

Case Report

Post-duodenal polypectomy pancreatitis: an uncommon iatrogenic trigger

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ABSTRACT

Acute pancreatitis (AP) is a major cause of gastrointestinal-related admission in the United States, characterized by epigastric pain radiating to the back, and nausea and vomiting. Although commonly associated with alcohol consumption and gallstones, cases involving less frequent triggers, such as post-endoscopy, have been reported. We present an unusual case of post-polypectomy pancreatitis following esophagogastroduodenoscopy (EGD). A 20-year-old woman underwent polypectomy during a bariatric EGD screening procedure, resulting in a 2 mm non-bleeding pedunculated polyp's polypectomy from the second part of the duodenum. She experienced excruciating abdominal pain, nausea, and vomiting within an hour. Following an emergency evaluation, the diagnosis of acute pancreatitis was made; this was supported by increased lipase enzyme and physical manifestation. While AP tends to be triggered by well-known reasons, this particular case highlights a distinctive iatrogenic trigger-duodenal polypectomy. Only a handful of similar cases have been documented. The duodenum's peculiar proximity to important pancreatic structures may result in mechanical injury or heat-induced pancreatic damage. The management, similar to typical acute pancreatitis care, underscores the importance of supportive measures. This rare case of post-polypectomy pancreatitis emphasizes the risk of iatrogenic complications. Timely diagnosis and surveillance following procedures involving the duodenum can help with early diagnosis.

Keywords: Duodenum, Endoscopy, Polyp, Pancreatitis, Iatrogenic

INTRODUCTION

Acute pancreatitis (AP) is the leading cause of gastrointestinal-related hospital admissions within the United States. Patients typically present with epigastric pain radiating to the back, nausea, and vomiting. Although most cases of AP are self-limiting, the mortality rate in severe cases can exceed 20%.¹ As a condition, AP also varies in its clinical course, aetiologies, and epidemiology. Although most cases of AP in developed countries can be attributed to alcohol use, gallstones, and

hypertriglyceridemia, several iatrogenic causes of AP have been reported in the clinical literature.² Iatrogenic AP is diagnosed by confirming AP and ruling out other common causes.

Diagnosis is established with the $\frac{2}{3}$ criteria: abdominal pain characteristic of AP; CT abdominal imaging with characteristic findings, and serum amylase/lipase three times the upper limit. Other laboratory studies with diagnostic value (i.e., trypsinogen, c-reactive protein, pancreatic proteases, IL-6, and IL-8) can help confirm the diagnosis and guide treatment plans. Common iatrogenic

causes of AP include medications (e.g., statins, valproic acid, diuretics, and ACE inhibitors).²⁻³ Less commonly reported iatrogenic causes include colonoscopy, epidural steroid injection, and gastrectomy.⁴⁻⁵ We present the case of a 20-year-old woman who was admitted to Medicine with AP post-polypectomy after an EGD.

CASE REPORT

History of present illness

A 20-year-old female presented to the Emergency Department with severe epigastric and chest pain, radiating to the back. Earlier that day, she underwent a screening EGD for bariatric surgery, during which a pedunculated duodenal polyp at the second part of the duodenum (Figure 1) was resected. She was discharged, but 3 hours later, she developed nausea accompanied by abdominal and chest pain as described above. No prior similar episodes were reported. Upon evaluation, she was noted to be in severe pain, which prompted further evaluation.

Past medical history

Her medical history includes obesity (BMI 45.9 kg/m²), prior psychiatric diagnoses (adjustment disorder, anxiety, depression, bipolar disorder), and a sexual assault in 2021. She was previously treated with fluoxetine 20 mg, quetiapine 25 mg, and aripiprazole 5 mg; however, these were discontinued eight months prior to admission. She had a partial salpingectomy for a paratubal cyst and currently has a right ovarian cyst. She also had an ablation for supraventricular tachycardia four months before admission.

Physical examination

Initial vital signs included a temperature of 37°C, heart rate of 129/min, blood pressure of 105/95 mmHg, respiratory rate of 18/min, and 99% room air saturation. Her BMI on admission was 45.6 kg/m². She was alert, and fully oriented, though in distress due to the pain. Bilateral chest auscultation was clear, and she exhibited tenderness in the epigastric region at palpation, globulous due to body habitus, no rebound or guarding.

Diagnostic workup

ECG showed a normal sinus rhythm. A qualitative pregnancy test was negative. Laboratory workup showed elevated Lipase (>600 ; $N=<60$), Leukocytosis (17.09×10^3 (3)/mcl; $N=4.80-10.80 \times 10^3$ (3)/mcl), normal triglycerides (98 mg/dL; $N=<150$ mg/dl). Other laboratory results, including lactic acid, basic metabolic panel, phosphorus, magnesium, and calcium, were unremarkable, as shown in Table 1. Further, computed tomography (CT) of the abdomen and pelvis scan with contrast revealed pancreatic fat induration and fluid accumulation (Figure 2). An abdominal ultrasound confirmed the absence of gallstones

and lack of biliary duct dilation in the gallbladder. The diagnosis of AP was confirmed based on the three diagnostic criteria (abdominal pain unequal from usual AP, an elevated serum lipase level three times above the normal threshold, and radiological imaging signs of pancreatitis).

