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Research Article

Necrotizing Pancreatitis After Gastric Bypass: A Unique Case Highlighting the Need for Guideline Adherence or Strategic Deviation for Better Outcomes

Running Title: Necrotizing Pancreatitis after gastric bypass

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ABSTRACT

Introduction: Bariatric surgery, including Sleeve Gastrectomy (SG) and Roux-en-Y Gastric Bypass (RYGB), is an effective procedure for weight loss but carries postoperative risks such as acute pancreatitis (AP). We present a case of AP post-RYGB leading to necrotizing pancreatitis, sepsis and respiratory failure, which required complex care.

Case presentation: A female in her early 50s presented to the emergency room with acute abdominal pain two and half months post-Roux-en-Y Gastric Bypass (RYGB). Initial tests revealed acute pancreatitis on CT, but biliary dilation and gallstones were not detected. Despite standard of care her condition worsened leading to transfer to intensive care unit. Repeat CT showed necrotizing pancreatitis, ascites and pleural effusions. At a tertiary care center, an EUS showed altered anatomy due to RYGB.

Initial drainage procedures were deferred until the pancreatic collection matured. Sixteen days later, an EUS-guided cyst-gastrostomy failed due to solid necrosis. She developed sepsis, requiring vasopressors and broad-spectrum antibiotics lead to IR-guided drainage of newly developed abscess. A follow-up CT indicated extensive necrosis, prompting an endoscopic ultrasound directed trans-gastric ERCP (EDGE) procedure with lumen apposing metal stent (LAMS) placement. The patient later developed a pleural effusion requiring chest tube insertion. Persistent necrosis led to an endoscopic necrosectomy and a double pigtail stent placement. She was discharged with oral antibiotics and follow-up recommendations after a prolonged hospital course involving multidisciplinary management and complex procedures.

Conclusion: Bariatric surgery-associated pancreatitis may require earlier, non-standard interventions due to altered anatomy; research is needed for guideline updates.

Keywords: Acute necrotizing pancreatitis; Gastric Bypass; Guidelines; EDGE procedure; LAMS; OTS clips

Introduction

Bariatric surgery is a widely recognized treatment for individuals with a high BMI. Sleeve Gastrectomy (SG) and Roux-en-Y Gastric Bypass (RYGB) are being the two most commonly performed procedures. Both surgeries are effective in promoting long-term weight loss and improving obesity-related health conditions, while also having comparable low perioperative risk profiles[1]. Biter LU et al. illustrated overall outcomes of the RYGB, with higher total weight loss following RYGB compared to SG and lower rates of de novo GERD and dyslipidemia[2].

While RYGB is generally safe and effective, there are still potential postoperative complications, such as early anastomotic leaks, which have an incidence rate of approximately 0.6% to 4.4%[3]. Other complications include gastrointestinal bleeding, venous thromboembolism and kinking or stenosis of the gastrointestinal tract, gallstones, small bowel obstruction, marginal ulceration and internal hernia[3]. Acute pancreatitis (AP) is a less common complication following bariatric surgery, with single-center studies reporting rates between 0.2% and 1.04% for both SG and RYGB[3,4]. The Mean time frame of developing AP after bariatric surgery is 3.5 years[5]. There was an increased risk of developing AP in patients who had undergone RYGB especially in the case of prior cholecystectomy, whereas the presence of only gallstones had a lower rate of incidence[6]. AP regardless of the type of bariatric surgery however tends to be mild and does not require escalations of care such as an ICU admission[3,4]. Kroner, et al. compared the mortality of AP in between patients with a history of bariatric surgery and without a history of bariatric surgery, resulting in patients with a history of bariatric surgery tend to have lower mortality, morbidity and resource utilization[7]. Nonetheless, it can be fatal and necessitates early diagnosis and treatment[5]. We present a case report of acute pancreatitis following RYGB, which got complicated with necrotizing pancreatitis, ascites, sepsis and acute hypoxic respiratory failure, requiring a prolonged hospital stay and necessitating uncommon procedures due to altered anatomy.

