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Case Report

Omental Infarction: What Lies Beneath? A Case Report

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Abstract

Omental infarction is a rare cause of abdominal pain, with just over 300 published cases since its first recognition in 1899. It is classified as primary and secondary; primary occurring due to torsion of the omental vascular pedicle, and secondary occurring due to the hypercoagulable environment created by other pathological processes. Omental infarction is a difficult pathology to diagnose clinically due to the non-specific nature of the symptoms it produces and its ability to mimic numerous other acute intra-abdominal pathologies. As such, omental infarction can initially be incorrectly diagnosed as appendicitis or cholecystitis, with up to 90% of cases of omental infarction diagnosed during laparoscopy for presumed appendicitis. This case report follows a patient who was initially diagnosed with primary omental infarction who deteriorated due to acute perforated cholecystitis.

This case follows a 76 year old male patient who presented to the emergency department with upper abdominal pain and signs of peritonism. He had had a similar presentation one year earlier, which computed tomography (CT) scan diagnosed as omental infarction. On examination, he was systematically well with right upper quadrant and upper abdominal tenderness and peritonism. Bloods showed raised inflammatory markers with normal liver function tests. CT abdomen/pelvis showed a further omental infarct, while the adjacent bowel and gallbladder were reported as normal. He became systematically unwell over the next 48 hours with signs of multi organ failure. Repeat CT showed acute gallbladder rupture/ischaemia with blood tests showing a multi organ picture. He was taken for a laparotomy with abdominal washout, total cholecystectomy and partial omentectomy. He was transferred to ITU but unfortunately failed to recover. This case emphasizes the importance of treating cases of omental infarction as secondary until proven otherwise, because it is the primary co-existing pathology which can result in significant morbidity and mortality. If untreated or overlooked, the patient is likely to deteriorate and have an adverse outcome, in spite of the fact that the primary pathology is usually curable.

Keywords: *Omental Infarction; Lies Beneath*

Introduction

Omental infarction is a rare cause of abdominal pain, with just over 300 published cases since its first recognition in 1899 [1]. It is classified as primary and secondary; primary occurring due to torsion of the omental vascular pedicle, and secondary occurring due to the hypercoagulable environment created by other pathological processes [1]. Both processes cause reduction in omental blood supply, leading to necrosis, extravasation of fluid and adhesions [2]. Risk factors for the development of primary omental infarction have been identified, of which the most important are obesity, trauma and diet [3].

Omental infarction is a difficult pathology to diagnose clinically. This is largely due to the non-specific nature of the symptoms it produces and its ability to mimic numerous other acute intra-abdominal pathologies [4,5]. In most cases abdominal pain features on

the right-hand side, but cases of left sided and migratory pains have also been reported [4,6]. As such, omental infarction can initially be incorrectly diagnosed as appendicitis or cholecystitis [5,7], with up to 90% of cases of omental infarction diagnosed during laparoscopy for presumed appendicitis [7].

The diagnosis of omental infarction is usually adopted after exclusion of other more common surgical emergencies, namely appendicitis, cholecystitis, and pancreatitis. In such cases, Computed Tomography (CT) scanning has proven to be the best diagnostic tool in deciphering the pathological process at play and is currently the gold standard for diagnosis of omental infarction [8].

Here, we present a case of an acute abdomen that was provisionally diagnosed with omental infarction after a CT scan. The patient deteriorated after two days of conservative treatment, became septic and was taken to theatre for an emergency laparotomy which surprisingly showed an acute gangrenous perforated cholecystitis with resultant generalised biliary peritonitis.

This case emphasizes the importance of treating cases of omental infarction as secondary until proven otherwise, because it is the primary co-existing pathology which can result in significant morbidity and mortality. If untreated or overlooked, the patient is likely to deteriorate and have an adverse outcome, in spite of the fact that the primary pathology is usually curable.

Case Report

A 76 year old male patient presented to the emergency department with upper abdominal pain and signs of peritonism. His past medical history included type 1 diabetes mellitus, class 3 obesity, ischaemic heart disease and hyperlipidemia.

