Surg Today (2007) 37:150–153
DOI 10.1007/s00595-006-3345-z

SURGERY TODAY

Epiploic Appendagitis: Report of Two Cases

RUPJYOTI TALUKDAR[1], NRIPEN SAIKIA[1], SUBHASISH MAZUMDER[1], CHANDRESH GUPTA[1], SUDEEP KHANNA[1], DEB CHAUDHURI[2], S.S. BHULLAR[2], and ARUN KUMAR[1]

Departments of [1]Gastroenterology and [2]Radiology, Pushpawati Singhania Research Institute, Sheikh Sarai Phase II, Press Enclave Marg, New Delhi 110017, India

Abstract
Epiploic appendagitis (EA) refers to primary or secondary inflammatory disease of the epiploic appendages: peritoneal pouches of subserosal fat, which run in parallel rows beside the taenia coli of the colon. It is an uncommon but self-limiting condition, which often mimics acute appendicitis or diverticulitis. An accurate diagnosis of EA can be made by performing an abdominal computed tomography scan. Establishing a correct preoperative diagnosis is important to avoid unnecessary exploratory laparoscopy or laparotomy. We report two cases of EA, which to our knowledge represent the first documented cases from India.

Key words Epiploic appendage · Inflammation · Torsion

Introduction

The epiploic appendages are peritoneal pouches of subserosal fat running in parallel rows next to the taenia coli of the colon. Epiploic appendagitis (EA), which is any primary or secondary inflammation of the epiploic appendages, commonly manifests as lower quadrant abdominal pain mimicking acute appendicitis or diverticulitis. Exploratory laparoscopy or laparotomy can easily be avoided by diagnosing this self-limiting condition preoperatively. We report two cases of this uncommon condition.

Reprint requests to: R. Talukdar
Received: April 7, 2006 / Accepted: June 22, 2006

Case Reports

Case 1

A 34-year-old, previously healthy man presented to our emergency department with a 5-day history of severe left lower abdominal pain. The pain was continuous and nonradiating, with no aggravating or relieving factors. He had no nausea or vomiting and no bowel or urinary complaints. His medical history was unremarkable, his vital signs were all well within normal limits and he was afebrile. Abdominal examination revealed a soft non-distended abdomen without signs of peritonitis. There was a localized feeling of fullness with tenderness in the left iliac fossa. Bowel sounds were normal, as were the hernial sites and external genitalia. The rest of the systemic examination was normal. Laboratory data showed hemoglobin, 14.7 g/dl; total leukocyte count 7300/mm^3, with 64% polymorphonuclear leukocytes, 32% lymphocytes, 3% monocytes, and 1% eosinophils; erythrocyte sedimentation rate 32 mm after 1 h and platelet count, 1.47×10^5/mm^3. Liver function tests, serum electrolytes, and creatinine were all within the normal range. An ultrasound (US) of the abdomen showed an ill-defined, echogenic, focal lesion just below the peritoneum. Sigmoidoscopic examination showed a normal colon. A contrast-enhanced computed tomogram (CECT) of the abdomen showed a well-defined fat containing structure with central hyperdensity, arising from the serosal surface of the descending colon (Fig. 1). It was adherent to the visceral peritoneum and there was stranding of the adjacent mesentery. Based on the CECT findings and the absence of any pre-existing bowel disease, we made a diagnosis of primary EA. The patient was treated conservatively with non-steroidal anti-inflammatory drugs. He responded well and was discharged after 6 days.

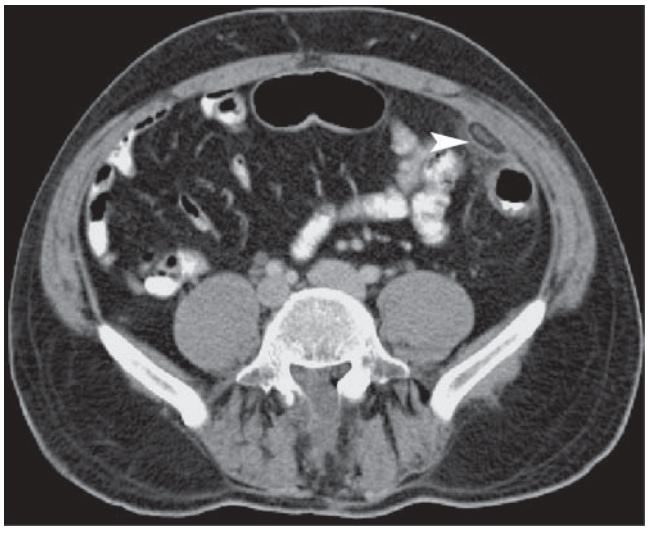

Fig. 1. Case 1. Contrast-enhanced computed tomography of the abdomen showing a well-defined fat-containing structure with central hyperdensity (*arrowhead*). The lesion is seen arising from the serosal surface of the descending colon adherent to the visceral peritoneum. There is also mesenteric stranding

