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Omental Necrosis Complicating Prepyloric Perforation Repair: A Case Report

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Abstract
Introduction: *Peptic ulcer disease (PUD) prevalence is declining globally to about 5-10%, particularly in developed countries, yet complications like bleeding, perforation, and obstruction still result in around 150,000 hospitalisations annually. Major complications include upper GI bleeding and perforation, with the latter having a high mortality rate (about 30%) and necessitating urgent surgical and sepsis management.*
Case Report: *A 45-year-old male in severe shock with acute abdominal pain and signs of peritonitis underwent emergency investigations revealing pneumoperitoneum. Post-resuscitation, an exploratory laparotomy identified a prepyloric perforation, repaired using the Cellan Jones technique. By postoperative day six, the patient exhibited seropurulent drainage (40-50 cc daily), and by day eight, a mild bilious tinge prompted re-exploration, revealing extensive omental necrosis and bile leaks at the repair site. Omentectomy and perforation reinforcement, with feeding jejunostomy, were performed.*
Conclusion: *Sepsis induced microvascular dysfunction and hemodynamic effects of inotropic agents likely compromised omental blood flow, causing ischemia and necrosis. This case underscores the need for early detection and intervention in managing prepyloric perforation repair complications.*

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I. Background

Peptic ulcer disease (PUD) prevalence is decreasing, now estimated at 5-10%.[1] Despite this, complications like bleeding and perforation cause nearly 150,000 hospitalisations annually.[2] Upper GI bleeding incidence is 19-57 per 100,000, and ulcer perforation 4-14 per 100,000. Risk factors include advanced age, Helicobacter pylori infection, and NSAID use.[3] Perforated ulcers have a high mortality rate of nearly 30%, requiring prompt surgical intervention and proactive sepsis management.[4]

Complications can arise postoperatively, leading to treatment challenges. Herein, we describe a case of omental necrosis complicating pre pyloric perforation repair, necessitating re-exploration and additional surgical interventions.

II. Case Presentation

A 45-year-old male presented to the emergency department in shock with a sudden onset of severe diffuse abdominal pain for two days, accompanied by multiple episodes of vomiting, abdominal distension, and non-passage of stool/flatus. On examination, the patient was drowsy with a temperature of 103.1°F, a feeble pulse of 152 bpm, and a BP of 46/20 mm Hg, exhibiting poor respiratory effort. Abdominal guarding and rigidity were present. After rapid assessment, resuscitation began. The patient was intubated and placed on mechanical ventilation (A/C PRVC at 16/min). As bolus fluid resuscitation showed no response, inotropes started and gradually increased (Noradrenaline @10 ml/hr, Vasopressin @2.4 ml/hr). The patient was catheterised, and a nasogastric tube was inserted. Emergency imaging, including an abdominal X-ray and ultrasound, revealed pneumoperitoneum and free fluid in the abdomen with internal echoes, suggesting a perforated viscus. The patient had no prior medical or surgical history.

Biochemical evaluation revealed severe acidosis, leukocyte count of 7.06x10^3/cmm with 86% neutrophils, CRP 199.47 mg/dl, Procalcitonin 100, and deranged KFT indicating acute kidney injury (BUN

114.90 mg/dl, S. creatinine 3.8 mg/dl). The patient tested positive for Hepatitis B with S. Bilirubin 2.08 mg/dl, INR 2.7 sec, and albumin 2.50 g/dl. These results indicated MODS (multiple organ dysfunction syndrome). Eight units of FFP were transfused. Continued resuscitation achieved optimal urine output, BP 114/74 mm Hg, and INR 1.35 sec, allowing for emergency exploratory laparotomy. This revealed a 1.5 cm prepyloric perforation with about 2 litres of bilio-purulent contamination. The perforation was regular with mildly indurated margins. After biopsy and freshening the margins, Cellan Jones pedicled omental repair was performed, followed by extensive peritoneal lavage and insertion of abdominal drains in Morrison's pouch and pelvis.

The patient initially showed clinical improvement, was off inotropes in 24 hours with good urine output and improved KFT, and was extubated after 48 hours. On the 3rd POD, the nasogastric tube was removed, and oral liquids were started, followed by semisolids, which the patient tolerated well. HPE of the perforation margins showed unremarkable pyloric glands with surrounding fibro-collagenous stroma with inflammatory cells, and no atypia/granuloma was seen. The drain in Morrison's pouch was removed on POD 5. On the 6th POD, the patient began having seropurulent drain output (40-50 cc). The abdomen was clinically unremarkable, and the patient was passing stools/flatus. USG showed no abdominal collection. The total leukocyte count increased to 14x10^3/cmm, prompting an upgrade in antibiotics. On postoperative day 8, a bilious tinge in the drain output led to re-exploration, revealing complete omental necrosis with point bile leaks at the previously repaired perforation site. Complete omentectomy was performed, and the prepyloric perforation repair was reinforced with additional sutures to ensure durable closure and prevent further leakage.