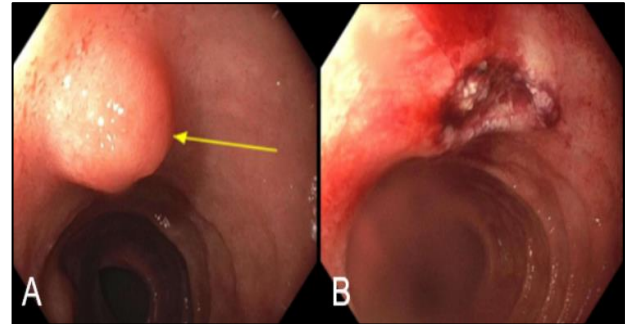


Figure 1: The screening esophagogastroduodenoscopy (EGD), shows a pedunculated polyp at the second portion of the duodenum proximal to the ampulla (A) (see yellow arrow). Polypectomy was performed using a hot snare (B).



Figure 2: Computed tomography (CT) of the abdomen with IV contrast. There is an induration of the fat and fluid surrounding the pancreas (see red arrows), suggesting acute pancreatitis.

Treatment and follow-up

Upon admission, she received IV fluids (litres), morphine for pain, and ondansetron for nausea. Later on, she transitioned from morphine to IV ketorolac, acetaminophen, and oral gabapentin. The following day, enoxaparin was started for DVT prophylaxis, and piperacillin-tazobactam was started due to suspicion of retroperitoneal perforation post polypectomy. On day 3, she began passing flatus after transitioning to clear liquids. On day 5, she walked with minimal pain, advanced to a general diet, and was given high-protein Ensure. She was

discharged on day 9 with minimal discomfort and prescribed acetaminophen and ibuprofen for pain

management, along with instructions regarding alarm symptoms.

Table 1: Summary of the laboratory results. Note the elevated Lipase levels that correlate with her current symptoms and CT scan of the abdomen.

Results (reference range)	
Complete blood count	Hemoglobin 12.4 g/dl (12.0-16.0 g/dl)
	Platelets 418×10 ³ /mcl (150-450×10 ³ /mcl)
	White blood cells 17.09×10 ³ /mcl (4.80-10.80×10 ³ /mcl)
	Neutrophils 81.9% (44.0-70.0%)
	Lymphocytes 12.9% (20.0-45.0%)
	Monocytes 4.4% (2.0-10.0%)
	Eosinophils 0.2% (1.0-4.0%)
	Basophils 0.2% (0.2-1.8 %)
Basic metabolic panel	Sodium 138 mmol/l (136-145 mmol/l)
	Potassium 3.9 mmol/l (3.5-5.1 mmol/l)
	Chloride 101 mmol/l (98-107 mmol/l)
	Calcium 9.4 mg/dl (8.4-10.5 mg/dl)
	Bicarbonate 24 mmol/l (22-29 mmol/l)
	Glucose 125 mg/dl (74-109 mg/dl)
	Creatinine 0.65 mg/dl (0.50-0.90 mg/dl)
	Magnesium 1.70 mg/dl (1.60-2.60 mg/dl)
	Phosphorus 3.3 mg/dl (2.5-4.5 mg/dl)
Lipid panel	Cholesterol 133 mg/dl (≤200 mg/dl)
	LDL cholesterol 75 mg/dl (≤129 mg/dl)
	Triglyceride 98 mg/dl (≤150 mg/dl)
Lactic acid	Lactic acid 1.5 mmol/l (0.5-2.2 mmol/l)
Lipase	Lipase >600 U/l (13-60 U/l)

DISCUSSION

Our patient presented with AP in which the common underlying etiologies such as alcohol misuse, gallstones, hypertriglyceridemia, hypercalcemia, infections, and medications known to induce pancreatitis were all conspicuously absent. The lack of a discernible cause for the pancreatitis prompted our exploration into less conventional pancreatitis triggers.

The phenomenon of post-duodenal polypectomy pancreatitis is exceedingly rare. Only four cases of post-duodenal polypectomy pancreatitis have been reported to date. It was first reported in 1975 by Alper et al, with subsequent reports in 2007, 2009, and 2020.⁶⁻⁹ Similarities between all reported cases, including ours, include: location of the polyps within the second segment of the duodenum, the onset of pancreatitis symptoms within a six-hour timeframe post-duodenal polyp excision, and the absence of any alternative plausible sources of acute pancreatitis. In theory, duodenal polypectomy could lead to pancreatic damage through two mechanisms. Direct mechanical injury in the second section of the duodenum after polypectomy could have led to edema which, due to their proximity to the major and minor duodenal papilla in the same section, could lead to transient obstruction, which could lead to stasis of pancreatic secretions and acinar

damage.¹⁰ Alternatively, transmitted heat to the pancreas during snare polypectomy, which occurs due to the proximity of the pancreas to the second portion of the duodenum, could lead to direct acinar destruction and trypsinogen activation, leading to further pancreatic damage.¹¹ Managing duodenal polypectomy-related pancreatitis aligns with the standard of care for acute pancreatitis.

Supportive care plays a pivotal role, encompassing fluid resuscitation to maintain hemodynamic stability, pain management through analgesics, and initially withholding oral intake, with resumption of feeding once symptoms improve.¹² Out of the four previous cases, only the latter reported by Kwak et al. resulted in mortality, with the other cases fully recovering.⁸

CONCLUSION

In conclusion, acute pancreatitis appears to be a rare and, in some cases, life-threatening complication of duodenal polypectomy. Since all reported cases occurred within 6 hours of polypectomy, clinicians may set a period of observation after polypectomy to allow for early diagnosis of this rare complication. Further studies are needed to determine the exact mechanism and relationship between duodenal polypectomy and acute pancreatitis.

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