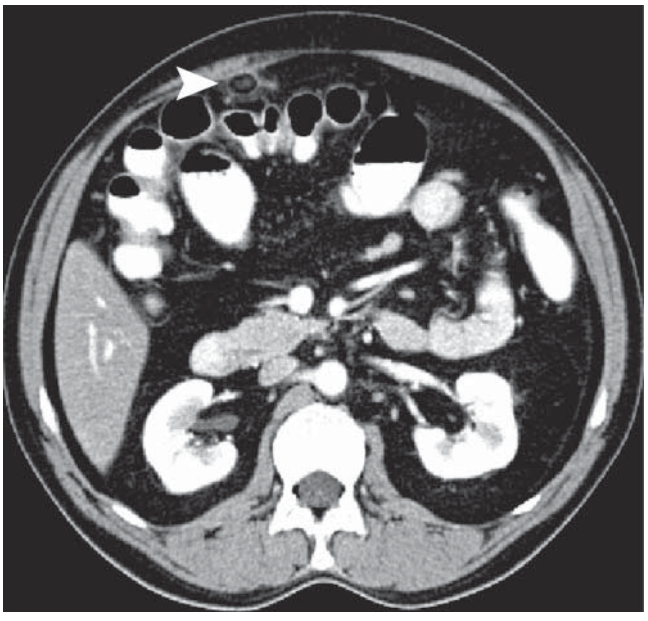

Fig. 2. Case 2. Contrast-enhanced computed tomography of the abdomen showing a well-defined fat-containing structure with central hyperdensity (*arrowhead*) arising from the transverse colon

Case 2

A 45-year-old man taking 5-aminosalicylic acid for Crohn's disease that had been in remission for 10 years presented at our emergency department with a 2-day history of right upper abdominal pain. The pain was colicky, nonradiating, had no referral, and was associated with nonprojectile bilious vomiting. There were no aggravating or relieving factors. He had not passed stools for 24 h. His vital signs were well within normal limits. Abdominal examination revealed a soft, distended abdomen with maximum tenderness in the right hypochondrium. There was minimal guarding but no rigidity. No abdominal viscera were palpable and the bowel sounds were exaggerated. His hernial sites and external genitalia were normal, as was the rest of the systemic examination. Laboratory data showed hemoglobin, 13.4 g/dl; total leukocyte count, 8400/mm^3, with 82% polymorphonuclear leukocytes, 15% lymphocytes, 1% monocytes, and 2% eosinophils; erythrocyte sedimentation rate 20 mm after 1 h and platelet count, 3.80 $\times$ 10^5/mm^3. Liver function tests, serum electrolytes, and creatinine were normal. An abdominal plain X-ray showed dilated small bowel loops with air fluid levels. Abdominal US also showed dilated small bowel loops in the periumbilical region. A CECT scan of the abdomen showed a narrowing in the ileocecal region with dilatation of the distal ileal loops. There was a well-defined fat containing structure with central hyperdensity arising from the transverse colon suggestive of EA, which was adherent to the visceral peritoneum (Fig. 2). Thus, we made a provisional diagnosis of subacute intestinal obstruction caused by distal ileal narrowing with EA. The patient was kept on nil orally and treated with antibiotics and nonsteroidal anti-inflammatory medication. He soon became pain-free and the obstruction was relieved by conservative management. He is now being followed up to investigate the cause of his distal ileal narrowing.