Case Presentation

A female in her early 50s presented to the emergency room with acute onset of abdominal pain, sharp in nature, located at periumbilical region without any radiation. Abdominal pain was progressively worsening without any aggravating or relieving factors and it was associated with nausea, non-bloody non-bilious vomiting and loss of appetite. She had a significant past medical history of Class 3 Severe Obesity status post Laparoscopic Roux-en-Y Gastric Bypass Surgery two and a half months before the current presentation. Other past medical history was urinary incontinence managed with urethral suspension and retropubic sling placement, asthma, obstructive sleep apnea, gastroesophageal reflux disease, anxiety, hyperlipidemia, prediabetes, breast augmentation and a tubal ligation.

Upon presentation, she was afebrile with a maximum temperature of 98.2°F, vitals were as follows: BP 170-180s/80-100s mm Hg, HR 80s-90s bpm, RR18 - 20s/min. Laboratory workup was noticeable for Amylase 1162 U/L, Lipase 2787 U/L, Bicarb 21 mmol/L, ALT 57 U/L, AST 67 U/L, glucose 170 mg/dL, BUN 24 mg/dL, creatinine 1.5 mg/dL, lactate 2.6 mmol/L and an elevated white blood cell count 14.9 x10^3/uL. CXR did not show any evidence of acute cardiopulmonary disease. CT scan of the abdomen and pelvis with IV contrast showed acute pancreatitis with extensive fluid and peripancreatic inflammatory changes throughout abdomen and possible developing ileus **(Figure 1A and 1B)**. However, no evidence of gallstone or biliary dilatation or organized fluid collection or necrosis were noticed. In ED, she received a total of 4396 ml of ringer lactate (LR), IV antibiotics (Piperacillin-tazobactam), IV morphine 4 mg x2, IV hydromorphone 1.2 mg and IV antiemetics. The Patient was admitted for the management of acute pancreatitis and she was started on ringer lactate (LR) at 200 cc/hr, PRN antiemetics and pain medication and IV Piperacillin-tazobactam. Patient was kept NPO due to nausea and vomiting and IV pantoprazole was started.

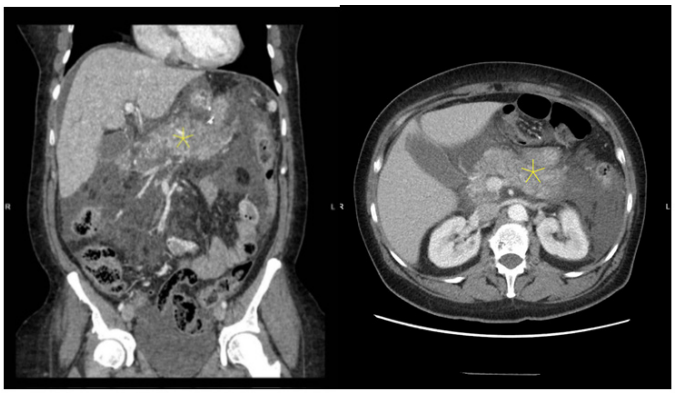

Figure 1A and 1B: CT scan image of showing inflammation of pancreas (Yellow Star).

The patient remained hemodynamically stable overnight but continued to experience severe abdominal pain for which she was started on hydromorphone PCA pump. Next morning,

she developed abdominal distension, obstipation, increased guarding, hypoactive bowel sounds and tympany, accompanied by persistent tachycardia and BP in 140s-150s/90s-100s mm Hg, despite her pain being well controlled with the PCA pump. She was saturating well on room air without any abnormal beathing sounds on examination. Her morning labs showed results of BUN and creatinine getting better (20 mg/dL and 1.00 mg/dL respectively), however, increasing of hematocrit (HCT) to 48.8 was concerning, hence, ICU evaluation was obtained for clinically worsening of pancreatitis and general surgery team was consulted for concern regarding abdominal compartment syndrome. Due to worsening of clinical condition including tachycardia, persistent hypertension, worsening abdominal distension, guarding and rigidity and persistent obstipation as well as newly developed oliguria, dyspnea and worsening HCT she was transferred to the ICU for closer monitoring and higher level of care. Subsequently, US abdomen revealed cholelithiasis and distended gallbladder. Due to progressively deteriorating clinical condition and worsening lactic acidosis, repeat STAT CT abdomen and pelvis was performed, which showed necrotizing pancreatitis **(Figure 2)**, large loculated ascites and bilateral pleural effusion with lower lobe atelectasis.

