

# Omental infarction in children: imaging features with pathological correlation

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## ABSTRACT

**Introduction:** Omental infarction is a rare occurrence in the paediatric population. It often presents as an acute abdomen that can mimic acute appendicitis and cholecystitis.

**Methods:** Six cases of omental infarction in children, proven on histopathology, were retrospectively reviewed for their clinical presentation and imaging findings on ultrasonography and computed tomography.

**Results:** These cases revealed clinical and imaging findings on computed tomography that were suggestive and helpful in the pre-operative diagnosis of omental infarction. Findings on ultrasonography were less specific. Histopathological specimens revealed findings of vasculitis in all cases.

**Conclusion:** There are clinical and imaging features that will help in the pre-operative diagnosis of this uncommon condition. We also postulate vasculitis as a possible underlying pathology for omental infarction.

**Keywords:** acute abdomen, computed tomography, omental infarction, ultrasonography, vasculitis

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## INTRODUCTION

Omental infarction is a rare cause of acute abdominal pain in the paediatric population, being more common in adults<sup>(1-4)</sup>. Children with omental infarcts typically present with a clinical picture similar to that of acute appendicitis<sup>(3-5)</sup>. We retrospectively reviewed six cases with the final surgical and histological diagnosis of omental infarction, and correlated with the clinical presentation and imaging findings on computed tomography (CT) and ultrasonography (US).

## METHODS

Over a four-year period from 2000 to 2004, six surgically- and pathologically-proven cases of omental infarction in paediatric patients from our

institution were retrospectively reviewed. The case records, pre-operative diagnostic imaging results, surgical and pathological findings were examined. All six patients (five boys and one girl; age range: 5 to 11 years) (Table I) presented with abdominal pain for a period ranging from one to seven days, with an average of 4.2 days. Four out of the six patients are noted to be obese, being greater than 90<sup>th</sup> percentile for weight in our local population.

More than one-half of the patients (4 cases) had right-sided abdominal pain that later localised to the right iliac fossa. Three cases had fever as the presenting symptom. None of the cases had gastrointestinal or prodromal symptoms of anorexia, nausea or vomiting that were typically associated with acute appendicitis. Four out of the six cases had normal total white blood cell counts. Four patients underwent CT, one patient was investigated by only US, and another patient had both examinations performed. All cases underwent surgery. Only two cases had pre-operative diagnosis of omental infarction, while the rest of the cases were diagnosed to have acute appendicitis on imaging.

## RESULTS

Of the five cases that underwent CT, two (Figs. 1 & 2) were confidently diagnosed to have omental infarction, based solely on the CT findings. Each showed a ring-shaped inflammatory fatty mass at the right lower quadrant just anterior to the caecum and ascending colon. The mass was bounded anteriorly by the abdominal wall and medially by small bowel loops. Three cases were misdiagnosed as acute appendicitis (Fig. 3), and the last case was diagnosed as having non-specific inflammatory changes in the right iliac fossa. All the cases demonstrated free fluid at the site of inflammatory changes, and in one case, there was fluid tracking along the right paracolic gutter to the right hypochondrium. Of the two cases that were diagnosed correctly as omental infarction on CT, a normal-looking appendix was identified in only one case. No normal-looking vermiform appendix could be identified in the rest of the cases. The only patient

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**Table I. Summary of presenting clinical and imaging features, and operative and pathological findings.**

