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Case Report: Delayed Marginal Ulcer Perforation Five Years Post-Mini Gastric Bypass (one anastomosis gastric bypass) – A Rare Complication with Diagnostic and Therapeutic Implications

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1. Abstract

Marginal ulcer (MU) perforation is a serious complication following one anastomosis gastric bypass (OAGB), typically occurring within the first two postoperative years. We present a rare case of MU perforation occurring five years in a 21-year-old male with no traditional risk factors (no NSAID use, smoking, or active Helicobacter pylori infection). The patient presented with acute peritonitis and diagnosed via CT imaging, which revealed pneumoperitoneum and contrast extravasation at the gastrojejunal anastomosis. Emergency laparoscopic repair with omental patch reinforcement performed successfully. This case underscores that delayed MU perforation can occur well beyond the typical risk period, necessitating a high index of suspicion and long-term awareness in post-bariatric patients.

2. Introduction

The Mini Gastric Bypass (MGB), or One-Anastomosis Gastric Bypass (OAGB), is an effective bariatric procedure. A known complication is the formation of a marginal ulcer (MU) at the gastrojejunal anastomosis, with an incidence ranging from 0.6% to 16% [1]. Perforation is rare but life-threatening sequelae of MU, with most cases occurring within the first 6-24 months postoperatively [2]. The etiology is multifactorial, involving gastric acid exposure, ischemia, foreign material (sutures/staples), H. pylori infection, NSAID use, and smoking [3]. Perforations occurring more than five years after surgery are exceptionally rare and poorly documented in the literature. We report a case of MU perforation five years post-OAGB in a patient without conventional risk factors, highlighting the diagnostic challenges, management strategies, and implications for long-term follow-up.

3. Case Presentation

3.1. History

A 21-year-old male with a history of laparoscopic OAGB (performed five years prior for morbid obesity, preoperative weight 124 kg, current weight 66 kg) presented with acute epigastric pain, nausea, vomiting for two hours. He denied NSAID use, smoking, or comorbidities (diabetes, hypertension).

3.2. Examination & Investigations

- Vitals: HR 87 bpm, BP 110/66 mmHg, RR 17, SpO_2 99%.
- Abdomen: Diffuse tenderness with guarding, maximal in the epigastrium.
- Laboratory findings:
- Mild metabolic acidosis (pH 7.34, HCO_3^- 22.6 mmol/L).
- Normal WBC (8.62 $\times 10^3$/mcL)
- Hypokalemia (K^+ 3.3 mEq/L).

3.3. Examination

Vitals

HR: 87(Peripheral) RR: 17 BP: 110/66 SpO2: 99%

Tongue mildly coated, Looks mildly dehydrated

No pallor or jaundice

Afebrile

Abdomen: tenderness all over abdomen, with guarding, more at upper abdomen, hernial orifices were intact and healed port sites scars with no swelling or bulge at port sites, bowel sounds positive.

Ct Study of Abdomen & Pelvis with Contrast

3.4. Findings

The stomach shows surgical clips along its wall. Inhomogeneous fat stranding with a few extra luminal air bubbles seen beneath the epigastric quadrant adjacent to the stomach. Focal defect is seen apparently in the adjacent jejunal wall, communicating with the peritoneal cavity. Following oral administration of dilute iodinated contrast, it is seen to extend into the peritoneal cavity across the defect confirming the perforation. Pneumoperitoneum also noted along the anterior abdominal wall in the intrahepatic region and close to the lower half of liver. Liver, gall bladder, pancreas, spleen and adrenal glands appear normal in size and density characteristics. The kidneys appear normal bilaterally. The ureters are not dilated. Small and large bowel loops are not dilated. Appendix is not made out separately from the bowel loops due to paucity of intra-abdominal fat. Aorta & IVC appear normal in caliber. Mild ascites noted in the pelvis. Urinary bladder is partially distended. No gross intra-vesical pathology noted. Prostate and seminal vesicles are normal.

Impression: Hollow viscus perforation as mentioned above.

3.5. Management

Patient received IV fluids, NG tube placement and IV broad spectrum antibiotic (Piperacillin Tazobactam 4.5Gm). Preoperative diagnosis was made as Perforated Viscus (Possible Marginal Ulcer Perforation as free gas was seen near stomach/liver area and oral contrast leak was from area at gastro-jejunostomy anastomosis site)

Patient was prepared and taken for Diagnostic Laparoscopy and proceed. Peroperatively following findings were found

1. Free fluid in peritoneal cavity- Mild contamination in upper abdomen and pelvis

2. Mild flecks of exudates present

3. Small perforation at the Gastro-jejunostomy site- on posterior aspect- confirmed by instillation of saline thru NG tube

3.6. Operative Management

Open Hassan technique for Pneumoperitoneum- done thru previously placed port site at supra-umbilical area.3 working ports placed under triangulation (10mm at supra-umbilical -camera port, 5mm at RHC, 10MM at LHC- thru previous scars)

Diagnostic laparoscopy showed mild free fluid in upper abdomen and pelvis.

