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Duodenal Peptic Ulcer Perforation in the Puerperium Case Report Series

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Abstract

Peptic ulcers and complications, such as perforation, are rare during pregnancy and the puerperium. Accordingly, many clinicians may place these diagnoses low on their differential diagnosis. We present two case reports of primigravida, advanced maternal-age females with a history of irritable bowel syndrome and nonsteroidal anti-inflammatory drug use found to have perforated duodenal ulcers after cesarean section. Postpartum surgical abdomens may not present with classic guarding and rigidity. A low threshold for imaging and identification of risk factors is critical to timely diagnosis and management.

Keywords

- peptic ulcer disease
- duodenal ulcer
- peptic ulcer perforation
- puerperium
- postpartum
- surgical abdomen
- NSAIDs
- *Helicobacter pylori*

Introduction

Peptic ulcers and complications, such as perforation and hemorrhage, are much less common in women compared to men. Peptic ulcers are even more rare during pregnancy, which is considered a protective period.[1]

Peptic ulcer disease (PUD) may remit during pregnancy due to gastric hypochlorhydria and increased gastric mucus production in the setting of increased plasma histaminase, estrogen, progesterone, and epidermal growth factor levels. Pregnant patients may also experience immunologic tolerance of *Helicobacter pylori*, avoidance of ulcerogenic factors, and improved nutrition.[2] Given the rarity of peptic ulcers and perforation in the puerperium, many clinicians may place these conditions low on their differential diagnosis.

The current literature on PUD in pregnancy suffers from outdated studies written before the etiologic identification of *H. pylori*, poorly controlled trials, and few endoscopically proven peptic ulcers.[2] This paper aims to synthesize the current literature in the context of two case reports to identify trends in clinical presentation, diagnosis, and management.

Cases

Patient A

A 38-year-old G1P0 female with a history of irritable bowel syndrome, remote *Clostridioides difficile* infection (in the setting of chronic antibiotic use for Lyme disease), in vitro fertilization pregnancy, Factor V Leiden heterozygosity, hyperlipidemia (previously on atorvastatin), anxiety/depression, and obstructive sleep apnea presented with nausea, emesis, and anorexia to the Emergency Department (ED) of a large urban academic hospital 7 days after cesarean section. She had received routine

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prenatal care, and her pregnancy was complicated in the third trimester by SARS-CoV-2 infection treated with nirmatrelvir/ritonavir, severe polyhydramnios, succenturiate placenta, and velamentous cord insertion. She took low-dose prophylactic aspirin throughout pregnancy. She was induced for polyhydramnios at 39 weeks and 6 days with misoprostol and oxytocin but ultimately underwent an uncomplicated cesarean section at 40 weeks and 1 day after meeting the criteria for arrest of dilation. The patient was discharged on postoperative day (POD) 3 on enoxaparin and as-needed pain management with acetaminophen and ibuprofen.

On POD 5, the patient reported nausea, one episode of bilious emesis, and anorexia. She was prescribed ondansetron without improvement. On POD 7, the patient presented to the ED with persistent symptoms. She denied abdominal pain and endorsed flatus. Her current medications included prophylactic postpartum enoxaparin, acetaminophen, and ibuprofen. On exam, the mucosa appeared dry, and her surgical incision was clean, dry, and intact. Her vital signs included a body temperature of 100.2°F, tachycardia to 120 beats per minute, and normotension. Her labs were notable for hypoglycemia to 60 mg/dL (for which she received a dextrose solution bolus), with complete blood cell counts, basic metabolic panel, liver and pancreatic function tests, as well as coagulation profile grossly within normal limits.

While in the ED, the patient reported the acute onset of new severe chest and abdominal pain, with diffuse tenderness to palpation. No guarding or rebound were noted. A bedside sonogram revealed possible abdominal fluid. The patient was sent for a computed tomography (CT) of the abdomen and pelvis with oral and intravenous (IV) contrast, which appeared consistent with a duodenal ulcer perforation (►**Fig. 1**). The patient received piperacillin-tazobactam, fluconazole, and a proton pump inhibitor (PPI) before proceeding to emergency diagnostic laparoscopy.