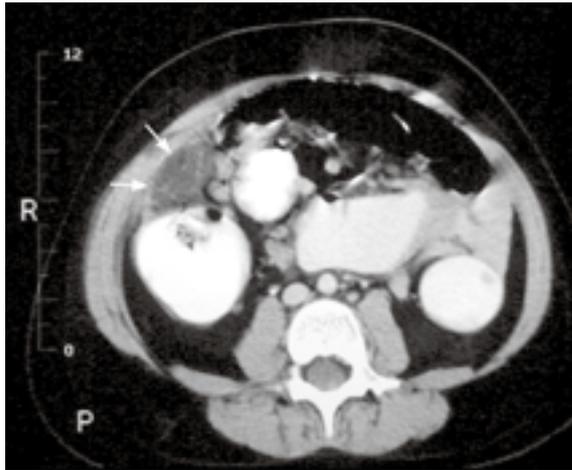
Patient	Age (in years)	Sex	Weight (in kg)	Presenting signs and symptoms	Raised WBC	US features	CT features	Pre-operative diagnosis	Operative findings	Pathological findings
1	5	M	25	2 days progressive RIF colicky pain, 1 episode of vomiting.	No	Not done	Inflammatory stranding of omental fat seen just anterior to the caecum and ascending colon. No free fluid or swollen appendix identified.	Omental infarct	Infarcted omentum adherent to the right lateral abdominal wall. No pus noted in peritoneal cavity. Congested tip of appendix.	Omentum show foci of infarction, fat necrosis, haemorrhage, fibrinous exudate and reparative reaction with early fibrosis. Normal appendix.
2	11	M	40.2	1 week abdominal pain, starting in left flank, progressing to right hypochondrium then RIF. 5 days fever.	No	Not done	4cm by 2cm ring density in thickened omental fat antero-medial to hepatic flexure. Fluid in RIF and pelvis. Normal appendix.	Omental Infarct	Congested omentum lying adjacent to abdominal wall. Free peritoneal fluid. Appendix normal.	Extensive omental fat necrosis with granulation tissue and vascular congestion.
3	10	M	Not taken	1 week abdominal pain, starting from epigastrium radiating to RIF	No	Mild thickening of distal ileal wall. Appendix not seen.	Stranding of omental fat antero-medial to the ascending colon, just below hepatic flexure. Free fluid in RIF and at root of mesentery. No swollen appendix identified.	Acute appendicitis	Haemorrhagic omentum with fat necrosis. Haemorrhagic fluid at right paracolic gutter. Appendix normal.	Omentum focally necrotic with vascular congestion and some fibroblastic proliferation.
4	9	F	57	1 day abdominal pain, periumbilical radiating to RIF. 1 day fever.	Yes	Not done	Pericaecal fat stranding extending to retrocaecal region. Free fluid in RIF. Appendix enlarged and slightly enhancing.	Acute appendicitis	Haemorrhagic omentum wrapped around appendix.	Omentum haemorrhagic with fat necrosis. Inflammation and granulation changes on serosal surface of appendix consistent with periappendicitis.
5	10	M	57	3 days right-sided abdominal pain, 1 day fever.	Yes	Not done	Pericaecal fat stranding extending upwards and to ascending colon. Thickening of caecal wall. Changes confined to right side of abdomen. Free fluid in RIF. No definite swollen appendix identified.	Non-specific inflammatory changes in RIF.	Necrotic friable omental mass adherent to caecum. Free fluid in peritoneal cavity. Appendix normal.	Omentum with patchy acute serositis, fibropurulent exudate and focal granulations.
6	8	M	23.4	3 day progressive RIF pain.	No	Increased echogenicity of fat in RIF, consistent with inflammation. No normal appendix identified.	Not done	Acute appendicitis	Infarcted omentum adherent to anterior abdominal wall. Surrounding inflammatory changes. Appendix normal.	Omental haemorrhagic infarction.

Key: RIF: right iliac fossa; M: male; F: female

who underwent US had features of non-specific inflammation with increased echogenicity of the intraperitoneal fat at the right iliac fossa, and no normal-looking appendix could be identified.

All the patients underwent surgery, with resection of the infarcted omentum as well as appendectomy. All cases had intra-operative diagnosis of omental infarction and had a normal-looking vermiform

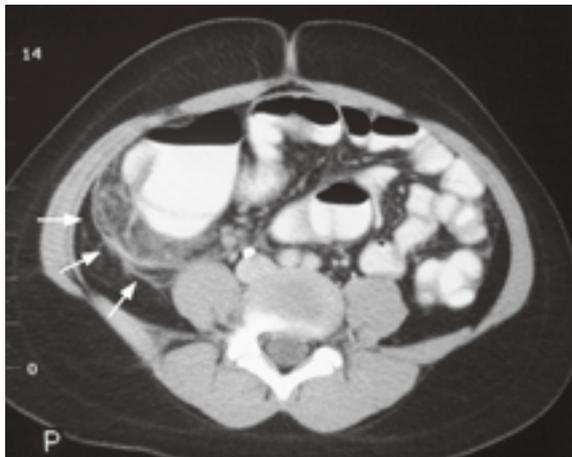
appendix. Surgical findings included congested and haemorrhagic omentum, free intra-peritoneal fluid, and inflammatory changes. In one case, the infarcted omentum was found wrapped around a normal-looking appendix. Pathological findings demonstrated a normal appendix in all cases. The resected omentum showed findings ranging from vascular congestion, haemorrhage, fat necrosis,



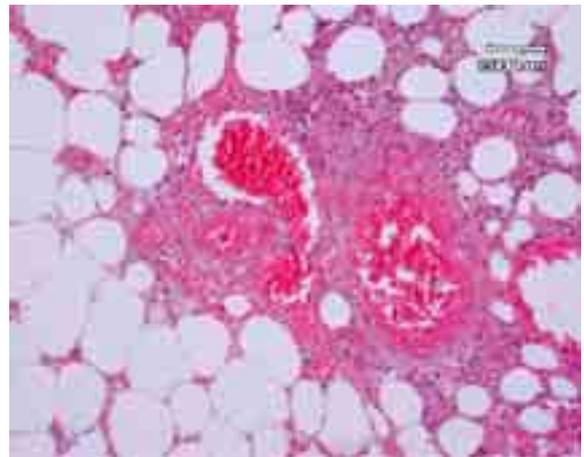
**Fig. 1** Enhanced axial CT image of patient 1 shows a triangular area of inflamed mesenteric fat (arrows) in the right lower quadrant just anterior to the ascending colon. Features are typical of omental infarction.



