RADIOLOGIC PATHOLOGIC CORRELATION / Gastrointestinal imaging

Radiological, clinical and histological correlations in a right segmental omental infarction due to primary torsion in a child

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Greater omental infarction is an uncommon cause of acute abdominal pain in children. It may be primary or secondary to torsion and can involve all or part of the omentum. Its basic histological findings allow us to understand its pathophysiology. Despite the relatively non-specific clinical presentation and the fact that it is relatively poorly understood, the widespread use of ultrasound and technical improvements in CT usually allow it to be diagnosed accurately. The approach to treatment is still contentious and while some patients benefit from laparoscopic resection of the necrotic omentum, many articles describe the merits of a conservative approach.

We describe a case of right greater omental segmental infarction due to primary torsion in a 12-year old boy diagnosed on imaging and treated by laparoscopic surgery, which rapidly resolved his symptoms.

Case report

A 12-year-old boy presented with moderate, isolated, right flank pain, which had developed gradually over 4 days. His clinical history did not reveal any triggering factors responsible for his symptoms and at the consultation the pain had become constant and was slightly worsened by movements and coughing. He had no bowel transit problems, nausea, and vomiting or urinary symptoms.

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Physical examination showed a slightly overweight patient with a temperature of 37.3°C with diffuse abdominal tenderness in the right quadrants and an exquisitely painful point in his right flank, with no palpable mass. His bowel sounds were present and he had no hernias. He had no systemic signs, particularly organomegaly or lymphadenopathy.

His white cell count was 8690 Giga/L [4000–10,000], including 5610 Giga/L leucocytes [1500–7000] with a C-reactive protein of 3 mg/L [<5]. His other routine investigations were normal and a urine test strip was negative.

Abdominal ultrasound showed painful infiltration of the deep abdominal fat with a hyperechogenic image (Fig. 1) 13 cm along its long axis, which was ovoid, homogeneous and located in the right flank between the abdominal wall and ascending colon. The appendix was healthy and the investigation was otherwise unremarkable.

Because of the child’s radiological and clinical features, an abdominal and pelvic CT scan was performed and showed extensive heterogeneous infiltration of the deep abdominal fat in front of the right colon, extending from the hypochondrium to the iliac fossa. This was 14 cm long and contained linear structures with a whirl sign around a main vein, the gastro-colonic trunk (Figs. 2 and 3). He had slight enhancement of the parietal peritoneum and thickening of the wall of the right colon.

As the boy did not have appendicitis or systemic signs and his pain was only moderate, he was started on medical treatment with analgesics, antibiotics and hydration. After 3 days, (i.e. 7 days from the time his symptoms started), he had not improved and an MRI was performed which showed a large area of heterogeneous infiltration in the deep abdominal fat in front of the ascending colon. This was hyperintense on T1, T2 and diffusion-weighted sequences and hypointense after fat suppression. It was peripherally enhanced after gadolinium injection (Fig. 4). No gastro-intestinal wall abnormalities or deep collection were present.

A diagnosis of right segmental greater omental infarction due to torsion was proposed and he was treated laparoscopically with resection of the infarcted omentum. Inspection of the abdominal cavity showed (Fig. 5):

- a large yellowish mass in the right flank with areas of loss of coloration, representing the pathological omentum and signs of venous distress with a combination of loss of color, a purplish appearance and venous congestion;
- torsion around its vascular pedicle;
- an adhesion to the anterior abdominal wall;
- a small free serohematic peritoneal effusion.

Histological examination confirmed a diagnosis of primary segmental omental infarction with no tumor cells. Bacteriology samples were negative.

The boy had improved by the next day and was able to leave a few days after the procedure.
Discussion

Omental infarction is an uncommon cause of acute abdominal pain, which is responsible for approximately 15% of the cases in children [1]. It is found in between 0.024% and 0.1% cases of surgery for suspected appendicitis [1,2]. Boys are more commonly affected than girls (M/F ratio 3:1) due to the fact that boys have more fat accumulation and therefore, a thicker greater omentum than girls [1]. Children under 4 years old do not appear to be affected as they have too little fat in their omentum [3,4]. The number of cases reported is increasing because of more widespread use of CT in abdominal emergencies and increasing numbers of overweight or obese children, which is thought to be a strong predisposing factor for the condition [4]. Our patient was overweight with a BMI of 22.5 (> 97th percentile).

Regardless of its origin and mechanism, greater omental infarction occurs as a result of venous stasis and/or thrombosis, causing edema, congestion and then early infarction. Ischemic arterial infarctions only occur secondarily. They result from distal ischemia due to microcirculatory compression or strangulation of a vascular pedicle aggravated by the infiltration of blood and edema [5]. When torsion occurs, the first right omental vein, a branch of the gastro-colonic trunk is generally the pivot point around which the mobile segment of omentum twists [5–7].

Our literature review did not find any histological studies on omental infarction. The pathophysiology of the disorder

Figure 3. Whirl sign in the centre of the fat mass on the lower image.