He had had a similar presentation one year earlier, which was investigated with a CT scan of the abdomen/pelvis with contrast, which showed a possible omental infarction. He was managed conservatively with antibiotics. He responded well to treatment and was subsequently discharged.

On this admission, he presented with a primary complaint of upper abdominal pain, localized to the right upper quadrant (RUQ). On examination, he was systematically well with RUQ and upper abdominal tenderness and peritonism. He was initially investigated with blood tests which showed: White Cell Count (WCC) 9.1 ($x10^3/mm^3$), C Reactive Protein (CRP) 122 (mg/L), bilirubin 28 (µmol/L), urea 9 (mmol/L) and creatinine 95 (µmol/L). Considering his right sided peritonism he was further investigated with a CT abdomen/pelvis with contrast which showed a further omental infarct, while the adjacent bowel and gallbladder were reported as normal (Figure 1 to 3). In light of his cardiac history, an electrocardiogram (ECG) and troponin levels were also requested, both of which were normal. He was admitted for conservative management of the suspected new omental infarction.

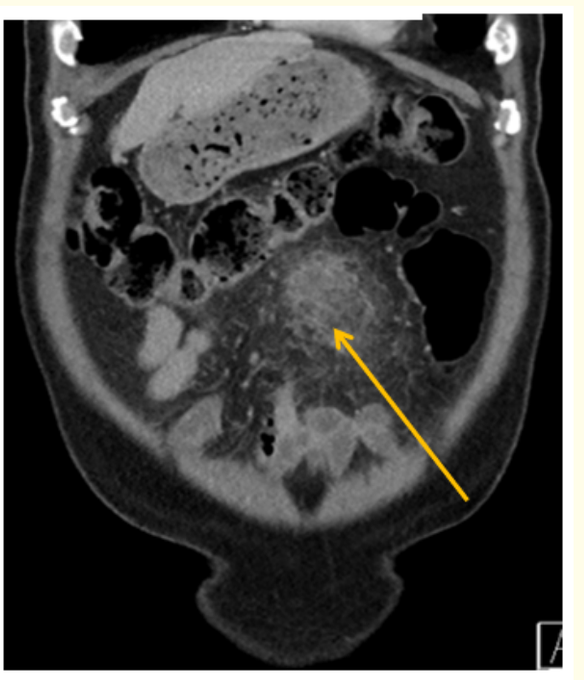

Figure 1: *Focus of omental infarction.*

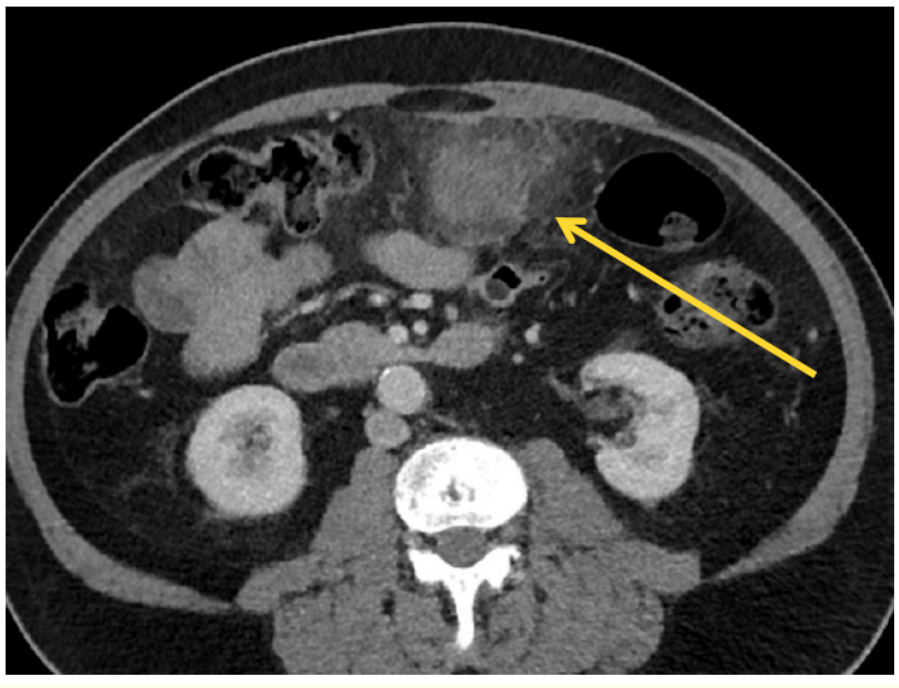

Figure 2: *Focus of omental infarction.*