The procedure was converted to open laparotomy upon discovery of a 3 cm perforated ulcer in the first portion of the duodenum, near the head of the pancreas, with concern for

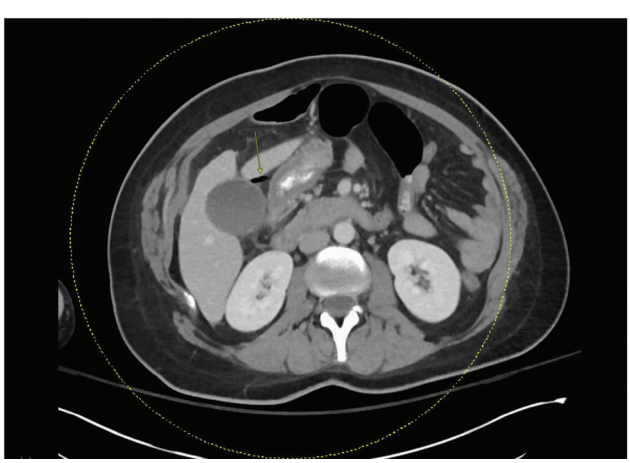

Fig. 1 Patient A's axial CT of the abdomen and pelvis demonstrates thickening of the duodenal bulb, a focus of free air adjacent to the postbulbar duodenum (yellow arrow), extraluminal contrast, as well as stranding and small fluid in the right upper quadrant, consistent with duodenal ulcer perforation. CT, computed tomography.

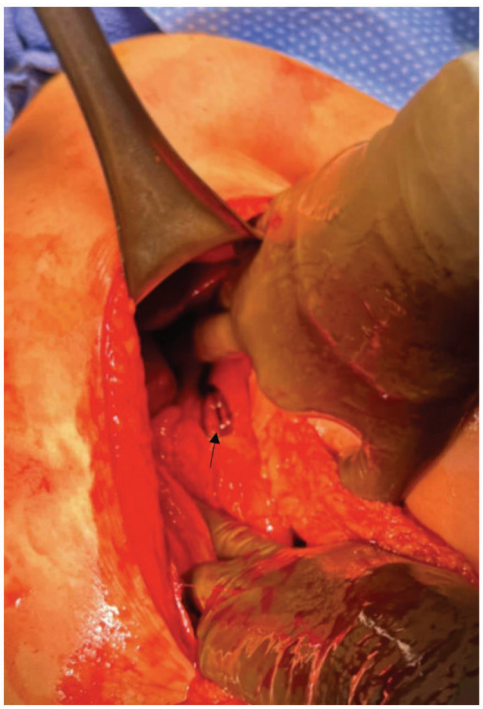

Fig. 2 Intraoperative gross clinical photo of patient A's 3 cm duodenal ulcer perforation (black arrow).

the integrity of the ampulla (►**Fig. 2**). An antrectomy with gastrojejunostomy and cholecystectomy was performed.

The patient's postoperative course was uncomplicated. She received pain control, antimicrobials, PPI, and antiemetics. Her diet was slowly advanced as tolerated. Stool testing and immunohistochemical stain of the operative specimen for *H. pylori* were both negative. The patient was discharged home on POD 7.

Patient B

A 41-year-old G1P0 female with a history of irritable bowel syndrome presented to the ED with intense abdominal pain 33 days after cesarean section. She had received routine prenatal care and took low-dose prophylactic aspirin throughout pregnancy. Her pregnancy was complicated by gestational hypertension (not on medication), diagnosed at 34 weeks, for which she was induced at 37 weeks and 1 day. She ultimately underwent an uncomplicated cesarean section after 1 day of induction due to fetal intolerance of labor (persistent fetal tachycardia with minimal variability despite resuscitative measures).

The patient's postoperative course was complicated by preeclampsia with severe features (by blood pressure) on POD 4. She received treatment with nifedipine and labetalol, as well as seizure prophylaxis with magnesium sulfate. The patient's postoperative course was also complicated by fever of unclear etiology in the immediate postpartum period (she received empiric treatment for intrauterine infection with piperacillin-tazobactam) and again on POD 6, when she tested positive for *C. difficile*.

