

A rare twist in the abdomen: diagnostic and therapeutic approaches to omental infarction - a case report and literature review

E. Ruts¹, M. Lefere², P. Bossuyt³

(1) Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp, Belgium; (2) Department of Radiology, Imelda General Hospital, Bonheiden, Belgium; (3) Department of Gastroenterology, Imelda General Hospital, Bonheiden, Belgium.

Abstract

Intraperitoneal focal fat infarction (IFFI) encompasses a group of rare conditions that are clinically and radiologically similar, arising from focal fatty tissue necrosis. These entities often mimic other acute abdominal conditions such as acute appendicitis or cholecystitis. We present the case of a 65-year-old female with progressive abdominal pain, ultimately diagnosed with IFFI using contrast-enhanced computed tomography (CT). Omental infarction (OI) was the leading diagnosis, though a definitive distinction from epiploic appendagitis (EA) could not be made on imaging. Conservative management with anti-inflammatory medication, analgesia, and low-molecular-weight heparins (LMWH) proved effective, resulting in complete resolution within five days. This case highlights the diagnostic value of CT-imaging in differentiating IFFI from other causes of acute abdomen, thereby avoiding unnecessary surgical interventions. The aetiology of OI will be discussed, along with a detailed focus on management strategies that may also apply to other causes of IFFI. (Acta gastroenterol belg., 2026, 89, 97-100).

Keywords: Abdominal infarction, omentum, ischaemia, acute abdomen, computed tomography, epiploic appendagitis.

Introduction

Intraperitoneal focal fat infarction (IFFI) comprises rare, predominantly self-limiting conditions of focal adipose necrosis, with infarction of the epiploic appendages being less extensive than that of the greater omentum (1,2,3). Omental infarction (OI) was first described by Eitel in 1899 (1). It predominantly affects male, middle-aged individuals, with a minority of cases reported in children. An infarction may theoretically occur in any abdominal region, mimicking a wide range of other causes of acute abdomen, but there exists a notable predilection for right-sided involvement (1,2). This case report outlines the differential diagnostic process for IFFI, with OI as the leading diagnosis. Based on a literature review, we will discuss the aetiology, treatment and clinical management.

Case history

A 65-year-old Caucasian female attended the emergency department with acute cramping abdominal pain in the right iliac fossa, onset one day prior. She was chronically taking daily pantoprazole 40 mg and allopurinol 300 mg. The patient did not consume alcohol, was a non-smoker and family history was unremarkable. Abdominal X-ray showed no signs of ileus or free air. The patient was discharged from the emergency after

administration of an enema. She was readmitted the day after, reporting increased cramping pain in the left iliac and umbilical region, aggravated by walking but not impacted by eating. There was no pain migration or radiation and she denied nausea, rectal bleeding, fever or night sweats. Flatus was still present.

On clinical examination, abdominal tenderness was noted in both iliac fossae, with localised muscle guarding and rebound tenderness.

Initial blood analysis revealed mild leucocytosis ($11.8 \times 10^3/\mu\text{l}$; norm: 3.5-11.0) with an elevated C-reactive protein (CRP) (13 mg/l; norm: <5). On readmission, CRP had risen to 200 mg/l. Gamma-glutamyl transferase was increased (272 U/l; norm: ≤ 60), though other liver function tests and lipase remained normal.

Abdominal CT with oral and intravenous contrast revealed a 'central dot' located on the cranial wall of the proximal transverse colon, distal to the hepatic flexure (Figure 1). This dense area, visualised in the venous phase, was indicative of a small venous thrombus. A peripheral rim of hyperattenuating mesenteric and omental fat tissue with streaky infiltration was observed, consistent with post-infarction inflammation. A multidisciplinary discussion favoured OI, though definitive radiological distinction from EA could not be achieved. Therefore, IFFI was deemed the more appropriate umbrella term.

A conservative approach was chosen, consisting of intravenous fluid therapy, non-steroidal anti-inflammatory drugs (naproxen 500 mg), analgesics as needed, and prophylactic nadroparin (3,800 international units once daily). After five days, the clinical symptoms resolved, and the patient was discharged. Prophylactic enoxaparin (40 mg subcutaneously) was prescribed for an additional two weeks.

