

CASE OF DU PERFORATION WITH SECONDARY OMENTAL NECROSIS- A RARE CASE REPORT

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Abstract

Introduction: The Omentum is rich in blood supply. Omental Infarction can be classified as primary or secondary depending on the pathogenesis.

Aims and Objectives: To report a case of DU perforation with secondary Omental Infarction.

Case Details: A 21 year old male patient came with complaints of generalized dull aching abdominal pain, associated with persistent vomiting and high grade fever since 3 days. On examination, he was drowsy, BP was not recordable and peripheral pulses were not palpable. Abdominal examination revealed guarding and rigidity. X-ray erect abdomen showed gas under the right dome of the diaphragm (pneumoperitoneum).

The patient was taken up for an exploratory laparotomy. Intraoperatively, findings included: 1) A 0.5*0.5cm in size perforation over the anterior first part of the duodenum, 2) approximately 3L of haemorrhagic peritoneal fluid 3) necrosed omentum and 4) petechial patches over the parietal wall of peritoneum. Primary repair of the DU perforation with omental plug (modified graham's repair) with omentectomy of the necrosed part of omentum was done.

The HPE report of excised specimen of omentum was suggestive of intense congestion and necro-inflammatory reaction of the omentum with necrosis and neutrophilic infiltrate.

Conclusion: A rare case of DU perforation with secondary omental necrosis is being reported.

Keywords: DU Perforation; Omental Necrosis; Omental Infarction; Modified Graham's patch

Abbreviations: OI- Omental Infarction, DU- Duodenum.

Introduction

The omentum and intestinal mesentery are rich in lymphatics and blood vessels. The omentum contains areas with high concentrations of macrophages which aid in the removal of foreign material and bacteria. Furthermore, the omentum becomes densely adherent to intraperitoneal sites of inflammation, often preventing diffuse peritonitis during cases of intestinal gangrene or perforation, such as acute diverticulitis or acute appendicitis [1].

Case Report:

A 21 year old male patient came with complaints of generalized dull aching abdominal pain, more severe in the right iliac fossa associated with persistent non-bilious vomiting and high grade fever since 3 days. He had a history of asthma in childhood and no major surgical interventions in the past. On examination, he was drowsy, responding to verbal commands with a GCS of 14/15. BP was not recordable and peripheral pulses were not palpable. Patient was immediately

started on iv fluids and ionotropic support. Abdominal examination revealed guarding and rigidity, with generalized tenderness on superficial palpation and absent bowel sounds on auscultation. ABG was suggestive of metabolic acidosis and USG showed free fluid in the abdomen. X-ray erect abdomen showed gas under the right dome of the diaphragm (pneumoperitoneum).

The patient relatives were counseled about the condition of the patient and high risk consent was taken. The patient was taken up for an exploratory laparotomy. Intraoperatively, a midline incision was taken. Findings included: 1) A 0.5*0.5cm in size perforation over the anterior first part of the duodenum [Image1], 2) approximately 3L of haemorrhagic peritoneal fluid with pus flakes along entire erythematous small bowel, 3) necrosed omentum [Image 2] and 4) petechial patches over the parietal wall of peritoneum.

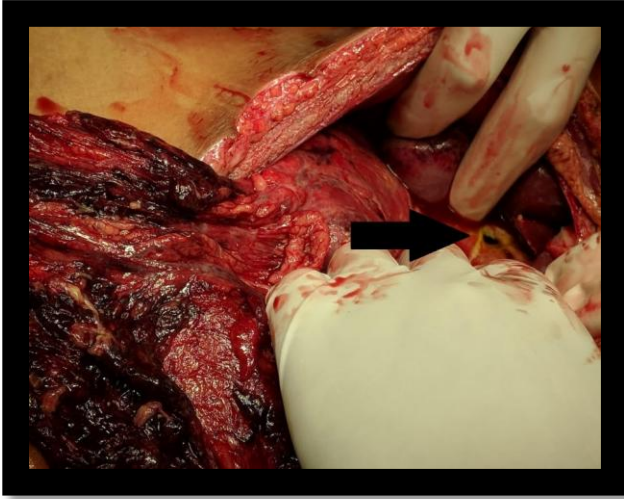


Figure 1: DU Perforation



Figure 2: Necrosed Omentum

Primary repair of the DU perforation with omental plug (modified Graham's repair) with omentectomy of the necrosed part of omentum was done. The peritoneal cavity was thoroughly washed with NS and an intra-abdominal drain was placed. Post-operatively, the patient was continued on ventilatory and inotropic support. The patient showed a clinical picture of ARDS with Septic shock. The CCU team continued management of AKI with pulmonary edema with hypotension. On POD5, the patient began to show improvement. RT feeds were started by POD6 and the patient was taken off ventilatory support on POD10. Liquid diet was initiated on POD7. Ryle's tube and abdominal drain were removed on POD11 and soft diet was started. POD16, the patient was discharged after suture removal. He was tolerating oral feeds well, had passed stools and had no abdominal or pulmonary complaints.

The HPE report of excised specimen of omentum was suggestive of intense congestion and necro-

inflammatory reaction of the omentum with necrosis and neutrophilic infiltrate.

Discussion:

The greater omentum develops in the eighth week of gestation from the dorsal mesogastrium[2]. It is composed of two mesothelial sheets which enclose predominantly adipocytes embedded in a loose connective tissue, and also aggregates of mononuclear phagocytic cells. The omentum has a rich vascular supply with numerous characteristic capillary convolutions[3].

Omental Infarction can be classified as Primary or secondary depending on the pathogenesis. A diagnosis of primary or 'idiopathic' OI is made when no discernable aetiology is found. Secondary causes for OI include hypercoagulability, vasculitides, polycythaemia, and for omental torsion, cysts, tumours, and adhesions [4].

Various methods of management of omental infarction have been tried. In a few studies, where OI was diagnosed by CT Imaging, conservative management (use of analgesic and anti-inflammatory medication with optimal fluid management) has been tried successfully [5]. General consensus holds OI and omental torsion as principally self limiting conditions, and this is supported by CT imaging data at 1–3 years follow up of patients [6]. This is supported by data published on OI [7,8,9] and torsion associated OI [10,11] elsewhere.

In studies recommending surgical intervention, the argument put forth is that surgery decreases post-operative recovery time and hospital stay [12,13].

In our case, on clinical examination and X-ray Erect Abdomen, we diagnosed the patient to have a hollow viscous perforation with peritonitis. As the patient presented in a state of hypovolemic shock (tachycardia, BP not recordable), the patient was taken up for an emergency exploratory Laparotomy. Intraoperatively, we identified features s/o DU perforation with omental necrosis. The operating Gastrosurgeon decided that the ideal operative treatment would be a primary repair of the DU perforation with an Omental plug (modified Graham's operation) followed by an omentectomy.

Post-operatively, once the patient recovered, he had no abdominal complaints, indicating that the line of management adopted was appropriate.

Conclusion:

The Omentum is a highly vascular organ but recently many cases of omental infarction followed by necrosis have been reported. The line of management differs depending on whether the etiology is Primary or Secondary. In the case reported by us, Surgical Intervention proved successful as it was the Rare scenario of a DU perforation with omental infarct diagnosed intraoperatively. A rare case of DU perforation with secondary Omental Necrosis has been reported.

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