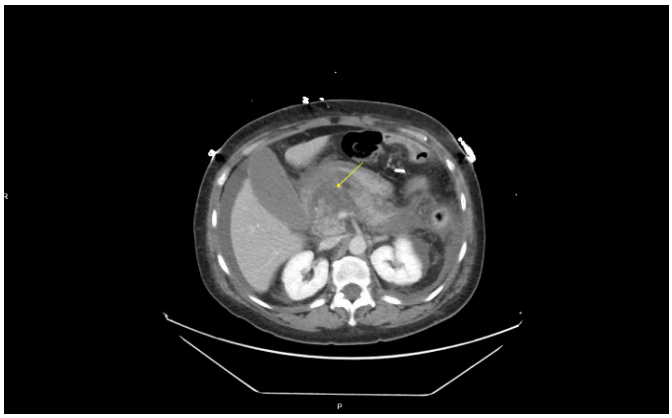

Figure 2: CT scan showing pancreatic necrosis (Yellow arrow).

On the next day, she LR rate was lowered at 75 ml/hr and eventually stopped and IV furosemide 20 mg was given, which yielded only 125 cc urine output. Due to progressively worsening dyspnea and a failed trial of NIV (non-invasive ventilation), the patient was intubated. Bladder pressure was elevated at 13 mmHg. Later, she was transferred to the tertiary center for higher level of care for possible endoscopic ultrasound (EUS) cysto-gastrostomy and necrosis drainage.

At the tertiary care center, she underwent Endoscopic Ultrasound (EUS) with a planned gastro-gastrostomy and stent placement. The EUS procedure revealed altered surgical anatomy consistent with a Roux-en-Y Gastric Bypass (RNYGB), with the esophagus, gastroesophageal (GE) junction and gastrojejunostomy (GJO anastomosis being normal. During the EUS, the excluded stomach was identified and distended using a 19-gauge needle under fluoroscopic guidance. However, it showed that most of the neck and body of the pancreas were best visualized from the pouch near the intended stent placement site. Due to concerns about interfering with pancreatic drainage, the decision was made to defer the EDGE (Endoscopic ultrasound directed trans-gastric ERCP) procedure for future peripancreatic fluid drainage when the collection becomes mature. Following the procedure, the patient was hemodynamically stable and was downgraded from the ICU and she was managed conservatively.

After 16 days, once the collection matured, she underwent endoscopic ultrasound (EUS) guided gastro-cystostomy but was unable to have the stent open properly due to the solid nature of collection, hence it was closed with over the scope clip (OTSC). Following this procedure, overnight she became febrile at 100.7-degree F, tachycardic at 110s-120s beats per minute, hypotensive 60s-70s/40s mm Hg, blood work-up was notable for WBCs 37,000/uL (from 7000/uL) and lactate 2 mmol/L, creatinine 1.5 mg/dl. Subsequently, she was transferred to the ICU for the requirement of the vasopressors, she was given one dose of meropenem, vancomycin and tobramycin each and was continued on meropenem. STAT CT abdomen and pelvis was performed which showed enhancing pancreatitis with slightly larger necrotic pancreatic wall, now containing multiple pockets of air concerning the superimposed infection, abscess or the fistulation to adjacent bowel. IR guided drainage and Jackson-Pratt (JP) drain placement showed purulent collection **(Figure 3)**. She was continued on meropenem for antibiotic and started improving transiently with coming off pressors next day of drain placement.