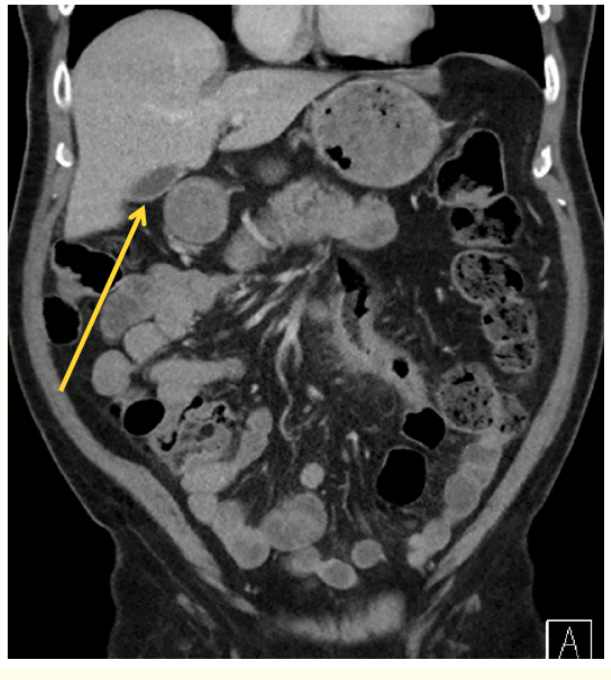

Figure 3: *Normal gallbladder.*

Due to his persistent RUQ pain, an ultrasound examination of his biliary tree was carried out the following day, revealing a thin-walled gallbladder with no evidence of cholelithiasis and acute cholecystitis.

During the next 48 hours, the patient became systematically unwell and developed features of septic shock (hypotension, tachycardia and pyrexia). Repeat blood testing showed a WCC rise to 13 ($x10^3/mm^3$) and a CRP rise to 500 (mg/L). His previously normal liver function tests had become deranged with his bilirubin now 52 (µmol/L), alanine aminotransferase (ALT) 52 (IU/L), aspartate aminotransferase (AST) 55 (IU/L), alkaline phosphatase (ALP) 34 (IU/L), and albumin 30 (g/L). His kidney function had also deteriorated; renal function tests showed urea was 27.7 (mmol/L), creatinine 466 (µmol/L) and eGFR 10, reflecting multi organ failure.

As a result, he was transferred to the high dependancy unit for organ support and had a repeat CT abdomen/pelvis with contrast. This revealed bibasal lung consolidation, pulmonary oedema and features suspicious for acute gallbladder rupture/ischaemia with RUQ inflammatory changes of a bulging and non-enhancing gallbladder fundus and secondary small bowel obstruction (Figure 4 and 5).