Discussion

First described by Vesalias in 1543,[1] epiploic appendages are peritoneal pouches of subserosal fat, which run in parallel rows next to the anterior and posterior taenia coli of the colon. These finger-like projections are enveloped by a serosal layer and are supplied by one or two small end arteries from the colonic vasa recta and drained by a tortuous vein through a narrow pedicle.[2,3] The embryologic development of epiploic appendages begins in the second trimester of fetal life[4] and they attain an average length of 3 cm (0.5–5.0 cm) and a thickness of 1–2 cm by adulthood, although they occasionally grow to as long as 15 cm.[5] Over 100 epiploic appendages span the entire colon, most of which are in the cecum and sigmoid colon. It is speculated that these structures may have a local bacteriostatic or anti-inflammatory function like a "miniature omentum," they may cushion the colon during peristalsis, and they may act as a res-

ervoir for blood when the colon and its blood vessels contract.[3,6] Some investigators have also suggested that epiploic appendages play a role in colonic absorption or serve as a fat-storing device.[1]

In 1853, Virchow suggested that the detachment of epiploic appendages was a source of intraperitoneal loose bodies.[7] The term "EA" was coined by Lynn et al. in 1956 to describe the primary inflammatory disease of these appendages.[8] The anatomy of the epiploic appendages makes torsion the most common cause of inflammation of these structures.[5] Torsion occurs along the long axis and is followed by edema, vascular compromise, thrombosis, and ischemia, finally culminating in gangrenous necrosis, which may give rise to focal peritoneal signs. Other causes of EA include spontaneous thrombosis of the draining vein, lymphoid hyperplasia, and bacterial invasion, occurring directly or secondary to diverticulitis.[9] Epiploic appendagitis may also develop secondary to Crohn's disease, appendicitis, cholecystitis, pancreatitis, or salpingitis.[10] Occasionally, EA results in peritoneal adhesions, leading to bowel obstruction.[6] Epiploic appendages may be involved in lipomatosis and metastatic disease and are sometimes incarcerated in a hernia.[4–6]

Epiploic appendagitis usually affects adults aged between 20 and 50 years, with an equal sex distribution,[10,13] although a few authors report a slight male preponderance.[7] Early studies projected a higher incidence in obese patients, but this was refuted in a subsequent larger series of 208 patients.[14] The most common presentation of EA is localized, nonradiating abdominal pain in the absence of severe illness. The pain usually involves the right lower quadrant (55%) despite the fact that 53% of infarcted appendages are found in the sigmoid colon.[15] Interestingly, Legome et al.[16] and Levret et al.[17] reported series in which 73% and 83% of patients, respectively, presented with left lower quadrant pain. Local peritonitis is uncommon and only 10%–30% patients have a palpable mass.[14] As the pain does not have any characteristic feature, it commonly mimics acute appendicitis or diverticulitis. However, the absence of migration and altered bowel habits clinically differentiates it from acute appendicitis and diverticulitis, respectively. Pain usually resolves within 1–2 weeks.

Before the description of US and CT criteria for EA, diagnosis was largely made by exclusion, or at laparotomy for acute abdomen. Currently, CT gives an accurate picture of EA, whereby unnecessary exploratory laparotomy can be avoided. A normal appendage is not visible unless there is free fluid in the abdomen. On US, an inflamed epiploic appendage appears as a noncompressible, solid, hyperechoic ovoid mass with a subtly hypoechoic rim located at the point of maximum tenderness.[18] The CT features are characteristic and include the following: a paracolonic oval fatty mass, representing the inflamed or infarcted appendage; a well-circumscribed hyperattenuated rim surrounding the mass, representing the inflamed visceral peritoneal lining; and occasionally, a high-attenuated central dot, representing a thrombosed or engorged central vessel or a central area of hemorrhage. Other findings include periappendageal fat stranding, parietal peritoneal thickening, and adjacent bowel compression.[19–23] The appearance of a lobulated mass signifies the presence of two or more contiguous infarcted appendages.[24] The CT features of omental infarction and AE are often similar, so they may be difficult to differentiate. The absence of a hyperattenuated rim and a central area of high attenuation increases the probability of omental infarction.[22] Moreover, omental infarction appears heterogeneous with a whorled pattern of fat stranding between the anterior abdominal wall and the transverse or ascending colon.[25] The anatomic location of pain in EA is adjacent to the colon, whereas that in omental infarction is centered in the omentum.

Epiploic appendagitis is a self-limiting illness which follows an uneventful clinical and radiological recovery with only analgesics.[19,20,26] Our first patient had clear primary EA, whereas our second patient had Crohn's disease in remission and a narrowed ileocecal region with features of small bowel obstruction. Since the site of obstruction was the ileocecal region, the inflamed appendage in the transverse colon was unlikely to have caused the obstructive symptoms.

In conclusion, EA is a self-limiting condition that resolves with symptomatic treatment and analgesia. Before the description of CT criteria, it was diagnosed at exploratory laparotomy. We presented these two cases to highlight the existence of this entity, emphasize its consideration in the differential diagnosis of acute, nonspecific abdominal pain, and to stress that it does not require surgery, but responds to conservative management. Diagnosing EA in the emergency department in a patient with acute abdomen will prevent unnecessary surgical intervention.