Figure 4. a: hyperintense mass on T2 sequence because of the edema and venous congestion; b: hyperintense mass on a diffusion sequence with reduced CDA (reduced diffusion in congested and/or thrombotic blood vessels); c and d: peripheral enhancement following gadolinium injection. Note that the wall of the right colon is normal in all of the sequences.
can be better understood from the various histological and cytological findings:

- **venous congestion** represented by dilated veins, the lumen of which are full of red blood cells and thrombosis. These are responsible for the edema and then omental congestion (early infarction) which progresses to hemorrhagic necrosis (infarction) (Fig. 6);
- **hemorrhagic suffusion** is seen with extravasation of red blood cells into the interstitial sector;
- the resulting adiponecrosis is made up of empty, ghost-like adipocytes which have lost their peripheral nuclei and whose walls are strongly eosinophilic on HES staining (Fig. 7a, b);
- **benign mesothelial hyperplasia** may be seen, representing thickening and stratification of the serosal membrane of the gastro-intestinal tract in response to injury (inflammation, ischemia, infection...);
- up to 6–7 days from the onset of symptoms before surgical excision, signs of inflammation are seen with granulomatous infiltrates containing neutrophils and to a lesser extent monocytes and lymphocytes.

![Figure 5](image1.png) Upper appearances: parietal adhesion (curved arrow) and dilated congested vein (straight arrow). Lower images: infarcted omentum, purple colored with thrombosed veins (straight arrows), intense inflammatory reaction in the parietal peritoneum next to the adhesion (short arrow) and hemorrhagic suffusion.

![Figure 6](image2.png) Venous congestion in the omental fat associated with fibrous inflammatory scarring changes in the adipose tissue (HES staining, ×5 magnification).
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These are predominantly found in the perivascular areas;
• after this period, signs of fibrosis appear with fibroblast infiltration, producing collagen which may cause disorganization of the omental adipocyte tissue (Figs. 7c, d and 8);
• finally, the peritoneal fluid cytology shows benign mesothelial cells and neutrophils. No tumor cells are present and bacteriology samples are negative.

The features found in our patient according to the history of the disorder (pre-operative diagnosis 4 days after the onset of symptoms, unsuccessful conservative approach and then surgery at 7 days):
• basic lesions: adiponecrosis, venous congestion, hemorrhagic suffusion, peritoneal fluid cytology rich in benign mesothelial cells;
• very few inflammatory cells with occasional perivascular monocytes and macrophages;
• sheets of fibrosis with bands of collagen, relatively extensive in some places;
• no signs of a lesion responsible: tumor cells were absent and bacteriology samples were negative.

Figure 7. a and b: ischemic adipocyte necrosis (ghost-like appearance with eosinophilic nuclei and cytoplasmic membranes) and intercellular edema (×20 and ×40 magnification); c: ischemic adipose tissue necrosis (above), chronic scarring inflammatory fibrosis (in the middle and below) ×10 in magnification); d: chronic scarring information of the adipose tissue with numerous fibroblasts and sheets of collagen (×20 magnification).

Figure 8. Edge of epiploic appendix with scarring fibroinflammatory changes in the adipose tissue and congested veins with no hyperplasia of the mesothelial lining (×10 magnification).
As was true of our case, in terms of macroscopic examination, the infarcted omentum takes on an inflammatory pseudo mass appearance, generally purplish in color with congested dilated veins, contrasting with the healthy omentum which is yellowish in color and pearly and contains narrow, normally oxygenated blood vessels. Some or all of the omentum may be necrotic and dark in color. An adhesion to the parietal peritoneum explains the intense, long lasting pain, which occurs both spontaneously and on palpation of the abdominal wall, and a free sero-hematic effusion may be found [8–10].

The causes of primary infarction with or without torsion have not been clearly established. Several studies [8–10] describe a role for anatomical variations, such as bisection, a bifid or accessory omentum, abnormal vascular supply and heterogeneous distribution of omental fat primarily in obese patients. The omental infarction may also be due to hyper-coagulability or vascular abnormalities or fragility promoting venous thrombosis or mesenteric vein congestion due to right heart failure and a cardiac liver [11]. Secondary torsion is due to scars and abdominal wall hernias, omental cysts or any other diseases, which may be associated with adhesions [5,8–10]. Precipitating factors causing displacement of the omentum have also been described, including a sudden increase in intra-abdominal pressure after a heavy meal, excess straining, sudden change in position, coughing or sneezing and recent abdominal surgery or trauma [4].