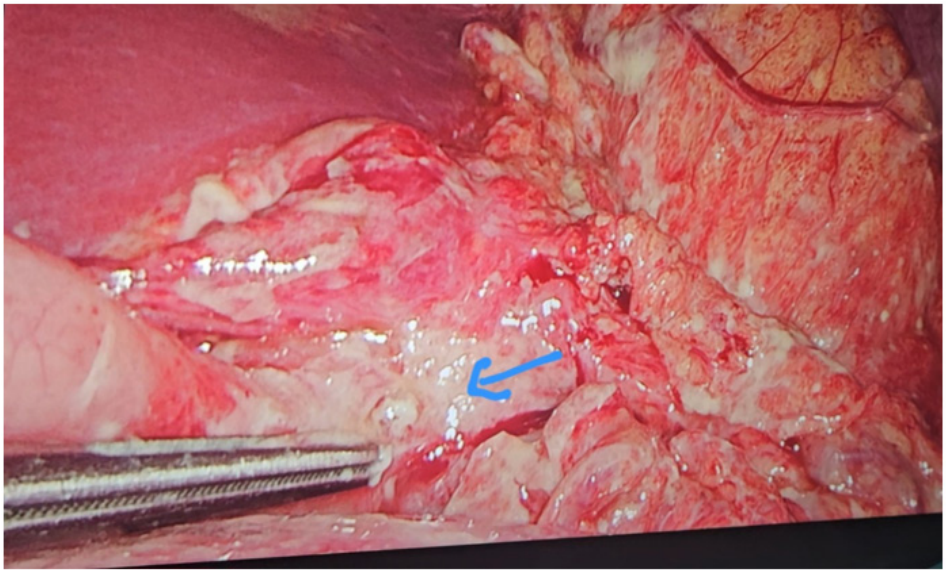
Small 2mm perforation found at posterior aspect of gastro-jejunostomy. Two more working ports placed for retraction and better work ergonomics. Two sutures of PDS 2/0 taken by interrupted technique- intracorporeal knot technique and perforation site closed.

Perforation site after primary closure re-enforced by placement of omental patch and secured. Hemostasis was ensured and through lavage of peritoneal cavity done till clear fluid. 19 Fr J-Vac drain placed close to gastro- jejunostomy site.

3.7. Post Op course

He has smooth post-operative recovery. Nasogastric tube was removed on 1st postoperative day and was started on clear liquid diet. Peritoneal drain was removed on 2nd postoperative day as output was minimal and it was only serosanginous fluid. His peritoneal fluid culture report showed following result. Infectious disease consultation was done and patient was recommended for continuation of Piperacillin Tazobactam. His inflammatory markers trended down gradually to normal levels so he was discharged on postoperative day 5. He was discharged on H. Pylori eradication therapy (Amoxicillin, Clarithromycin) and PPI therapy for 1 month. He was followed in surgical clinic in 1 week time and he was found not to have any postoperative complications and recovering well. He was recommended for further follow-up with his primary Bariatric surgeon and to consider for upper GIT endoscopy and barium studies after lapse of more than one month so that cause of marginal ulcer formation could be determined and further catastrophic events can be prevented.

Radiological Imaging



Pus Culture -
Result Status - Auth (Verified)

Micro Reports | **Susceptibilities** | Specimen | Comments | Acti

	A	B
1	Streptococcus agalactiae (Group B)	
2		MIC Interp
3	Ceftriaxone	S
4	Chloramphenicol	S
5	Clindamycin	S
6	Erythromycin	S
7	Levofloxacin	S
8	Linezolid	S
9	Moxifloxacin	S
10	Tetracycline	R
11	Tigecycline	S

4. Discussion

The Mini Gastric Bypass (MGB), or One-Anastomosis Gastric Bypass (OAGB), is established as a effective and technically simpler alternative to Roux-en-Y Gastric Bypass (RYGB) [1]. While its efficacy is comparable, its distinct anatomical configuration a long, narrow gastric tube anastomosed directly to a loop of jejunum creates a unique pathophysiological environment predisposing to complications like marginal ulcers (MUs). Our case of a perforated MU presenting a full five years postoperatively in a patient devoid of traditional risk factors is a paradigm-shifting event. It compels a critical re-examination of the natural history of MUs after MGB, moving beyond the established early-postoperative timeline and forcing a consideration of chronic, procedure-specific etiologies that demand lifelong vigilance.

