A Case of Primary Omental Infarction in an Adult Female Presenting as Right Upper Quadrant Pain

Mansukhani SMH
Bañez VP

Section of Gastroenterology and Digestive Endoscopy
Manila Doctors Hospital
Manila, Philippines

Correspondence:
Dr. Sujata May Mansukhani
suji_mansukhani@yahoo.com

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Abstract

Background: Primary omental infarction is a rare cause of abdominal pain that may mimic other disease entities hindering timely management. Only over 400 cases have been documented and as of this writing, there are no published local data. *Clinical Presentation:* A 55-year-old hypertensive Filipino female presented with a five-day history of epigastric pain radiating to the right upper quadrant. Examination revealed direct right upper quadrant tenderness with Murphy's sign. Work-up revealed leukocytosis and elevated total and indirect bilirubin. Initial impression was acute cholecystitis. Abdominal ultrasound showed normal gallbladder and biliary tree. Inconclusive findings prompted a contrast-enhanced MRI of the upper abdomen with MRCP which showed a well-defined lobulated mass-like lesion with thin rim enhancement in the right upper anterior peritoneal cavity with ascites, inflammatory changes and edema of the overlying subcutaneous fatty layer, indicative of omental infarction or panniculitis. *Management and Outcome:* Exploratory laparotomy with omentectomy was done revealing a concavity between the subcostal area and segments IV/V of the liver where the omentum was trapped. The omentum was hyperemic, thickened with central fat necrosis, and adherent to the anterior abdominal wall, greater omentum and proximal transverse colon. Histopathology revealed fibro-collagenous tissue and adipose tissue with chronic inflammation, hemorrhage and congestion. She was discharged stable after three days. *Recommendations:* Awareness of this disease, its mimics and diagnostic strategies are keys to early diagnosis, treatment, and prevention of complications.

Keywords: case report, omental infarction, primary omental infarction, omental torsion

Introduction

Omental infarction is a rare cause of abdominal pain in adults with an incidence of less than four cases per 1000 cases of appendicitis.[1] Only over 400 cases have been documented and, as of this writing, there are no published local data. We report a case of primary omental infarction in a 55-year old Filipino female mimicking acute cholecystitis.

Case Presentation

A 55-year old Filipino female presented with five-day history of epigastric pain radiating to the right upper quadrant. She self-medicated with antispasmodics and antacids with no relief of pain. On physical examination, she had stable vital signs, afebrile, with a body mass index (BMI) of 33 kg/m^2. Abdominal examination revealed direct right upper quadrant tenderness with Murphy's sign. Complete blood count showed a slightly elevated WBCs (11.88 X 10^9/L) with predominance of neutrophils (0.69). Blood chemistries showed direct hyperbilirubinemia (20.68 umol/L vs. total bilirubin 23 umol/L). Amylase, lipase, prothrombin time, creatinine,

sodium, potassium, SGPT and SGOT were normal. Initial impression was acute cholecystitis. She was placed on nothing-per-orem diet and initiated on intravenous hydration. Ciprofloxacin 400 mg IV every 12 hours, tramadol 50 mg IV, and hyoscine N-butylbromide 10 mg IV every eight hours were given. There was no relief of pain despite pain medications. Ultrasound of the whole abdomen revealed fatty liver, normal gallbladder, biliary tree, pancreas, spleen, kidneys, uterus and urinary bladder. Work-up could not explain the persistent pain. Since the primary consideration was a biliary disease, contrast-enhanced upper MRI of the upper abdomen with MRCP was requested. It showed omental infarction or panniculitis with minimal ascites, inflammatory changes and mild gallbladder sludge (**Figure 1**).

