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BENIGN DUODENOCOLIC FISTULA: A RARE COMPLICATION OF PEPTIC ULCER DISEASE

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ABSTRACT

Background: Duodenocolic fistulas are uncommon but serious complications of peptic ulcer disease (PUD), resulting from the erosion of a duodenal ulcer into the colon. This connection typically forms when a chronic ulcer perforates or penetrates through the duodenum, leading to inflammation and necrosis that disrupts the normal anatomical barriers.

Case Report: A 66-year-old male with a history of inadequate medical follow-up, chronic nonsteroidal anti-inflammatory drug use, alcohol use disorder, and PUD, developed a duodenocolic fistula secondary to a perforated duodenal ulcer. Endoscopy revealed a large duodenal ulcer with fistulisation into the colon. The patient underwent definitive surgical treatment, including fistula takedown, partial colectomy, antrectomy, and gastrojejunostomy.

Conclusion: This case highlights the importance of early recognition and thorough evaluation of duodenocolic fistulas, which can lead to severe complications such as sepsis, malnutrition, and gastrointestinal bleeding. While conservative management may be appropriate in select cases, surgical intervention remains the definitive treatment for symptomatic or complicated fistulas. Prevention through targeted interventions, such as effective management of PUD and routine screening for high-risk conditions, is crucial. A multidisciplinary approach is essential for accurate diagnosis and individualized treatment of duodenocolic fistulas.

KEYWORDS

Peptic ulcer disease, PUD, benign duodenocolic fistula, enterocolic fistula

LEARNING POINTS

- Duodenocolic fistulas are a rare yet serious complication of peptic ulcer disease. Timely diagnosis and management of duodenocolic fistulas is vital to prevent complications including gastrointestinal bleeding, sepsis, and malnutrition.
- Effective treatment of duodenocolic fistulas requires a multidisciplinary approach. While some cases may be managed conservatively, surgical intervention remains the definitive treatment for complicated or symptomatic fistulas.
- Preventing the development of duodenocolic fistulas involves proactive management of the underlying disease processes.

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INTRODUCTION/BACKGROUND

Peptic ulcer disease (PUD) is a prevalent gastrointestinal disorder characterized by mucosal erosion extending into the muscularis mucosa. Peptic ulcer disease has an estimated prevalence of 8.4% in the United States[1]. Epidemiological data indicate a higher incidence among males, individuals who smoke, and those with chronic comorbidities[1]. Advancing age has also been recognized as an independent risk factor. The primary aetiologies of PUD are *Helicobacter pylori* infection and the use of non-steroidal anti-inflammatory drugs (NSAIDs)[1]. Additional risk factors include smoking, alcohol consumption, chronic corticosteroid use, or Zollinger-Ellison syndrome to name a few[1].

PUD can manifest as gastric or duodenal ulcers, with duodenal ulcers being more common. Characteristic symptoms of PUD include epigastric pain, bloating, nausea, loss of appetite, and weight loss[1]. If left untreated, PUD can result in serious complications, including gastrointestinal bleeding, perforation, penetration, and gastric outlet obstruction[1]. In particularly rare cases, perforation of duodenal ulcers may lead to duodenocolic fistulas. These fistulas arise from the underlying ulcerative process, which starts with localized tissue damage and inflammation, eventually advancing to mucosal erosion and extension into the adjacent colon.

In this article, we describe our experience managing a case of duodenocolic fistula in a 66-year-old male with a known history of peptic ulcer disease along with a review of the literature.

CASE DESCRIPTION

A 66-year-old male with a history of longstanding inadequate medical follow-up, chronic NSAID use, alcohol use disorder, and PUD, presented with a 3–4-month history of intermittent episodes of "black" emesis with undigested food, worsening in the last few days prior to admission associated with generalized weakness and light headedness. He reported a history of chronic diarrhoea with intermittent dark stools and an unintentional weight loss of at least 40-pounds in the past 6 months. He had not undergone any prior endoscopic evaluations or medical follow-ups in over 25 years.

The patient had been chronically taking 800 mg of ibuprofen every 4 hours for back pain. Otherwise, the patient took no other medications. His alcohol consumption consisted of 1-2 cans of beer per day, which was decreased from his previous consumption of a 12-pack beer per day for many years.