The clinical presentation is non-specific and generally involves acute pain in the right abdominal quadrants. Very few left segmental infarctions have been reported in children [12,13]. In 50% of the cases, the infarction is associated with fever, anorexia, nausea, vomiting and dysuria. Occasionally, as with our patient, the pain may change with movements, which is a suggestive factor for the diagnosis [1,2,14]. Physical examination reveals tenderness or even signs of peritoneal irritation. A mass may occasionally be palpable [9]. Understanding the abdominal innervation helps to explain the physical presentation: the parietal peritoneum in contact with the anterior abdominal wall shares somatic innervation with the contiguous muscles and skin [15]. Irritation of the parietal peritoneum can therefore cause contractions and muscle or even skin pain. In addition, because of its size and mobility, the diseased omentum may irritate the parietal peritoneum at many sites, as has been reported by Park et al. [9]. In our case, laparoscopy showed an area of adhesion of the necrotic omentum to the parietal peritoneum, which was intensely inflamed after being detached, at least partly, explaining the patient’s pain.

The laboratory findings are also non-specific and may be normal or may show neutrophil hyperleukocytosis and a moderate rise in the erythrocyte sedimentation rate and/or C-reactive protein [10].

Imaging of omental infarction (mostly ultrasound) was firstly described by Puyhaubert [16]. As with our patient, ultrasound shows uncompressible, ovoid, hyperechogenic appearances near the tenderest point [16,17]. This may be adherent to the anterior abdominal wall because of irritation of the parietal peritoneum and a small, free pleural effusion may be noted [6,16,17]. Although it is very specific, it is relatively insensitive (60 to 80% [17]) and the lesion may be easily missed on ultrasound. Ultrasound, however, remains the investigation of choice in pediatric abdominal emergencies and, most importantly, can exclude a clinically obvious cause.

When infarction occurs without torsion, a CT scan shows a fatty mass containing linear structures [3,6,16,18,19], representing the dilated thrombosed veins and/or sheets of fibrosis [17]. The masses are often deeply localised in front of the colon [6,16,19] and adhering to the parietal peritoneum. Peripheral enhancement is sometimes seen after contrast injection [3,6,18,19] and the parietal peritoneum is often inflamed, enhanced and thickened. Moderate thickening of the gastro-intestinal tract walls as a result of local extension of the inflammation and a small free peritoneal effusion may be seen [6,16,18,19]. If torsion is present, the “whirl sign” and “vascular pedicle sign” are described in addition to these findings [3,6,18]. These reflect the same effect: torsion of a portion of the omental fat accompanied by its vessels around an enhanced vascular pedicle which is enlarged in relation to the fixed point around which it has twisted [6]. Omental infarction is therefore easy to distinguish from its main differential diagnoses (acute appendicitis, cholecystitis, diverticulitis) [10] although it is more difficult to distinguish from appendigitis as both display a fatty mass: the main difference is the size of the lesion. A fatty mass within the omentum is also described in benign and malignant conditions, such as lipoma, angiomyolipoma, teratomas, mesenteric lipodystrophy, peritoneal pseudomoma, liposarcoma and peritoneal mesothelioma or in peritoneal metastases (usually following ovarian cancer) [6,9,16]. The differential diagnoses in children also include Meckel’s diverticulitis and mesenteric lymphadenitis [1–3,9].

Very few cases of omental infarction imaged by MRI have been described [9,12,19]. This shows a fat mass containing linear structures which are hyperintense on weighted T1 sequences with hypointense linear structures inside the mass (representing the congested mesenteric vessels with reduced flow or sheets of fibrosis) and hyper intense on T2 sequences because of venous congestion and edema [12,19]. In our case, MRI showed these basic findings and excluded disease of the ascending colon because of its thickness on the CT scan.

There are two approaches to treatment. Historically, the diagnosis and treatment of omental infarction were surgical. Since then, many studies have reported cases which have been improved with conservative treatment which is indicated when the diagnosis is not sufficiently certain and in the absence of adverse clinical or imaging features, particularly in secondary forms of infarction [10]. This involves close interval monitoring, analgesia, appropriate hydration and prophylactic antibiotic therapy. It is justified for the following reasons:

- there are no comparative studies between the two approaches;
- the surgical incision for suspected appendicitis is unsuitable for investigation and research on the infarcted omentum which is often located in the right upper quadrant of the abdomen;
- the complications of the conservative approach (particularly omental abscesses) are hypothetical, are not seen in children and can be prevented with prophylactic antibiotic therapy [6,9,14,20].
When the correct diagnosis is made by imaging, laparoscopy is preferred to conventional surgery [6,9,20], as it can:
- examine the whole abdomen;
- identify the diseased omentum, torsion and number of spirals;
- exclude a secondary cause, take bacteriology samples and check the viscera, particularly the colon, appendix and gallbladder;
- remove the infarcted omentum or just release the torsion and perform careful peritoneal lavage;
- minimize surgical invasion, post-operative pain and the complications of laparotomy [9,14,20].

**Conclusion**

Despite an increasing number of observations in children, primary greater omental infarction is still an uncommon cause of acute abdominal pain. There are few microscopic histological descriptions published in the literature. However, the existing descriptions are useful in understanding the pathophysiology and imaging appearances, particularly the possible changes in radiological features depending on the clinical evolution.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

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