4.1. The Enigma of Delayed Presentation and the Bile Reflux Hypothesis

The cardinal feature of this case is its exceptionally late presentation. The consensus, largely derived from RYGB literature, posits that over 90% of MUs occur within the first two years, with technical factors (tension, ischemia, stapling issues) and acute exposures (NSAIDs, smoking, H. pylori) being the primary culprits [2,3]. Our case, occurring five years postoperatively without these classic triggers, suggests a different, more insidious pathogenesis. The most compelling etiological agent specific to the loop anatomy of MGB/OAGB is chronic alkaline reflux [4,5]. The long biliopancreatic limb, essential for the procedure's malabsorptive component, creates a direct conduit for bile and pancreatic secretions to reflux into the acid-producing gastric tube and onto the unprotected jejunal mucosa at the anastomosis. This creates a "double-hit" phenomenon: direct chemical injury from bile acids disrupting the mucosal barrier, followed by acid-driven digestion of the compromised tissue. This chronic, low-grade injury may take years to erode through the mucosa and manifest as a symptomatic or perforated ulcer. This mechanism is less prevalent in RYGB due to the protective nature of the Roux-en-Y configuration, which diverts biliopancreatic secretions away from the anastomosis [6].

4.2. Beyond Bile: Other Contributing Mechanisms in a Multifactorial Model

While bile reflux is a strong candidate, other factors likely contribute to this delayed presentation. Chronic ischemia at the anastomosis, due to the division of small vessels and potential tension from a mobile jejunal loop, can create a watershed area with impaired healing capacity [7]. Furthermore, a foreign body reaction to non-absorbable sutures or staples can incite a per-

sistent local inflammatory state, acting as a nidus for chronic irritation that, over time, progresses to ulceration [2]. The location of the perforation on the posterior aspect is noteworthy, as this is a technically challenging area to assess for perfect serosal apposition and freedom from tension during the initial surgery. It is also plausible that subclinical nutritional deficiencies in micronutrients like zinc or vitamins crucial for mucosal integrity (e.g., Vitamin A, B12) over many years could impair the innate repair mechanisms of the jejunal epithelium, making it more susceptible to other injurious factors [8].

4.3. Diagnostic Nuances and the Pitfall of Normal Labs

This case perfectly illustrates the critical role of a high clinical index of suspicion. The differential diagnosis for an acute abdomen in a post-bariatric patient is broad and includes internal hernia, band erosion, perforated peptic ulcer, and MU. Contrast-enhanced CT is the cornerstone of diagnosis, with findings of pneumoperitoneum, extra luminal oral contrast extravasation at the GJ anastomosis, and surrounding inflammatory fat stranding being pathognomonic for perforated MU [9]. A key learning point is the unreliability of laboratory markers in the hyper acute phase. Our patient's normal white blood cell count is a classic pitfall, as the systemic inflammatory response may not have had time to manifest, underscoring that management must be guided by clinical and radiological findings, not wait for laboratory confirmation.

4.4. Surgical Management and Evolving Techniques

The laparoscopic approach is unequivocally the standard of care. The principles employed here—thorough lavage, primary repair with interrupted absorbable sutures, and reinforcement with a well-vascularized omental patch—are time-tested and effective [10]. The omentum is not merely a mechanical plug but provides a source of neovascularization and immunologic defense, which is crucial for healing in a potentially ischemic and contaminated field. For recurrent or complex ulcers, more definitive procedures like conversion to RYGB (to divert bile) or antrectomy (to reduce acid production) may be necessary, but were not required in this first-time presentation [11].

4.5. Paradigm Shift in Long-Term Management: From Episodic to Lifelong Care

This case has profound implications that should reshape post-MGB patient management. It definitively dismantles the concept of a "cured" patient after a few years. Instead, it mandates a paradigm of structured, lifelong follow-up. This includes:

1. Indefinite Patient Education: Patients must be counseled for life on the symptoms of MU (epigastric pain, nausea, vomiting) and the absolute, non-negotiable avoidance of NSAIDs.

2. Re-evaluation of PPI Therapy: The routine practice of short-term PPI prophylaxis post-MGB must be questioned. Given the chronic acid and bile insult, a strong case can be made for long-term or indefinite PPI therapy in all MGB patients, a strategy supported by a growing body of opinion [4, 12].

3. Proactive Surveillance: A low threshold for performing elective upper endoscopy to investigate any new upper GI symptoms, even decades after surgery, is essential. This allows for the early detection and treatment of non-perforated ulcers, preventing catastrophic complications.

4. Nutritional Vigilance: Long-term monitoring and supplementation for micronutrients vital for mucosal health should be a standard part of follow-up protocols.

5. Conclusion

this case report serves as a critical alert to the bariatric community. Perforated marginal ulcer after MGB is not solely an early complication but can be a late and devastating event driven by procedure-specific factors like chronic biliary reflux. It necessitates a permanent shift in our mindset from episodic post-operative care to lifelong, proactive patient management to mitigate this serious risk.

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