

Spontaneous Omental Infarction: An Unusual Etiology of Abdominal Pain

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INTRODUCTION

Omental infarction is a surgical rarity with a reported incidence of 0.1% during laparotomies for acute abdomen.¹ It happens twice in male as compared to female, frequent in the 40 to 50-year-old age group with only 15% of reported cases in the paediatric population.^{1,2} Omental infarction may be primary or secondary. Secondary cause happens either due to omental torsion precipitated by postoperative adhesions, tumour, hernia, cyst, localized inflammation, trauma or miscellaneous namely vasculitis, hypercoagulability, and polycythaemia.³ Torsion of the omentum exists as a result of organ twist leading to a compromised vascularity.⁴ As compared to secondary cause, primary or spontaneous omental infarction is an extreme surgical enigma. Imaging modalities namely sonography and computed tomography (CT) are useful to identify the involved pathology. Herein, we report a case of spontaneous omental infarct and discuss its extreme discovery.

ABSTRACT

A 49-year-old lady with previous scars complained of acute abdominal pain for two days. Her right hypochondrium was tender and guarding upon assessment. The laboratory investigations were unremarkable. Due to a diagnostic incongruity, computed tomography of the abdomen was performed showing a suspicious lesion at anterolateral aspect of the ascending colon. Surgical intervention was decided and intraoperative finding was consistent with spontaneous omental infarction. Omentectomy was undertaken and final histology was compatible with the intraoperative diagnosis. Although it is exceptional, omental infarction should be considered as part of the differential diagnoses of right-sided acute abdominal pain with normal laboratory investigations. This case highlights its unexpected discovery and we describe its literature reviews.

KEY WORDS

Acute abdomen, Appendicitis, Cholecystitis, Infarction, Omentum

CASE REPORT

A 49-year-old lady presented to us with a stabbing abdominal pain, localized at center of the abdomen for two days. The pain became worse on the day of admission and it migrated to the right hypochondrium. It was not relieved by analgesia but aggravated by movement. She denied history of trauma. However, she had an appendicectomy 20 years ago and previous caesarean sections in 1997 and 2003.

On examination, there was right hypocondrial tenderness and guarding. Her laboratory investigations were within normal range. Abdominal radiographs and sonography were unremarkable. Due to a diagnostic incongruity, CT of the abdomen was performed, which showed a suspicious oval shaped lesion measuring 4.1 x 5.0 x 6.1 cm (AP x W x CC) located at the anterolateral aspect of the ascending colon (fig. 1a and 1b). It showed a heterogeneous enhancement with peripheral hypodensity of fat whereas the central part

was hyperdense with swirling sign at the left superolateral aspect which could represent a thrombosed vein. Minimal ascites was present as well.

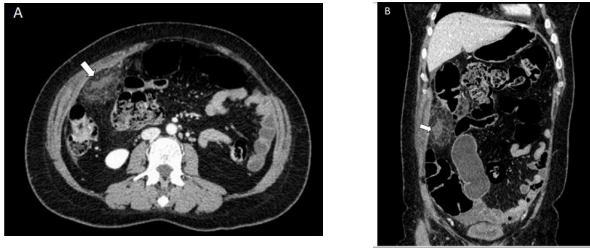


Figure 3. (a) A suspicious oval shaped lesion (white arrow) with fat surrounding streakiness located at the anterolateral aspect of the ascending colon on axial view. (b) Similar lesion (white arrow) seen on coronal view.

In view of persistent pain and equivocal findings on CT, an emergency diagnostic laparoscopy under general anesthesia was performed. Upon entry, intraoperatively we noticed a minimal ascites with hard and fixed omental mass adhering to the hepatic flexure. In view of difficult manipulation and risk of bowel injury, we had decided to convert to open surgery. Upon laparotomy, the omentum was adhered to the hepatic flexure of colon and anterior abdominal wall. With extra care and meticulous dissection, omentectomy was decided as a result. The rest of the visceral organs deemed unremarkable. The bivalve specimen revealed a congested omentum measuring 10 x 8 cm (fig. 2a) with gangrenous cavity (fig. 2b).

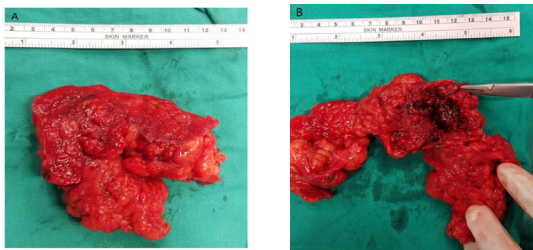


Figure 2. (a) Macroscopic specimen showing a clump of oedematous omentum measuring 10 x 8 cm without evidence of gangrene. (b) Cut section of the omentum revealing infarction and necrosis at the centre of the omentum

She had an uneventful recovery. The microscopic histopathology revealed a marked hemorrhage, vascular dilatation and congestion with active chronic inflammation. No evidence of malignancy was visualized. The final diagnosis was consistent with omental infarction with necrosis.

DISCUSSION

Omentum is a fat laden peritoneal remnant of embryological development. It is anatomically divided into the greater and lesser omentum. Primary omental torsion develops when a mobile segment of omentum rotates around a proximal fixed point in the absence of any associated

intra-abdominal pathology. It is caused by hyperperistalsis, trauma, and anatomical variations of the omentum such as accessory or bifid omentum and narrowed omental pedicle.⁵ Once lack of blood supplies to the omentum, it will become ischemic and eventually infarcted. In view of its low incidence and non-specific presentation, it always leads to misdiagnosis of appendicitis, peptic ulcer disease, cholecystitis, and pancreatitis.

Abdominal sonography is specific but not sensitive for diagnosing omental infarction with abnormalities detected in less than 50% of the cases.⁶ The findings are usually unremarkable; but a complex mass, mixture of solid material, and hypoechoic zones can be visualized. The use of CT in acute abdomen has allowed preoperative diagnosis to be useful. It has been shown to have a high sensitivity and specificity to detect an intraperitoneal focal fat infarction. Localized fat density lesions are seen in omental infarction. Concentric linear strands or the 'whirl' sign and hyper-attenuated streaky infiltration have both been described in omental torsion.^{7,8} In preoperative radiological diagnoses via CT or ultrasound, conservative management is achievable in up to 78% of the cases.⁹

Conservative management by resting, analgesia and anti-inflammatory drugs can be chosen after a confirmatory diagnosis via radiological modalities. The decision is opted in hemodynamically stable patients for the first 24-48 hours while ensuring active thorough observation and antibiotic administration. If the diagnosis is in doubt or failed conservative treatment, surgical intervention should be performed without a delay.¹⁰ Since our patient had persistent pain with intimidating abdominal signs, a decision for surgery was made.

Diagnostic laparoscopy should be considered as it enables a thorough abdominal exploration and omental necrosectomy. This minimally invasive technique can reduce postoperative pain, wound-related complications, and it is associated with low morbidity and rapid recovery hence reduced length of hospital stay. Unfortunately, our patient needed a conversion to open surgery due to the size of the omental mass and the adherence to adjacent hepatic flexure.

In conclusion, omental infarction should be suspected for any patient presented with unexplained acute abdominal pain. If imaging modality has confirmed the diagnosis, we can observe the patients' clinical conditions for 48 hours provided they are stable. However, failed conservative management requires a surgical intervention for therapeutic measures. Since there are no definite guidelines on management of omental infarction, further prospective study needs to be done to compare the outcome between conservative and surgical management.

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