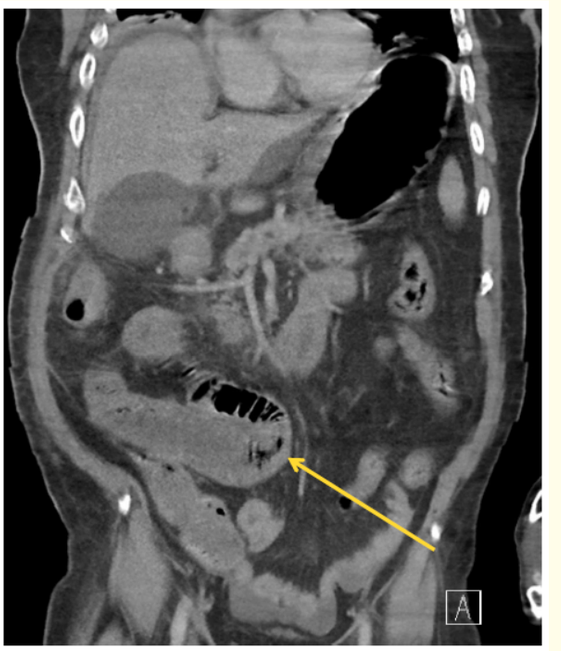

Figure 4: *Dilated small bowel loops indicating small bowl obstruction.*

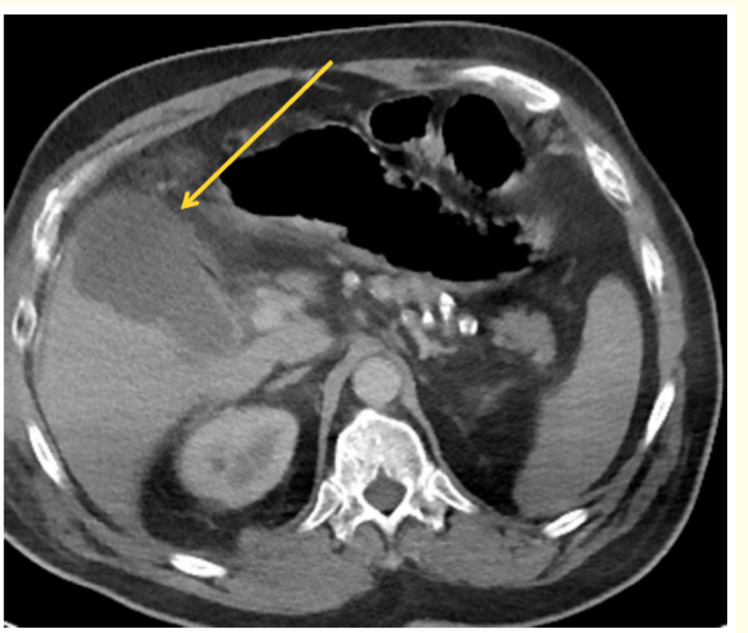

Figure 5: *Acute gallbladder rupture/ischaemia.*

In light of his clinical deterioration and CT scan findings, the patient was taken to theatre for an emergency exploratory laparotomy. Preoperatively, the P-possum mortality was reported as 56%, and the patient had an episode of peri arrest but was revived successfully.

The intraoperative findings showed the following

- Generalised four quadrant biliary peritonitis
- Acute gangrenous cholecystitis with a gallbladder perforation (confirmed on post operative histology)
- Omental inflammation and an area of infarction of the greater omentum
- Small bowel obstruction secondary to fibrinous inflammatory adhesions.

Thorough lavage of the abdominal cavity was carried out followed by a total cholecystectomy and omentectomy. Penrose drains were placed in the sub hepatic space and the pelvis. Postoperatively, he was transferred to intensive care unit for post op care and organ support, subsequently requiring inotropic support and haemofiltration.

During postoperative surgical review, he had a soft abdomen with haemoserous drain content and no bile. Unfortunately, his multiorgan failure continued to worsen and he passed away the following day.

The post operative histology confirmed the intra operative findings of acute gangrenous cholecystitis with gallbladder perforation

Conclusion

Omental infarction is a difficult diagnosis to reach due to variations and inconsistencies in its presenting clinical features. As this case reflects, it is imperative to treat omental infarction cases as secondary until proven otherwise and rule out all other pathologies which may be the root cause. This is vital as it is often the underlying secondary cause which will determine the patient outcome and as such, omental infarction should only be accepted as primary once all other, more aggressive secondary pathological processes have been excluded.

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