Discussion

Aetiology

We present a case of IFFI in a female patient, with OI

Correspondence to: Dr. Peter Bossuyt, Imelda Ziekenhuis, Imeldalaan 9, 2820 Bonheiden, Belgium.

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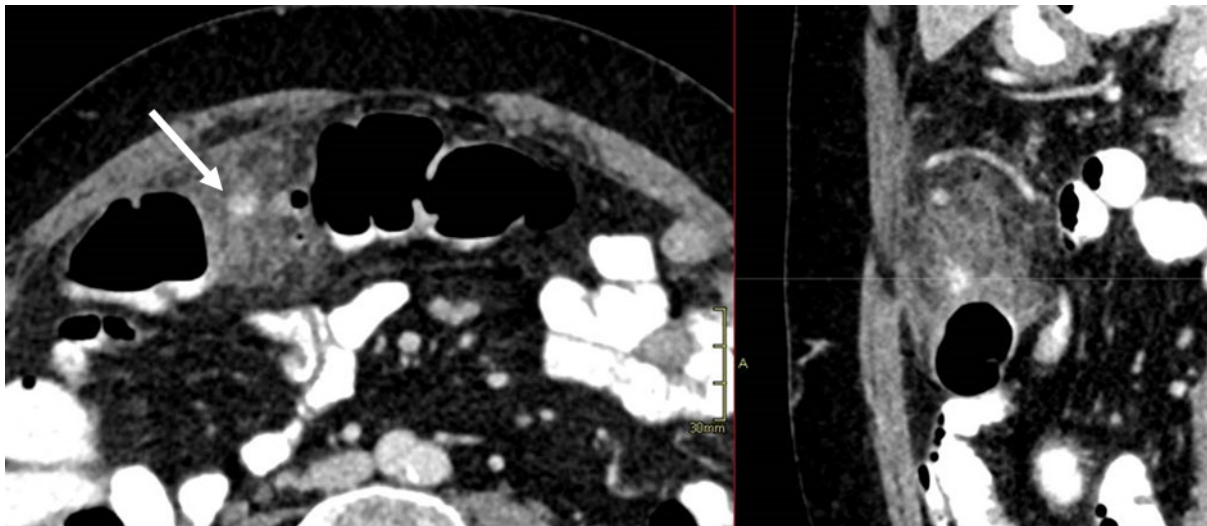


Figure 1. — Axial (a) and sagittal (b) CT images, acquired in the portal venous phase, show focal fatty infiltration adjacent to the transverse colon. A central hyperattenuated focus (arrow in a), lacking colonic lumen continuity, likely represents a thrombosed vascular pedicle.

as the leading diagnosis. OI can be classified as primary (idiopathic) or secondary, and with or without torsion (2). In idiopathic infarction, the aetiology remains unknown, although several reports suggest anatomical variants of the omentum—such as a bifid omentum, vascular variations including aberrant blood supply or venous kinking induced by strenuous activity or coughing, and irregular fat distribution within the omentum (2,4,6). Additionally, obesity is a significant risk factor for primary OI (1,2). A right-sided predilection of OI is observed, potentially associated with the greater length and mobility of the right omentum. Torsion of an omental segment can impede vascularity and often occurs idiopathically. However, torsion secondary to hernias, cysts, tumours, surgical scars or inflammation is typically located near the site of the underlying surgical or pathological condition (4). Non-torsional infarctions, as in this case, result from thrombosis and may be secondary to a hypercoagulable state, vasculitis, mesenteric venous congestion caused by right-sided heart failure or abdominal trauma (2). Regardless of the underlying mechanism, arterial or venous stasis and subsequent thrombosis develop, leading to oedema, congestion, haemorrhagic necrosis, and extravasation of serosanguineous peritoneal fluid.

Diagnosis

Omental infarction presents heterogeneously. It typically manifests as acute abdominal pain of increasing intensity without associated gastrointestinal symptoms. In our case, the pain—initially right-sided and later extending to the left iliac fossa—is most likely attributable to progressive ischaemia and subsequent post-ischaemic inflammation. Approximately 12% of patients present

with fever ($\geq 37.5^{\circ}\text{C}$) (1,2). Leucocytosis is a common finding and is often mild, typically $< 15000/\mu\text{l}$, and the erythrocyte sedimentation rate (ESR) and CRP levels may be elevated (1,4,6).