Initial laboratory testing was significant for haemoglobin of 5.5 g/dl and an elevated international normalised ratio of 6.0, although the patient was reportedly not taking any anticoagulation medications. He received 2 units of emergency release packed red blood cells, 2 units of fresh frozen plasma and was administered 10 mg of Vitamin K. A computed tomography (CT) scan of the abdomen/pelvis revealed a large contained duodenal ulcer with fistulisation into the hepatic flexure of the colon *(Fig. 1)*. Esophagogastroduodenoscopy revealed a deformed antrum, a large 2 cm cratered ulcer with a necrotic base at the duodenal bulb without active bleeding, and a fistula connecting to the colon with brown faecal matter layering the colonic mucosa *(Fig. 2)*. A biopsy of the gastric antrum revealed chronic inactive gastritis, negative for *H. pylori*, and no evidence of dysplasia. Biopsy of the fistula revealed fragments of benign enterocolic tissue with inflammatory cell infiltrates but no granuloma, dysplasia or malignancy.

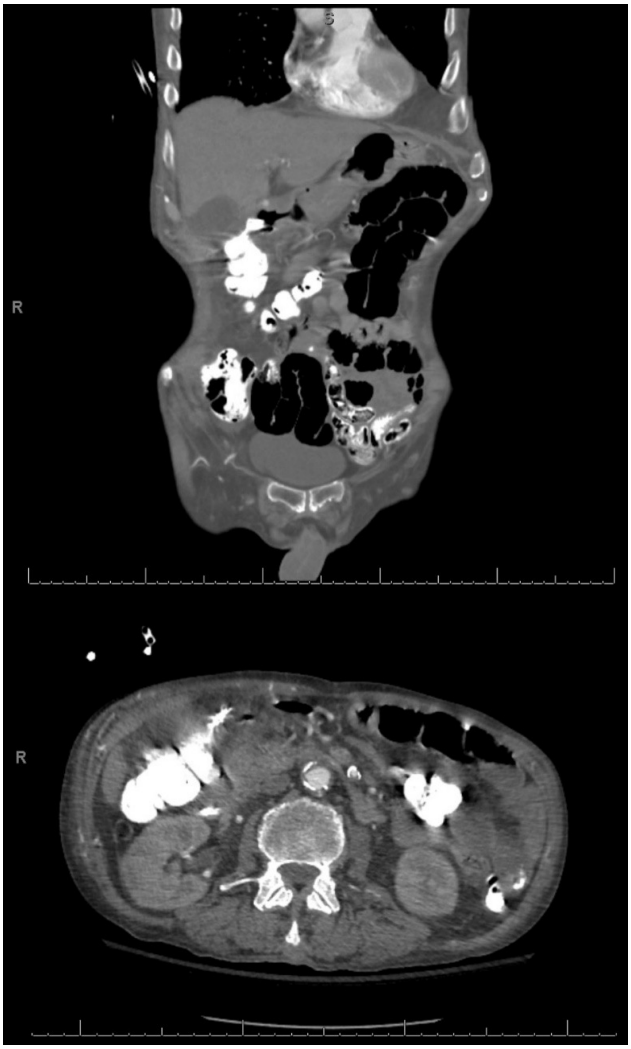


Figure 1. A computed tomography scan of the abdomen/pelvis revealed an approximately 7 × 2 cm large contained duodenal ulcer with evidence of fistulization between the ulcer and hepatic flexure of the colon with surrounding inflammatory fat stranding, fluid, and prominent lymph nodes. The fistula was demonstrated in both the coronal (top) and axial (bottom) planes.

After a detailed discussion at a multidisciplinary team meeting, it was concluded that the fistula was most likely caused by a perforated duodenal ulcer, and the decision was made to proceed with surgery. Surgical exploration revealed a significantly inflamed and friable duodenocolic fistula from the first part of the duodenum (D1) to just distal to the hepatic flexure. Accordingly, the patient underwent takedown of colic-duodenal fistula with partial colectomy and antrectomy with gastrojejunostomy. Final pathology revealed an ulcerated coloenteric fistula in the fistula



Figure 2. Esophagogastroduodenoscopy revealed 2 cm cratered ulcer with a necrotic base, at the duodenal bulb. There was no evidence of active bleeding. Inferior to this ulcer was a fistula connecting the ulcer to the colon with overlaying brown stool. Cold forceps biopsy was taken from the edge of the ulcer to rule out malignancy and dysplasia.

takedown specimen and mild chronic gastritis with intestinal metaplasia.