The patient was discharged on POD 6 on the same inpatient antihypertensive regimen, as well as fidaxomicin, enoxaparin,

and as-needed pain management with acetaminophen and ibuprofen. She was normotensive with no complaints when followed up in the clinic on POD 9. On POD 23, the patient presented to the ED with facial cellulitis. She reported taking up to 3,200 mg of ibuprofen orally daily at home for pain control. She was treated with amoxicillin/clavulanate as well as oral vancomycin for *C. difficile* prophylaxis.

On POD 33, the patient presented to the ED again, now with sudden, severe abdominal pain. On exam, she appeared in acute distress with abdominal distension, tenderness to palpation, guarding, and rebound. Her vital signs were stable and within normal limits. Her labs were notable for leukocytosis (27 K/uL), with other blood cell counts, basic metabolic panel, and liver and pancreatic function tests grossly within normal limits. The patient underwent CT of the abdomen and pelvis with oral and IV contrast, which appeared consistent with a duodenal ulcer perforation (►**Fig. 3**). The patient was started on cefepime, metronidazole, and a PPI while her home oral medications were held. A nasogastric tube was placed. The patient underwent diagnostic laparoscopy, which identified a small duodenal perforation (►**Fig. 4**). A modified graham patch repair of the perforation was performed laparoscopically using omentum.

The patient's postoperative course was uncomplicated. She received pain control, antimicrobials, PPI, and antiemetics. Her diet was slowly advanced as tolerated. Stool testing for *H. pylori* was negative (no intraoperative specimen for immunohistochemical staining was taken since a resection was not indicated and a biopsy would have unnecessarily enlarged the small ulcer site). The patient was discharged home on POD 5.

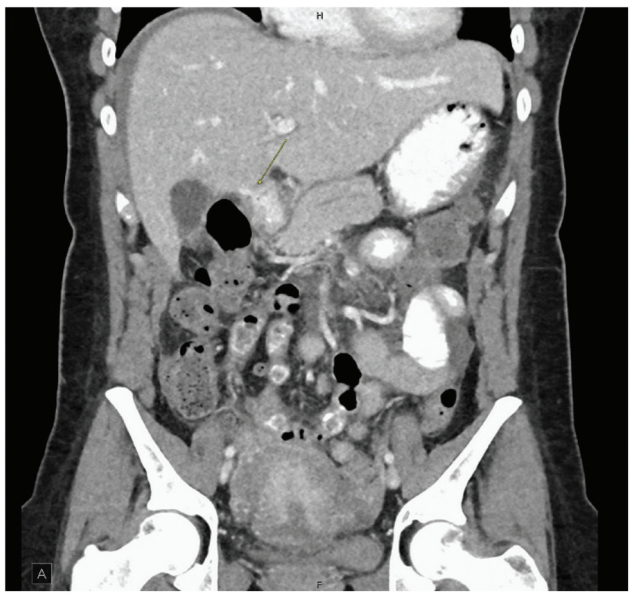

Fig. 3 Patient B's coronal CT of the abdomen and pelvis demonstrating small volume pneumoperitoneum and abdominopelvic ascites with focal extravasation of oral contrast at the duodenal bulb (yellow arrow) and layering adjacent to the liver in the porta hepatis, consistent with duodenal ulcer perforation. CT, computed tomography.

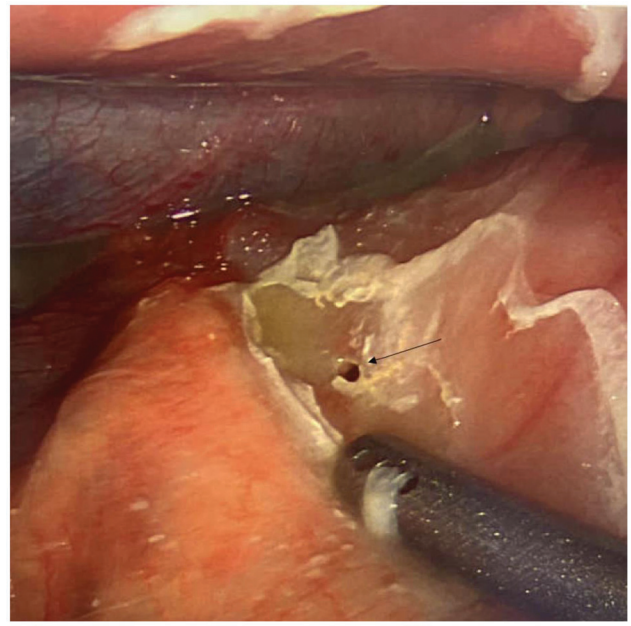

Fig. 4 Intraoperative gross clinical photo of patient B's small duodenal ulcer perforation (black arrow).