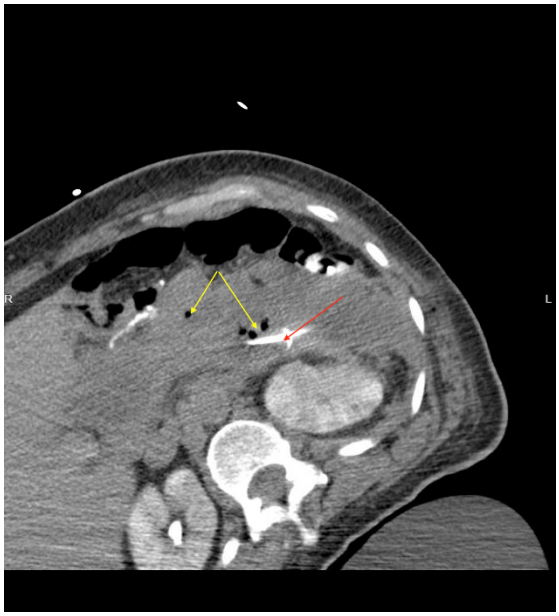

Figure 3: CT image showing multiple air pockets (Yellow arrow) and JP drain placement (Red arrow).

Five days later, a follow-up CT scan of the abdomen and pelvis revealed extensive pancreatic and peripancreatic necrosis extending bilaterally to the pelvis, with some improvement in the previously noted walled-off necrosis in the body and neck, where a pigtail catheter was already in place. After careful consideration by a multidisciplinary team, it was decided to proceed with an EDGE procedure, involving the placement of a lumen-apposing metal stent (LAMS) at a cysto-gastrostomy through a gastro-gastric (GG) fistula at the remnant stomach, to drain necrotic pancreatic and peripancreatic material **(Figure 4)**. Two weeks later, the JP drain was removed. However, shortly after removal, the patient developed a large left-sided pleural effusion **(Figure 5)**, causing tension physiology, for which a chest tube was placed. Meanwhile, repeat CT imaging showed no change from the prior scan, indicating a failed cysto-gastrostomy stent in draining the collection. As a result, the patient underwent an endoscopic necrosectomy with the placement of a double pigtail stent to aid drainage. The patient was eventually discharged with oral antibiotics and advised to follow up with outpatient

gastroenterology. The stent was removed 40 days after the initial placement and plan was made to do cholecystectomy.

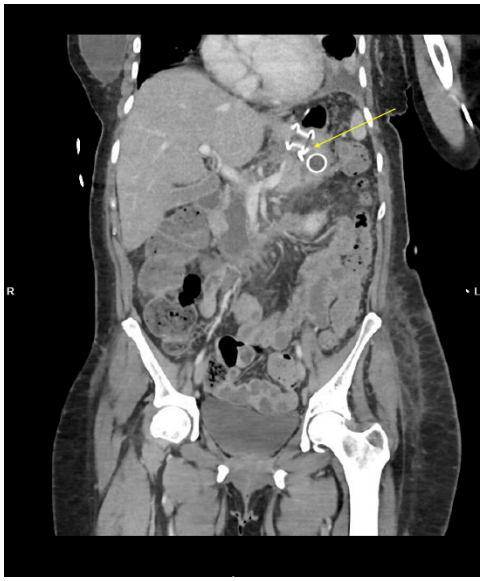

Figure 4: CT image with necrotizing pancreatitis and lumen-apposing metal stent (LAMS) at a cysto-gastrostomy (Yellow arrow).

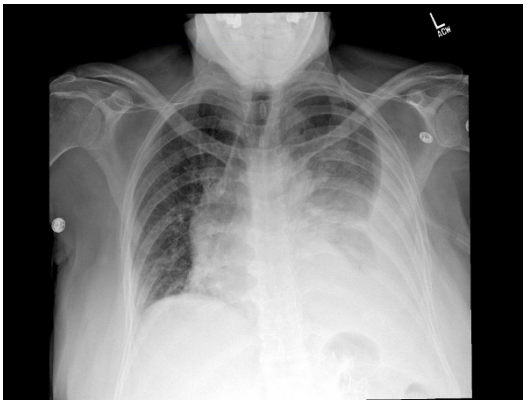

Figure 5: Chest Xray showing large left sided pleural effusion.