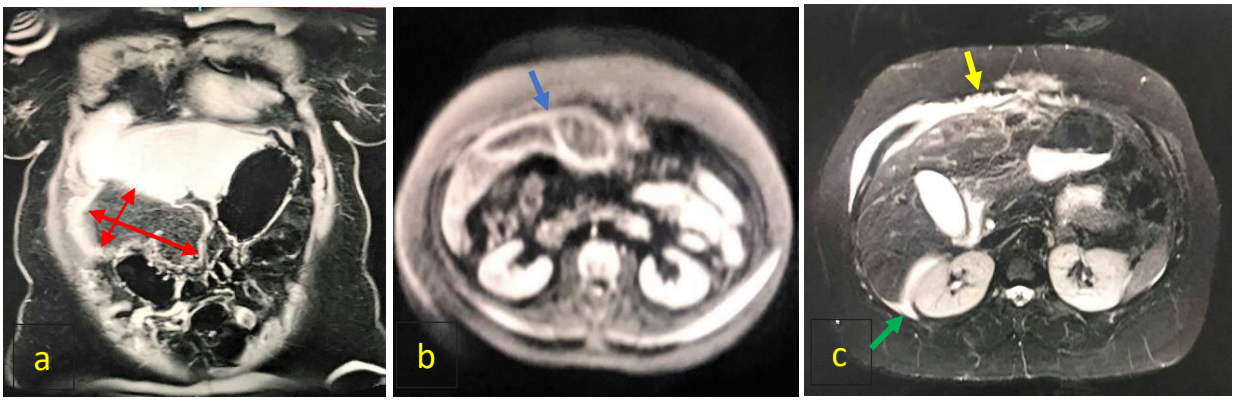

Figure 1. MRI with MRCP of the upper abdomen with contrast. T1 image: Well-defined lobulated mass-like lesion with fatty and edematous signals within the right upper anterior peritoneal cavity measuring 3.4 cm x 11.2 cm x 8.8 cm (red arrows). (a) *DWI image.* The well-defined lobulated mass lesion exhibits a thin enhancing rim (blue arrow). It is located anterior to the left hepatic lobe and superior to the transverse colon. (c) *T2 TIRM image*. Minimal perihepatic and perisplenic fluid (green arrow), inflammatory changes involving the right anterolateral abdominal wall with moderate edema of the overlying subcutaneous layer (yellow arrow).

The patient underwent diagnostic laparoscopy showing a concavity between the subcostal area and liver where the omentum was trapped, resulting to a hyperemic and thickened omentum with areas of necrosis. This prompted conversion to exploratory laparotomy with omentectomy (**Figure 2**).

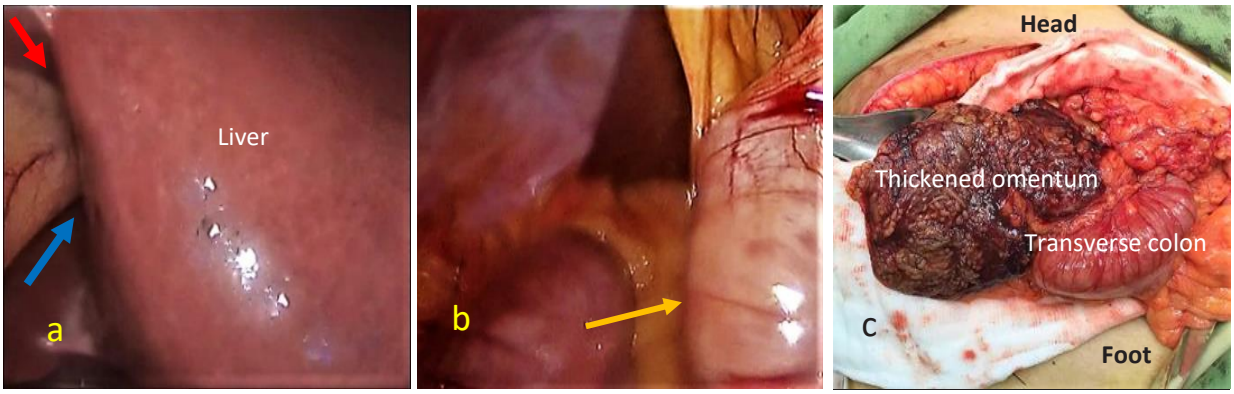

Figure 2. Intraoperative findings. (a) 3 cm x 4 cm concavity between the subcostal area and segments IV/V of the liver (blue arrow) with trapped omentum within (red arrow); (b) Omentum adherent to the proximal transverse colon (yellow arrow), anterior abdominal wall and greater omentum causing it to be displaced and pulled towards the infarcted area; (c) hyperemic and thickened omentum with areas of necrosis.

The patient tolerated the procedure well and was discharged stable after three days. Histopathologic findings revealed fibrocollagenous tissue and adipose tissue with chronic inflammation, hemorrhage and congestion consistent with omental infarction (**Figure 3**).