A feeding jejunostomy was created to facilitate enteral feeding. Close monitoring and timely adjustments in management resulted in the gradual resolution of the patient's clinical abnormalities and normalisation of laboratory parameters. Jejunostomy feeding began 48 hours later, which the patient tolerated well. After demonstrating tolerance of oral intake, the feeding tube was removed 28 days post-re-exploration. The patient resumed oral feeding without further complications and was subsequently discharged.

III. Discussion

Perforated peptic ulcers pose serious threat to life, with mortality rate nearing 30%. Primary treatment approach involves prompt surgical intervention and proactive management of sepsis.[4]

Gona S K et al reported that among 161 patients operated on for perforated peptic ulcer, 36 (27.5%) experienced complications and 31 (19.3%) died. The most frequent postoperative complications were septic shock (11.8%), hypovolemia (9.9%), and wound sepsis (9.3%). Septic shock was significantly more common in deceased patients (61.3% vs. none) compared to those with only complications.[5] Wilhelmsen et al observed complications in 726 patients post-peptic ulcer perforation repair, with 124 (17.1%) requiring re-operative surgery, usually within 6.7 days. Reperforation was the leading cause of reoperation.[6]

In our patient, surgical repair was compromised by omental necrosis, necessitating re-exploration. Omental necrosis, although rare, is a serious complication, potentially arising without preceding torsion and linked to systemic conditions like sepsis, vasculitis, hypercoagulability, or thrombi.[7]

Sepsis-induced microvascular dysfunction and hemodynamic effects of inotropic agents likely impaired omental blood flow, causing ischemia and necrosis. Managing this involved resecting necrotic omentum, reinforcing the perforation repair, and creating a feeding jejunostomy, with close postoperative monitoring and timely intervention aiding recovery.

IV. Conclusion

We report a case of omental necrosis complicating prepyloric perforation repair, emphasising the importance of vigilance for postoperative complications following exploratory laparotomy. Early recognition and appropriate surgical intervention are crucial for optimising patient outcomes and minimising morbidity.

Disclosure

The authors declare no conflicts of interest.

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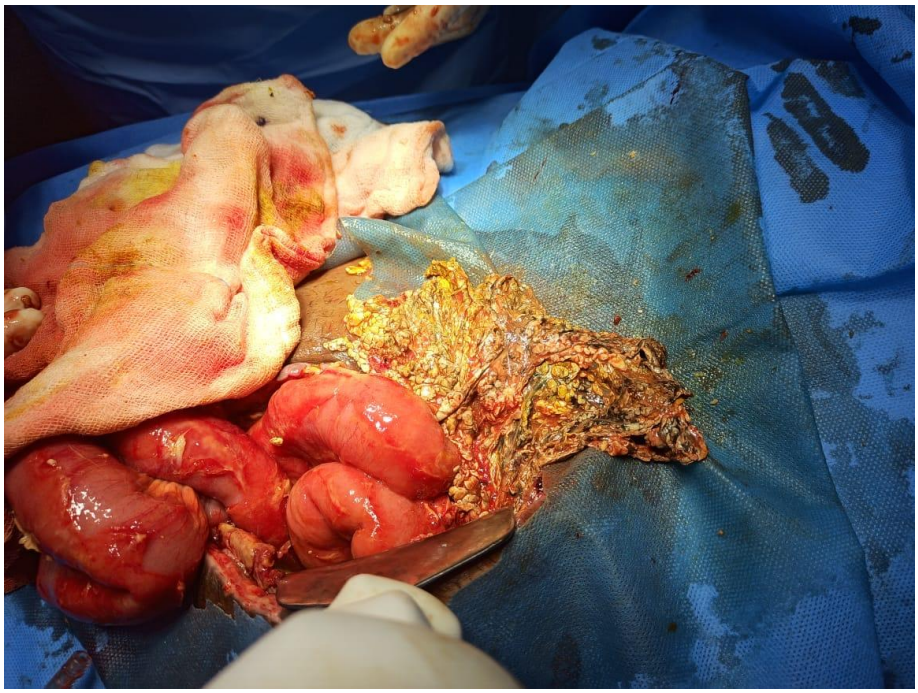

Pic 1: Intraoperative Picture During Re-Exploration Showing Omental Necrosis