Historically, 90% of right-sided infarction cases were diagnosed intraoperatively when assessing patients for more common pathologies (i.e. appendicitis or cholecystitis) (1). Left-sided infarctions may mimic acute diverticulitis, EA and mesenteric panniculitis (4). Other differential diagnoses include ischaemic colitis and inflammatory bowel disease. Due to its rarity, OI is often not included in the clinical differential diagnosis during initial evaluation (6).

Contrast-enhanced CT is highly useful in the diagnostic workup, potentially preventing unnecessary surgical interventions (2,5,6). Typical CT signs of OI are a triangular or oval, heterogeneous fatty mass typically located between the anterior abdominal wall and the transverse or ascending colon (5). The affected fat shows stranding, and a central area of decreased attenuation may be visible, reflecting necrosis. A ‘whirling pattern’ may also be present, often raising suspicion for volvulus. However, the pattern seen in volvulus is typically more centrally located, as the torsion involves the larger vessels of the small bowel mesentery or mesocolon. In contrast, in OI the vascular swirling is more focal and less pronounced, involving smaller omental vessels. Furthermore, volvulus is usually accompanied by associated small-bowel or colonic abnormalities (e.g. distension, air–fluid levels) secondary to obstruction, which are absent in OI. Additional features such as free serosanguineous fluid may appear in advanced omental torsion, indicating venous congestion, fat necrosis, or aseptic peritonitis (6).

Differential diagnosis should be made with several common conditions. Diverticulitis typically involves bowel wall thickening, adjacent diverticula, and abscess formation, which are absent in OI. Reactive bowel wall thickening adjacent to an omental infarct is rare and lacks associated lymphadenopathy (4,5). Mesenteric panniculitis (MP) presents as a fatty, inhomogeneous mass with a hyperattenuating rim centred at the root of the jejunal mesentery, distinguishing it from the more peripheral location of OI (4).

Differentiating epiploic appendagitis from omental infarction can be challenging, though some features may be helpful. In EA, a <5 cm lesion typically arises near the caecum or sigmoid colon, avoiding the medial position seen in OI (3,6). It generally exhibits a hyperdense rim

or ‘ring sign’ due to thickened visceral peritoneum, a feature usually absent in OI (2,4). A ‘central dot sign’ is a central, hyperattenuating, ill-defined round area within a well-circumscribed fat-density ovoid lesion indicative of thrombosis linked with EA (3). In our case, this sign was visible on the venous phase; however, as it represents a focal thrombus, it would also appear hyperdense on a non-contrast CT (3). Although this finding supported EA, the absence of a ring sign and the location of the lesion on the proximal transverse colon suggested otherwise. Definitive differentiation between the two conditions cannot always be established. However, given their identical treatment and prognosis, the distinction holds no practical relevance, and the term IFFI is often used as common denominator (2). An overview of the

Table 1. — Differential diagnosis of omental infarction: key clinical, laboratory, and radiological features.