Discussion

Acute pancreatitis is not an uncommon complication of bariatric surgeries like sleeve gastrectomy or Roux-En-Y gastric bypass. This case highlights a rare situation where a patient admitted for acute pancreatitis quickly progressed to necrotizing pancreatitis within 48 hours, gallstones noted on ultrasound could be a contributing factor, however, gallstones can be a byproduct of rapid weight loss because of bariatric surgery. This case also highlights an altered anatomy after gastric bypass which might have contributed to such a prolonged and complicated hospital course and forced us to explore some unconventional routes in making interventions.

Although post-gastric bypass pancreatitis is typically mild, severe cases have been reported. Baran et al. and Wang et al. described instances of severe pancreatitis due to blood clots in the biliary tree after laparoscopic Roux-en-Y, likely caused by the reflux of pancreatic and biliary contents through the ampulla of Vater[5,8]. Daster, et al. suggested that the mechanical effects of the surgery could lead to pancreatic inflammation from biliary leakage[9]. However, unlike these cases where pancreatitis occurred during or shortly after surgery, our patient developed acute pancreatitis months later. Weight loss after bariatric surgery improves cardiovascular health, potentially reducing the severity of pancreatitis[7]. Kerbage, et al. found that mortality and outcomes in patients with post-surgery pancreatitis are similar to those without weight loss surgery[5].

Furthermore, gallstones are the most common risk factor for developing necrotizing pancreatitis[10]. Rapid weight loss after bariatric surgery, particularly in patients with a BMI over 40 kg/m², is a known cause of cholelithiasis[11], as is the case with our patient. Bariatric surgery mobilizes cholesterol and triglycerides from adipose tissue into the blood, leading to increased liver uptake. The liver then secretes these lipids into bile, causing supersaturation and gallstone formation. In addition to above, reduced gastrointestinal and gallbladder motility, due to lower cholecystokinin levels, contributes to cholestasis and promotes gallstone formation[12]. The incidence of gallstones after bariatric surgery, in fact, have been so common that physicians have considered prophylactic concurrent cholecystectomies. This has also been thought to mitigate the risk of acute pancreatitis caused by gallstone formation[11,13]. A conservative approach to prevent gallstones and reduce pancreatitis risk post-surgery is a 6-month course of 500-600 mg daily ursodeoxycholic acid (UDCA) as explained by Miller et al. and Sugerman, et al.[14,15]. Interestingly enough, afferent loop syndrome which is the distention of the biliopancreatic-jejunal anastomosis after a Roux-en-Y procedure has been shown to cause pancreatitis, be it an early or late complication[16,17]. While it not being the primary cause, afferent loop distention may be contributory in the outcomes of the case above and undoubtedly puts the patient at a higher risk of developing pancreatitis than the general population.

In severe cases of acute pancreatitis, the leakage of pancreatic enzymes can result in damage to the pancreatic tissue, leading to complications such as fluid accumulation in spaces, including ascites and pleural effusions. Additionally, a compromised blood supply to the pancreatic tissue can result in necrosis. Necrotic tissue is at risk of an infection, which can progress to sepsis and multi-organ failure. Infected necrotizing pancreatitis carries a mortality rate of nearly 100% without intervention and up to 30% even with surgical treatment[18,19]. Current guidelines recommend moderately aggressive fluid resuscitation during the initial 24-48 hours to mitigate inflammatory leakage and prevent necrosis[20]. Evidence also supports early enteral nutrition as soon as it is tolerated, as it reduces the risk of infectious complications by preventing gut bacterial translocation[20]. However, while early feeding helps lower the risk of infection, it does not necessarily prevent necrosis, making frequent monitoring essential during the first 48 hours, ideally every 6-8 hours[20,21]. This includes tracking BUN and hematocrit (HCT) levels, along with bedside assessments of vital signs, urine output and overall fluid status. Ideally, decreasing BUN and HCT levels indicate adequate hydration and renal perfusion. Conversely, persistent or rising levels are associated with a poor prognosis and may necessitate escalation of care. In our case, the observation of an elevated heart rate, blood pressure and rising HCT levels suggested clinical deterioration, prompting notification of intensive care for higher-level management. When a patient does not respond to optimal treatment, investigations for complications are warranted. In our case, repeat CT imaging revealed necrotizing pancreatitis that rapidly progressed to infected necrosis, despite adherence to guideline-based management.