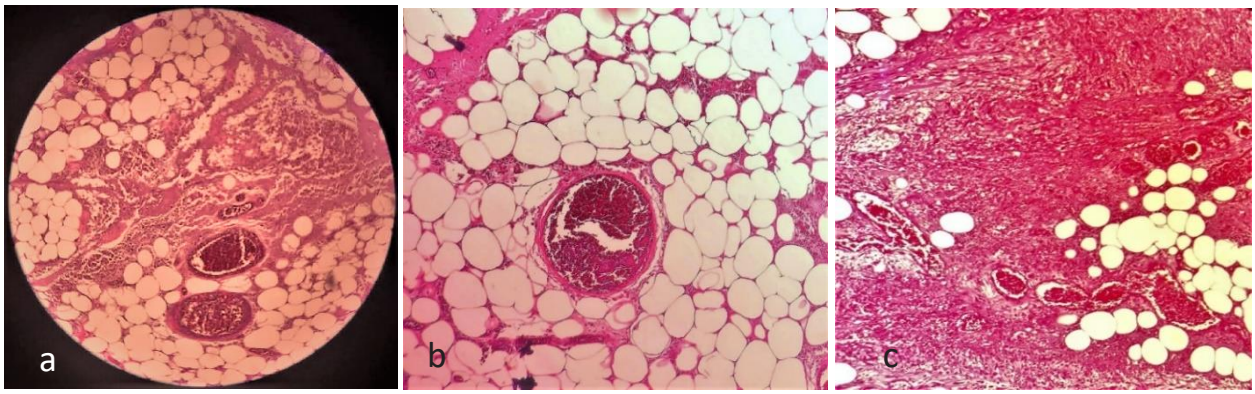

Figure 3. Histopathology, H & E stain. (a) & (b) Low power magnification showing numerous mononuclear inflammatory infiltrates admixed with red blood cells; (c) high power magnification showing vessels and blood-filled surrounding stroma indicating congestion and hemorrhagic change.

Discussion

Omental infarction results from venous stasis leading to edema and congestion of the omental vessels causing hemorrhagic necrosis and extravasation into the interstitium with thrombosis in the omental veins, thus, resulting to peritoneal irritation and pain.[3]

Omental infarction may occur with or without torsion of the greater omentum. Infarctions with torsion are classified either as: (a) idiopathic, or (b) secondary; which is associated with intra-abdominal adhesions, hernia and tumor. Omental infarctions without torsion are usually caused by hypercoagulability and vascular abnormalities.[3] Primary omental infarction (also known as idiopathic segmental infarction of the greater omentum or ISIGO) occurs when a mobile segment of the omentum rotates around a proximal fixed point in the absence of any intra-abdominal pathology. Torsion may be triggered by compression of the greater omentum between the liver and abdominal wall after local trauma, excessive exercise, occupational vibration, increased intra-abdominal pressure, sudden body movement, laxative use and acute changes in body position.[1,2,4] Anatomic malformations, such as bifid or accessory omentum and redundant omental veins, may also predispose to primary omental infarction.[2]

Obesity is a well-documented risk factor for primary omental infarction. In one study, almost 70% of patients with omental infarction were obese.[5] It is hypothesized that fatty accumulation in the omentum impedes the distal right epiploic artery and the additional structural mass potentially precipitates torsion.[1] Our patient had a BMI of 33 kg/m^2, classifying her as Obese Class I according to the WHO criteria, predisposing the development of primary omental infarction.

The major symptom of omental infarction is sudden onset pain over the right flank and lower abdomen which does not radiate to the abdominal wall.[4,5] Physical examination may demonstrate signs of local peritonitis and laboratory work-ups may show non-specific inflammatory response.[2] Computed tomography is the imaging modality of choice with high sensitivity (90%) and specificity.[6,7] The most common diagnostic finding is an ill-defined heterogeneous fat density with surrounding inflammatory changes.[1,6] Ultrasound is utilized to rule out other more common conditions such as acute cholecystitis.[7] Findings are usually normal. However, in <50% of cases, omental infarction is suggested by a hyperechoic, incompressible, ovoid mass.[8] In this case, since the initial impression was a biliary disease, MRI with MRCP of the abdomen was performed instead of a CT scan. Abdominal MRI is rarely used. On review of literature,

only a single report employed the use of this imaging modality. The MRI findings in this study showed a focal region of heterogeneous fat with surrounding inflammation and fluid,[9] similar to our patient.

Omental infarction is managed conservatively or via laparoscopic excision. Conservative treatment with bed rest, analgesics, anti-inflammatory medications, and fluid resuscitation are for hemodynamically stable patients.[1,2,7] Antibiotic prophylaxis is often used in the setting of conservative management when there is a risk of secondary infection of the infarcted fat.[3] Early surgical intervention via omental necrosectomy, on the other hand, reduces the duration of abdominal pain and incidence of complications, namely: necrosis, abscess formation and adhesions.[1]

Summary

Omental infarction is a rare cause of abdominal pain and is often diagnosed intraoperatively. Reports of this uncommon disease entity could raise awareness for clinicians to investigate other causes of acute abdominal pain, to employ appropriate imaging techniques in cases of uncertain diagnosis, and institute appropriate management to avoid complications.

Conflict of Interest

The authors declare no conflicts of interest.

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