Condition	Clinical Features	Laboratory Findings	CT Findings
Omental infarction (1,2,4,5,6)	Acute abdominal pain, progressive over hours; often right-sided, may extend contralaterally; usually no GI symptoms; focal tenderness, low-grade fever in ~12%	Mild leucocytosis (<15,000/ μ L); ESR/CRP <u>may</u> be \uparrow	Triangular/oval heterogeneous fatty mass (>5 cm) between anterior abdominal wall and colon; fat stranding; central low attenuation (necrosis); focal vascular swirl; free serosanguinous fluid in advanced cases
Epiploic appendagitis (2,3,4,6,9)	Acute, localized, non-migratory pain (often left lower quadrant (LLQ) or near caecum); pain duration <30 min and radiating to lower back/groin; focal tenderness	Labs often normal or mild leucocytosis	<5 cm ovoid fat-density lesion abutting colon; ring sign (hyperattenuating rim); central dot sign
Acute appendicitis (4)	Right lower quadrant pain, pain migration, anorexia, nausea, fever, focal tenderness	Leucocytosis, neutrophilia	Enlarged appendix \geq 6 mm; mural thickening; periappendiceal fat stranding; possible appendicolith
Acute diverticulitis (4,5)	Acute LLQ pain; focal tenderness, fever	Leucocytosis; \uparrow CRP	Colon wall thickening; adjacent diverticula; possible abscess/fistula; associated lymphadenopathy
Mesenteric panniculitis (4, 10)	Subacute/chronic vague abdominal pain; abdominal distention, changed bowel habits, systemic symptoms variable	Nonspecific, sometimes mild leucocytosis; \uparrow ESR/CRP	Fatty, inhomogeneous mass with hyperattenuating rim centred at root of jejunal mesentery. Lymph nodes, tumoral pseudocapsule sign and fat halo sign
Ischaemic colitis (11)	Crampy abdominal pain, haematochezia, non-peritoneal abdominal tenderness	Leucocytosis; metabolic acidosis, \uparrow lactate	Segmental uniform colonic wall thickening; submucosal edema; hypoenhanced bowel wall, pericolonic fluid; pneumatosis/portal venous gas if severe
Intestinal volvulus (6)	Colicky abdominal pain, vomiting, abdominal distension	Labs nonspecific; leucocytosis or acidosis if ischaemic	Whirling pattern centrally located; dilated loops; transition point; ‘beak’ sign; mural hypoenhancement if ischaemic
Acute cholecystitis (2,6)	Right upper quadrant pain (RUQ), colicky; fever, nausea/vomiting; peritoneal tenderness RUQ, Murphy’s sign	Leucocytosis; \uparrow CRP; abnormal liver transaminases possible	Distended gallbladder; wall thickening; pericholecystic fluid; stones (variable)
Inflammatory Bowel Disease (12)	Chronic/recurrent abdominal pain; (bloody) diarrhea; systemic symptoms in flares; extraintestinal manifestations	\uparrow CRP, \uparrow ESR, anemia, hypoalbuminemia in flares	Bowel wall thickening; mucosal hyperenhancement; fat stranding; mesenteric ‘comb sign’ (Crohn’s); strictures, fistulas possible

differential diagnosis, including further details on the clinical, laboratory, and radiological features, is presented in Table 1.

Treatment

The medical literature is inconclusive regarding the treatment of OI. Traditionally, surgical resection has been the management for omental torsion. However, the use of CT-imaging enables the selection of conservative versus surgical treatment when appropriate (6). An increasing number of reports highlight conservative management as the preferred approach in most cases (1,6). The inflammatory process is thought to be self-limiting, typically resolving within two weeks. Conservative management with IV fluids, anti-inflammatory medication and analgesia may suffice (2). Additionally, we recommend the administration of LMWH, as OI is associated with low-flow vascular states (7). In more advanced presentations, laparoscopic resection of the necrotic segment has been associated with a shorter hospital stay, faster symptom resolution, less need for follow-up, and prevention of future complications such as abscess formation or intra-abdominal adhesions. Moreover, when conservative management fails, subsequent laparoscopic surgery is associated with a higher likelihood of conversion to laparotomy (1). However, unnecessary surgical trauma should be avoided. Ultimately, it is essential to carefully select the appropriate patients for surgical treatment in a multidisciplinary team setting. Predictive factors for failed conservative management include younger age and elevated white blood cell counts at admission, warranting a lower threshold for surgical intervention. Although a temperature $\geq 37.5^{\circ}\text{C}$ is not directly linked to failure of conservative treatment, it often leads to immediate surgical intervention (1).

Finally, since thrombosis in non-torsional infarctions occurs in unusual vascular beds, we recommend screening for hereditary thrombophilia (deficiencies in protein S, protein C, and antithrombin, Factor V Leiden, and Factor II mutation) and antiphospholipid syndrome (8).

In conclusion, OI remains a rare but important cause of acute abdominal pain. This case highlights the crucial role of contrast-enhanced CT-imaging in distinguishing IFFI from mimicking conditions. Multidisciplinary input is essential to guide the selection of the appropriate treatment strategy. Further research is required to establish conclusive treatment recommendations and improve strategies for managing complications and thrombotic risks in OI.

Declarations

Conflict of interest statement: The authors have no relevant conflicts of interest to declare.

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