In this case, the altered anatomy following Roux-en-Y gastric bypass presented significant challenges for performing

advanced endoscopic procedures. According to the American College of Gastroenterology (ACG) guidelines, in cases of acute necrotizing pancreatitis involving stable patients, interventions should ideally be delayed until the necrotic collection matures (ideally 4 weeks)[20]. Our management approach attempted to align with these recommendations by initially deferring the EDGE (endoscopic ultrasound-directed trans-gastric ERCP) procedure to a later stage. However, due to clinical deterioration, we proceeded with the intervention around the three-week mark, which ultimately failed and necessitated a necrosectomy with drain placement.

Kroner et al. proposed that patients with a history of bariatric surgery may experience reduced mortality, morbidity and resource utilization in cases of acute pancreatitis[7]. This is likely attributed to post-surgical changes affecting pancreatic and gastrointestinal functions, including both hormonal and anatomical modifications. In retrospect, evaluating whether this patient truly aligned with the typical profile of a stable acute necrotizing pancreatitis case is essential. The altered anatomy from the Roux-en-Y gastric bypass, specifically the gastric and peripancreatic surgical changes, likely played a significant role in compromised peripancreatic drainage. This could have contributed to inadequate fluid evacuation and increased the risk of local and systemic complications. The consideration arises as to whether performing an EDGE procedure earlier might have provided any benefit by addressing the drainage issue earlier and preventing complications from progressing.

Furthermore, the patient's unique anatomical configuration could have predisposed her to a higher risk of infection and sepsis due to the impaired drainage. The necessity for multiple interventions may have exacerbated this risk, potentially compounded the inflammatory response and facilitated septic progression. Ultimately, the combination of altered anatomy and delayed but necessary procedures extended the clinical course and may have necessitated prolonged management and monitoring. These insights underscore the importance of individualized decision-making in cases involving complex postoperative anatomy, where early intervention strategies might need to be adapted to optimize patient outcomes and prevent severe complications.

Conclusion

Bariatric surgery may be directly or indirectly associated with acute pancreatitis (AP) by promoting gallstone formation because of rapid weight loss. This case involves acute pancreatitis that progressed to necrotizing pancreatitis, which subsequently became infected and leaded to sepsis, despite adherence to initial guideline-based management. Here, we emphasize the importance of monitoring critical parameters to assess the prognosis and initiate earlier escalation of care. A significant point of discussion is about the potential benefits of earlier intervention that deviates from standard guidelines, particularly in patients with altered functional and anatomical conditions. Based on our hypothesis and observations, we advocate for further investigation into whether early endoscopic intervention results in improved outcomes for patients with a history of an altered anatomy who develop necrotizing pancreatitis.

Declaration

Authors declare no conflict of interest regarding the publication of this article.

Disclosure

Authors declare no financial/funding source(s) regarding the publication of this article.

Previous Publication/presentation/submission

This manuscript has never been considered for publication/presentation/submission as a full manuscript or part of it.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Author Contribution

- **KJY:** Conceptualization, Data Collection, Writing-Review and Editing, Project Administration, Visualization, Validation.
- **SA:** Writing-Review & Editing, Resources, Data Collection.
- **PC:** Writing Original Draft, Data Collection.
- **MP:** Writing Original Draft.
- **JU:** Writing Original Draft.
- **NN:** Writing Original Draft.
- **AK:** Writing Original Draft.
- **AK:** Writing Original Draft
- **DK:** Review & Editing, Project Administration, Supervision.
- **AVP:** Review & Editing, Project Administration, Supervision.
- **SS:** Review & Editing, Supervision.
- **WG:** Review & Editing, Project Administration, Supervision, Validation.

Data Availability Statement

All data used to support the findings of the study are included within the article, no additional data required.

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