DISCUSSION

Duodenocolic fistulas are abnormal communications between the duodenum and the colon, with malignancy, most commonly colonic adenocarcinoma, being the leading cause[2]. Amongst the benign causes, PUD and Crohn's disease are the most prevalent, with more unusual cases including complications of prior gastric surgery, ingested foreign bodies, diverticula, cholelithiasis, pancreatitis, ruptured appendicitis, and regional enteritis[3].

In this case, long-standing untreated PUD led to fistula formation, highlighting the critical need for early diagnosis and intervention in high-risk patients. Preventive care plays a pivotal role in reducing the incidence of such complications. Increased screening for colon cancers has significantly decreased the incidence of malignant tumours[4] while the widespread use of proton pump inhibitors (PPI) and *H. pylori* eradication therapy has significantly decreased the occurrence of peptic ulcers[3]. However, patients with chronic NSAIDs use, alcohol use disorder, and inadequate medical follow-up—like our patient—remain particularly vulnerable.

In our case, the patient's progressive symptoms of epigastric pain, hematemesis, weight loss, and chronic diarrhoea should have prompted medical evaluation much earlier. These symptoms are typical of many of the underlying causes of duodenocolic fistulas, but do not always represent progression to a fistula[5]. More specific symptoms of duodenocolic fistulas include defecation of undigested food due to gastric contents bypassing the small intestines, and feculent vomiting due to retrograde movement of colonic material back into the duodenum[6], which were not reported with our patient.

Diagnosis of a duodenocolic fistula typically involves both radiographic imaging and endoscopic evaluation[7]. The most sensitive imaging modality for the diagnosis of duodenocolic fistulas is a barium enema, which would demonstrate a direct flow of contrast from the colon to the duodenum through the fistula. A barium meal can occasionally also demonstrate the fistulous tract; however, it is less sensitive than a barium enema, likely due to the relatively increased pressure inside the colon compared to the duodenum which limits anterograde passage of gastric contents in most cases[2]. An abdominal CT scan with or without contrast will often show the fistulous tract and can also be helpful in distinguishing the cause of the fistula as it may show the presence of masses, strictures, abscesses, migrated gallstones, or inflammation within the gastrointestinal tract[8]. While endoscopy is less sensitive for the initial detection of a fistula[9], it remains an important step of the workup as it allows for direct visualization and biopsy of the surrounding tissue[7].

Once a duodenocolic fistula develops, management requires a multidisciplinary approach involving gastroenterologists, radiologists, and surgeons. While conservative management with nutritional support and somatostatin analogues may be considered in select cases, surgical intervention remains the definitive treatment for most patients[10]. The choice of surgical approach depends on the extent of tissue damage and patient stability. Our patient required a fistula takedown, partial colectomy, antrectomy, and gastrojejunostomy, which is a high-risk but necessary procedure in such advanced cases. The prognosis for a duodenocolic fistula resulting from PUD is generally good with proper surgical intervention, especially when diagnosed and treated early; however, the outlook can be guarded due to potential complications like malnutrition, severe diarrhoea, and electrolyte imbalances, which can significantly impact quality of life if not managed effectively[11]. Long-term follow-up is crucial to ensure adequate nutritional support, monitor for recurrent ulceration, and address postoperative complications that may arise[9].

Physicians should maintain a high index of suspicion for

fistulisation in patients with chronic ulcer symptoms, weight loss, vomiting, and diarrhoea. Moreover, preventive strategies—including judicious NSAIDs use, appropriate H. pylori screening, and early PPI therapy—are essential in mitigating complications. In patients with limited access to healthcare, efforts should be made to improve medical follow-up and patient education to prevent the unchecked progression of PUD into life-threatening conditions.

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