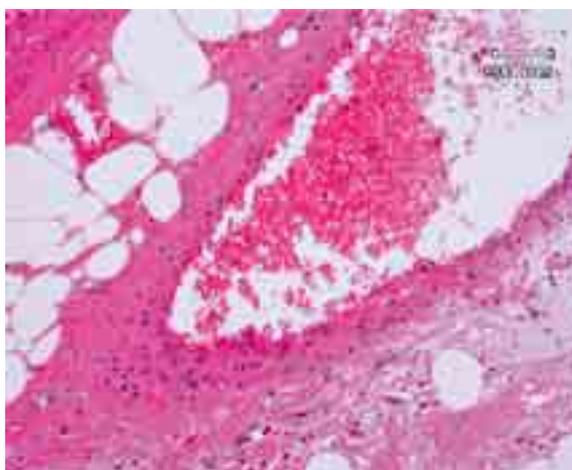
**Fig. 2** Enhanced axial CT image of patient 2 shows an inflamed mesenteric mass (arrows) at the right lower quadrant just anterior to the ascending colon, causing mass effect. Appearance and location is typical for omental infarction.



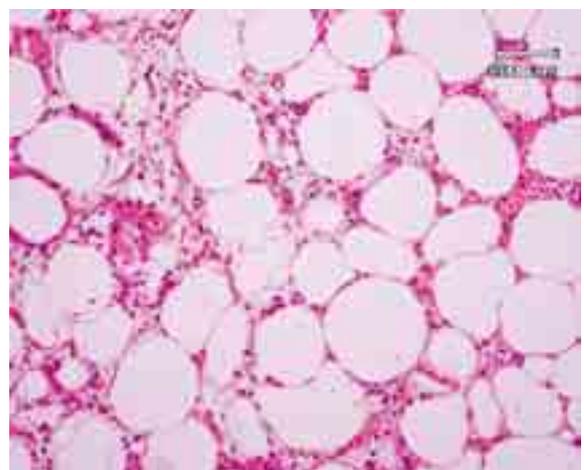
**Fig. 3** Enhanced axial CT image of patient 4 shows the inflamed mesenteric mass (arrows) in an atypical pericaecal location.



**Fig. 4** Photomicrograph shows vasculitis of small arteries with fibrinoid changes of the vascular wall with partial occlusion (Haematoxylin & eosin).



**Fig. 5** Photomicrograph shows vasculitis of medium-sized artery with infiltration of artery wall by neutrophils (Haematoxylin & eosin).



**Fig. 6** Photomicrograph shows small vessel vasculitis giving a picture of panniculitis (Haematoxylin & eosin).

fibropurulent exudate, granulation tissue and fibrosis (Figs. 4-6). After surgery, all patients had dramatic relief of symptoms, with uneventful and rapid recovery.

## DISCUSSION

Omental infarction is rare in the paediatric population, with less than 15% of cases occurring in children<sup>(4)</sup>. There are altogether fewer than 300 cases reported in the literature to date<sup>(4)</sup>. Almost all cases presented with abdominal pain that is commonly right-sided, with the majority localised to the right iliac fossa over time. There are clinical and imaging findings that are highly suggestive of omental infarction.

Omental infarction is reportedly more common in men, with a male to female ratio of 2:1. All the paediatric series have so far reported an increased incidence in male children<sup>(2-4)</sup>. This was also reflected in our series where five out of the six patients were boys. In most of the series, the children were afebrile and had no gastrointestinal symptoms such as nausea and diarrhoea. This results in a delayed clinical presentation. In our series, the average duration of symptoms was four days. In appendicitis, by contrast, the onset of symptoms is usually more acute.

The aetiology of omental infarction is uncertain<sup>(1,4)</sup>. Many authors favour the hypothesis that an embryonic variant of blood supply to the omentum makes it susceptible to kinking, torsion and infarction. Other theories postulate kinking of veins associated with raised intra-abdominal pressure, or vascular congestion after large meals<sup>(3)</sup>. Obesity is a well-known predisposing factor. Most of the paediatric series have reported omental infarction to occur in obese children (defined as greater than 90<sup>th</sup> percentile in weight for their age in the local population)<sup>(1,3)</sup>. There was no mention if the children were obese in two of the series<sup>(4,5)</sup>. Omental infarction is also more common in obese adults<sup>(9)</sup>.

