR.12.8493.
15. Sabaté JM, Torrubia S, Maideu J, et al. Sclerosing mesenteritis: imaging findings in 17 patients. AJR Am J Roentgenol 1999;172(3):625–629. DOI: 10.2214/ajr.172.3.10063848.
16. Ezhapilli SR, Moreno CC, Small WC, et al. Mesenteric masses: approach to differential diagnosis at MRI with histopathologic correlation. J Magn Reson Imaging 2014;40(4):753–769. DOI: 10.1002/jmri.24690.
17. Noel P, Patel K, Durgampudi C, et al. Peripancreatic fat necrosis worsens acute pancreatitis independent of pancreatic necrosis via unsaturated fatty acids increased in human pancreatic necrosis collections. Gut 2016;65(1):100–111. DOI: 10.1136/gutjnl-2014-308043.
18. Gupta P, Dawra S, Chandel K, et al. Fat-modified computed tomography severity index (CTSI) is a better predictor of severity and outcome in patients with acute pancreatitis compared with modified CTSI. Abdom Radiol (NY) 2020;45(5):1350–1358. DOI: 10.1007/s00261-020-02473-y.
19. Chen H, Tsang Y, Wu C, et al. Perirenal fat necrosis secondary to hemorrhagic pancreatitis, mimicking retroperitoneal liposarcoma: CT manifestation. Abdom Imaging 1996;21(6):546–548. DOI: 10.1007/s002619900123.
20. Pedrosa I, Naidich JJ, Rofsky NM, et al. Renal pseudotumors due to fat necrosis in acute pancreatitis. J Comput Assist Tomogr 2001;25(2):236–238. DOI: 10.1097/00004728-200103000-00014.
21. Takao H, Yamahira K, Watanabe T. Encapsulated fat necrosis mimicking abdominal liposarcoma: computed tomography findings. J Comput Assist Tomogr 2004;28(2):193–194. DOI: 10.1097/00004728-200403000-00007.
22. Andaç N, Baltacioglu F, Cimşit NC, et al. Fat necrosis mimicking liposarcoma in a patient with pelvic lipomatosis. CT findings. Clin Imaging 2003;27(2):109–111. DOI: 10.1016/s0899-7071(02)00519-3.