References

1. Fieber SS, Forman J. Appendices epiploicae: clinical and pathological considerations. Report of three cases and statistical analysis of 105 cases Arch Surg 1953;66:329–38.
2. Pines B, Rabinovitch J, Biller SB. Primary torsion and infarction of the appendices epiploicae. Arch Surg 1941;42:775–87.
3. Ross JA. Vascular loops in the appendices epiploicae. Br J Surg 1950;37:464–6.
4. Ghahremani GG, White EM, Hoff FL, Gore RM, Miller JW, Christ ML. Appendices epiploicae of the colon: radiologic and pathologic features. Radiographics 1992;12:59–77.
5. Singh AK, Gervais D, Rhea J, Mueller P, Noveline RA. Acute epiploic appendagitis in hernia sac. Emerg Radiol 2005;11: 226–7.

6. Kulacoglu H, Tumer H, Aktimur R, Kusdemir A. Epiploic appendagitis in inguinal hernia sac presenting as an inguinal mass. Hernia 2005;9:288–90.
7. Harrigan AH. Torsion and inflammation of the appendices epiploicae. Ann Surg 1917;66:467–8.
8. Ghosh S, Bilton JL Torsion and infarction of the appendices epiploicae: report of five cases. Dies Colon Rectum 1968;11:457–61.
9. Vinson DR. Epiploic appendagitis: A new diagnosis for the emergency physician. Two case report and a review. J Emerg Med 1999;17:827–32.
10. Lynn TE, Docherty MB, Waugh JM. A clinic pathologic study of the epiploic appendages. Surg Gynoecia Obstet 1956;103:423–33.
11. Legome EL, Sims C, Rio PM. Epiploic appendagitis: Adding to the differential of acute abdominal pain. J Emerg Med 1999;17: 823–6.
12. Kingenstein P. Some phases of the pathology of the appendices epiploicae. Surg Gynoecia Obstet 1924;38:376–82.
13. Desai HP, Tripod J, Gold BM, Burakoff R. Infarction of an epiploic appendage: review of the literature. J Clin Gastroenterol 1993;16:323–5.
14. Thomas JH, Rosoto FE, Patterson LT. Epiploic appendagitis. Surg Gynaecol Obstet 1975;138:23–5.
15. Vlahakis E. Torsion of an appendix epiploica of ascending colon. Med J Aust 1973;2:1148–9.
16. Legome EL, Belton AL, Murray RE, Rao PM, Noveline RA. Epiploic appendagitis: the emergency department presentation. J Emerg Med 2002;22:9–13.
17. Levret N, Mokred K, Quevedo E, Barret F, Pouliquen X. Primary epiploic appendagitis. J Radiol 1998;79:667–71.
18. Rioux M, Langis P. Primary epiploic appendagitis: clinical, US and CT findings in 14 cases. Radiology 1994;191: 523–6.
19. Rao PM, Wittenberg J, Lawrason JN. Primary epiploic appendagitis: evolutionary changes in CT appearance. Radiology 1997;204:713–7.
20. Torres GM, Abbit PL, Weeks M. CT manifestations of infarcted epiploic appendages of the colon. Abdom Imaging 1994;19: 449–50.
21. Van Breda Vriesman AC, Lohle PN, Coerkamp EG, Puylaert JB. Infarction of omentum and epiploic appendage: diagnosis, epidemiology and natural history. Eur Radiol 1999;9:1886–92.
22. Molla E, Ripolles T, Martinez MJ, Rosello E. Primary epiploic appendagitis: US and CT findings. Eur Radiol 1998;8: 435–8.
23. Singh AK, Gervais DA, Hahn PF, Rhea J, Mueller PR. CT appearance of acute appendagitis. AJR Am J Roentgenol. 2004;183: 1303–7.
24. Ng KS, Tan AG, Chen KK, Wong SK, Tan HM. CT features of primary epiploic appendagitis. Eur J Radiol 2006;59:284–8.
25. Singh AK, Gervais DA, Hahn PF, Sagar P, Mueller PR, Noveline RA. Acute epiploic appendagitis and its mimics. Radiographics 2005;25:1521–34.
26. Puylaert JB. Right-sided segmental infarction of the omentum: clinical, US and CT findings. Radiology 1992;185:169–72.