On histopathology, 83% (5 out of 6) of patients had features of vasculitis involving small arteries (Fig. 4) and medium-sized arteries (Fig. 5). The involved vessels showed fibrinoid changes of the muscular wall, with partial occlusion of the lumen by fibrin thrombi. One of the six cases showed findings of interstitial panniculitis (Fig. 6). We believe that this is the first reported series where all cases had histopathological features of vasculitis. In the absence of other surgical or pathological findings of other inflammatory processes such as infection or abscess, we believe that the vasculitis could be the primary cause for the omental infarction.

Lyon et al pointed out that adipose tissues secrete a number of factors that contribute to systemic and vascular inflammation. These factors are collectively known as adipokines. These factors have now been

shown to regulate, directly or indirectly, a number of processes that contribute to the development of atherosclerosis, including hypertension, endothelial dysfunction, insulin resistance, and vascular remodeling<sup>(7)</sup>. Several adipokines are preferentially expressed in the visceral adipose tissue, and the secretion of proinflammatory adipokines is elevated with increasing adiposity. Bounas et al incidentally discovered asymptomatic necrotising intra-abdominal vasculitis after peripheral gastric bypass surgery for morbid obesity, further strengthening an association between vasculitis and inflammation in obese individuals<sup>(8)</sup>.

In our institution, US is the imaging modality of choice in children as it is readily available, portable, and has no radiation ionising hazards. On US, the identification of a hyperechoic non-compressible mass in the right lower quadrant just underneath the anterior abdominal wall and a normal vermiform appendix suggests the possibility of omental infarction. In our series, the two patients who underwent US examination were thought to be due to non-specific inflammation. On retrospective review, this was likely to be due to operator inexperience. Helmrath et al attributed correct diagnosis of omental infarction by US in seven of the last eight cases in their series to be due to increased operator experience<sup>(4)</sup>. Schlesinger et al also attributed two falsely-negative cases to inexperience with the ultrasonographical findings<sup>(5)</sup>. US is also limited by obesity due to poor ultrasonic beam penetration. There may also be overlap of ultrasonographical findings with acute appendicitis and inflammatory bowel disease.

CT is indicated if symptoms persist and the ultrasonographical findings are negative or equivocal. CT findings that are helpful to the diagnosis include the identification of a well-defined area of fat stranding, sometimes with a ring configuration, typically situated anterior to the caecum and ascending colon. The inflamed fatty mass typically lies just below the right lower anterior abdominal wall and may be adherent to it. In our series, four out of five patients who underwent CT showed imaging features typical of omental infarction. The only case that was atypical showed pericaecal inflammatory changes extending to the retrocaecal location. We also found the delineation of a normal-looking appendix especially useful in aiding diagnosis. Secondary signs of inflammation are less specific and includes thickening of the adjacent bowel, which may become incorporated as part of the inflammatory mass, making diagnosis difficult. The presence of free intraperitoneal fluid is likely to represent serosanguinous fluid secondary to the infarction process.

It is important to be able to make a pre-operative diagnosis of omental infarction because firstly, it affects the surgical approach. An incision in the right lower quadrant may not be adequate for exposing the infarcted omentum. Laparoscopy is ideal, and laparoscopic resection of the infarction omentum can be easily done. Secondly, accurate pre-operative diagnosis may also allow for conservative management<sup>(1)</sup>. Some cases of omental infarction that are treated conservatively are self-limiting, with spontaneous resolution of symptoms<sup>(6)</sup>. However, the benefits of removing the infarcted omentum outweigh the risk of conservative treatment. The latter includes prolonged pain, abscess formation and adhesions with possible bowel obstruction<sup>(3)</sup>. Helmraht et al reported one case with secondary infection after conservative management. Post-operative recovery is dramatic and rapid, with most patients being discharged three days after admission<sup>(3,4)</sup>.

In conclusion, omental infarct is a rare cause of acute abdominal pain in children. Its clinical presentation is often confused with that of acute appendicitis. In obese children presenting with right lower quadrant pain, no fever, few prodromal symptoms and a normal total white count, the suspicion of omental infarction should be raised. From our small series of cases, we conclude that a combination of certain clinical signs and imaging findings can help us in the pre-operative diagnosis of omental infarction. CT features are the most helpful and the role of CT in pre-operative assessment cannot be overemphasised. An attempt to identify a normal-looking appendix should be done in all cases.

Increased operator experience in US would also help in the diagnosis of omental infarction. We also postulate vasculitis to be a primary underlying cause for omental infarction as there is evidence to show an increased risk of vasculitis in obese individuals. Moreover, in all the cases reviewed, there are no clinical signs and symptoms or serology to suggest collagen vascular disease in the patients.

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