Discussion

Diagnosis of PUD and perforation may be delayed or missed in the puerperium, given the significant overlap of PUD symptoms and the physiologic postpartum course. Both providers and patients may be more inclined to attribute pathologic symptoms to normal healing and, therefore, delay presentation to care, as exemplified by patient A originally presenting to care 2 days prior to perforation.[3] PUD classically presents with epigastric pain, anorexia, nausea, vomiting, and abdominal distension. Perforation typically presents with acute worsening of severe abdominal pain, nausea, vomiting, rigidity, rebound, guarding, hypoactive bowel sounds, hypotension, and leukocytosis.[2] These symptoms may be masked or present differently in pregnant patients. Perhaps most notably, postpartum surgical abdomens may not present with guarding, rebound, and rigidity due to laxity and thinning of the abdominal muscles.[3–7] Patient A did not present with a classic surgical abdomen on exam, while patient B presented with guarding and rebound.

Given the rarity of peptic ulcers and their potentially non-classic presentation in the puerperium, a high clinical suspicion is necessary for timely diagnosis. First, a thorough history is critical. While a past medical history of known PUD is an obvious clue, patients with a history of any chronic gastrointestinal complaints should raise concerns. In both of our case reports and at least one other published case, the patient had an existing diagnosis of irritable bowel syndrome prior to pregnancy.[6] Especially if these symptoms remit during pregnancy, it is possible that the patients had mis- or undiagnosed PUD prior to conception. Interestingly, at least one other case of *H. pylori*-negative peptic ulcer perforation in the postpartum period has been documented.[8]

Another important risk factor for PUD is the use of nonsteroidal anti-inflammatory drugs (NSAIDs), which results in an estimated ulcer incidence of 2 to 4% annually.[2] In at least 5 cases of perforation, including ours, patients had a recent history of NSAID use.[4,5,9,10] In the era of enhanced recovery after cesarean protocols, multimodal analgesic regimens reduce opioid use but may inadvertently expose patients to higher doses or prolonged courses of NSAIDs.[11] Avoidance of ibuprofen in favor of pain control medications with fewer gastrointestinal side effects like celecoxib or acetaminophen may be reasonable in pregnant patients with a history of gastrointestinal issues.[9] Other risk factors for peptic ulcers include genetic predisposition, smoking, alcohol use, advanced age, and increased parity.[6]

Both patients were also prescribed prophylactic low-dose aspirin in the antepartum. While several clinical guidelines recommend the use of PPIs in patients taking low-dose aspirin to reduce the risks of adverse gastrointestinal and cardiovascular events, the overall evidence is rather weak.[12] It is possible that the antepartum and postpartum use of NSAIDs may increase the risk of PUD, so it may be reasonable to co-prescribe PPIs with prophylactic low-dose aspirin in patients with the aforementioned risk factors.

In addition to a thorough history, the workup of puerperal patients presenting with symptoms concerning for PUD and perforation should include labs for complete blood counts, electrolytes, liver function, amylase, and lipase. A low threshold should be maintained for bedside ultrasound, abdominal X-ray, and/or CT of the abdomen and pelvis, which were critical in the rapid diagnoses of our patients.[2,9,13,14] Once perforation is suspected, rapid stabilization with electrolyte correction, IV fluids, non-NSAID pain control, and broad-spectrum antibiotics should be performed before emergent surgical repair.[2]

Using case reports to build an illness script for PUD in pregnancy may help raise clinical suspicion for this diagnosis. Given the high maternal mortality rates associated with delay in diagnosis, these patients must be promptly identified and appropriately managed.[2] While clinicians may be inclined to attribute a patient's symptoms to cesarean section recovery when peritoneal signs are not impressive, we must use the body of reports presented here as a reminder to trust a patient's pain narrative and escalate workup accordingly.

Conflict of Interest
None declared.

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