CLINICAL VIGNETTE

Omental Infarction: A Rare Cause of Abdominal Pain

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Case 1

An 82-year-old female with a history of breast cancer, hypothyroidism, and prior hysterectomy presented to her gastroenterologist with three days of worsening sharp mid-epigastric pain. She also reported nausea and anorexia but denied diarrhea, constipation, fever, and vomiting. Laboratory tests showed a white blood cell count (WBC) of 7.23 x10E3/uL with normal differential and an elevated C-Reactive Protein (CRP) of 71 mg/L. A Computed Tomography (CT) of the abdomen showed "focal induration in the superior portion of the omentum with halo of edema surrounding a fat density central component. The appearance suggests fat necrosis and an omental infarction." She was given a diagnosis of omental infarction and treated conservatively with outpatient observation and pain management. Her pain subsided within a week.

Case 2

An 86-year-old female with a history of MALT lymphoma, coronary artery disease (CAD), congestive heart failure (CHF), presented to clinic with 4 days of right lower quadrant pain described as 9/10 in severity. She denied nausea, vomiting, fever, constipation, diarrhea, and urinary symptoms and was able to tolerate oral intake. She also had two prior abdominal surgeries and peripheral vascular disease (PVD0). A recent CT scan two days earlier showed "mild soft tissue stranding in the right upper quadrant adjacent to the tip of the right lobe of the liver and ascending colon without evidence of bowel wall thickening in the adjacent bowel. Laboratory tests included WBC of 7.97 x10E3/uL with normal differential and mild elevation in CRP of 15 mg/L. She had unchanged chemistries including lipase. Given the persistent pain another CT scan was obtained showing "mild soft tissue stranding anterior to the right hepatic lobe that may represent an omental infarct." A surgical consult was obtained, and she was treated conservatively as an outpatient and the pain eventually resolved in 4 weeks.

Discussion

The greater omentum is a double layer of fibrous fatty tissue of the peritoneum that extends from the greater curvature of the stomach to the transverse colon, inferiorly covering the abdominal organs. It is a complex immunologic organ which hosts inflammatory and stem cells and is an important defense against intraabdominal infections.[1]

Omental infarction is a rare cause of abdominal pain with an incidence of less than 4 cases per 1000 cases of suspected appendicitis.[2] The clinical presentation can vary but usually involves nonspecific abdominal pain over a few days with associated nausea and anorexia. A series of patients with omental infarction at four Australian hospitals confirms the rarity, with only 61 cases in 10 years.[3] The abdominal pain more commonly presents on the right side compared to the left, however, the location correlates to the site of infarction so may present anywhere in the abdomen. The nonspecific presenting of symptoms makes it challenging to diagnosis. Omental infarction can mimic other common acute abdominal pathologies such as appendicitis, cholecystitis, epiploic appendagitis or diverticulitis.[4] The low prevalence of this disease means it will often be forgotten on a differential diagnosis of acute abdominal pain. It has not been reported in children and adults of varying ages with children and middle-aged adults with the highest prevalence.[4]

Omental infarction is divided into primary and secondary etiologies and can be caused by omental torsion or changes in venous blood supply leading to reduced flow or venous thrombus.[3] Primary omental torsions are idiopathic.[5] Secondary omental torsions are more common torsions and can be caused by tumors, cysts, hernias, surgical scars, or adhesions.[6] The omentum is longer and more mobile on the right side which may explain the higher incidence of right-sided pain and infarction. Omental infarction without torsion can be caused by trauma, thrombosis, vascular anomalies, venous congestion, or sequala of abdominal surgery.[7] Proposed risk factors include obesity, excessive exercise, sudden changes in position, laxative use, or heavy food intake.[7]

Since omental infarction can mimic many acute abdominal conditions, an accurate diagnosis is important to avoid unnecessary surgery. Omental infarction is diagnosed by contrast enhanced CT scan of the abdomen or diagnostic laparoscopy. Classic findings on CT include a heterogenous fat-density lesion within the omentum without a peripheral rim with streaks and whirl like appearance of the soft tissue.[8] Disproportionate fat stranding in relation to bowel wall thickening is also seen.[9] Omental infarcts can be distinguished from acute epiploic appendagitis by the larger size of the fat-density lesion and lack of a hyperdense rim.[10] Additionally, epiploic appendagitis usually abuts the colonic wall. Greater than 2-week follow-up imaging of omental infarctions show a well-defined, smaller

fat-density lesion with a hyperdense rim that can persist for months to years.[11]

Omental infarction can be treated conservatively or with surgery, and is usually self-limited. Conservative treatment consists of intravenous fluids and pain management with close monitoring for sepsis and peritonitis. Surgical treatment usually involves laparoscopic omentectomy if conservative therapy fails.[3] Conservative therapy was successful in 68% of patients in the prior mentioned Australian case series.[3] The decision to treat these two patients conservatively was based on their advanced age and comorbid conditions. Both patients had several potential risks factors for omental infarction. One was obese, a known risk factor which allows for greater propensity for points of torsion or may interrupt the blood supply.[12] Additionally, both CHF and PVD which may lead to congestion of the mesenteric veins and hemorrhagic ischemia. Both patients also had a history of abdominal surgery so another proposed etiology would be omental torsion secondary to adhesions.

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