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CASE REPORT

Phlegmonous gastritis after biloma drainage: A case report and review of the literature

Kai-Chun Yang, Hsin-Yu Kuo, Jui-Wen Kang

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Abstract

BACKGROUND

Phlegmonous gastritis (PG) is a rare bacterial infection of the gastric submucosa and is related to septicemia, direct gastric mucosal injury, or the direct influence of infection or inflammation in neighboring organs. Here, we present a patient who had spontaneous biloma caused by choledocholithiasis and then PG resulting from bile leakage after biloma drainage.

CASE SUMMARY

A 79-year-old man with a medical history of hypertension had persistent diffuse abdominal pain for 4 d. Physical examination showed stable vital signs, icteric sclera, diffuse abdominal tenderness, and muscle guarding. Laboratory tests showed hyperbilirubinemia and bandemia. Contrast computed tomography (CT) of the abdomen showed a dilated common bile duct and left subphrenic abscess. Left subphrenic abscess drainage revealed bilious fluid, and infected biloma was confirmed. Repeated abdominal CT for persistent epigastralgia after drainage showed gastric wall thickening. Esophagogastroduodenoscopy (EGD) showed an edematous, hyperemic gastric mucosa with poor distensibility. The gastric mucosal culture yielded *Enterococcus faecalis*. PG was diagnosed based on imaging, EGD findings, and gastric mucosal culture. The patient recovered successfully with antibiotic treatment.

CONCLUSION

PG should be considered in patients with intraabdominal infection, especially from infected organs adjacent to the stomach.

Key Words: Phlegmonous gastritis; Epigastric pain; Choledocholithiasis; Bile leakage; Antibiotics; Case report

Core Tip: We report a case of spontaneous biloma caused by choledocholithiasis followed by phlegmonous gastritis (PG) resulting from biloma rupture after biloma drainage. Additionally, we analyzed 44 PG cases reported from 2012 to 2022. The etiology of PG is mainly direct microbial invasion from gastric mucosa injury or hematogenous/lymphogenous spread and the most important risk factor for PG is an immunocompromised state. In our case, the patient was immunocompetent and PG was caused by bile leakage after biloma drainage rather than the direct influence of infected biloma.

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INTRODUCTION

Phlegmonous gastritis (PG) is a rare bacterial infection of the gastric submucosa and is related to septicemia, direct gastric mucosal injury, or direct influence of infection or inflammation in neighboring organs. It is fatal if not diagnosed and treated immediately. An immunocompromised state associated with malignancy, chemotherapy-induced neutropenia, acquired immunodeficiency syndrome, alcoholism, and immunosuppressive drugs is considered the main risk factor[1,2].

Here, we present a patient with spontaneous biloma caused by choledocholithiasis followed by PG induced by bile leakage after biloma drainage.

CASE PRESENTATION

Chief complaints

A 79-year-old male complained of persistent diffuse abdominal pain for 4 d.

History of present illness

Initially, the abdominal pain occurred abruptly after eating a big meal, 4 d prior to admission. The initial abdominal pain was mainly located in the right upper quarter abdominal area and then migrated to the whole abdomen. Additionally, the patient experienced nausea, vomiting, constipation, and fever. Recurrent abdominal pain was noted for the 4 d as well. Sonography-guided percutaneous catheter drainage of the left subphrenic abscess, as shown by contrast computed tomography (CT) of the abdomen, was performed. The bilirubin level was 76.0 mg/dL and volume of abscess drainage was around 600 mL. The abscess culture yielded *Enterococcus faecalis* and *Enterobacter cloacae* complex. The blood culture yielded no pathogen isolates. Biloma was confirmed. However, the patient still complained of epigastric pain after drainage.

History of past illness

The patient had a medical history of hypertension and had taken an antihypertensive drug regularly.

Personal and family history

The patient's personal and family histories were unremarkable.

Physical examination

Initial vital signs were a temperature of 37.8 °C, heart rate of 126 beats/min, blood pressure of 163/93 mmHg, and respiratory rate of 32 breaths/min. There was no apparent loss of consciousness. Physical examination showed icteric sclera, abdominal fullness, diffuse tenderness, and muscle guarding. Follow-up vital signs before repeat abdominal CT for persistent abdominal pain were a temperature of 37.5 °C, heart rate of 116 beats/min, blood pressure of 162/84 mmHg, and respiratory rate of 20 breaths/min. Severe muscle guarding and diffuse tenderness were observed.

Laboratory examinations

Abnormal laboratory findings included hyperbilirubinemia (total bilirubin: 3.0 mg/dL; reference range: ≤ 1.2 mg/dL), mildly elevated alkaline phosphatase (138 U/L; reference range: 40-129 U/L), hyponatremia (sodium: 127 mmol/L; reference range: 136-145 mmol/L), impaired renal function (creatinine: 1.59 mg/dL; reference range: 0.70-1.20 mg/dL), white blood cell count of 3200/μL (reference range: 3400-9500/μL), and 8% band form of white blood cells (reference range: 0.0%-4.2%).

Imaging examinations

Plain abdominal radiography showed ileus and contrast abdominal CT showed a dilated common bile duct (CBD) and left subphrenic abscess (Figure 1A). Repeat contrast CT of the abdomen was performed for persistent abdominal pain after biloma drainage and showed bile leakage and gastric wall thickening (Figure 1B). Esophagogastroduodenoscopy (EGD) showed an edematous, hyperemic gastric mucosa with poor distensibility (Figure 2). Endoscopic retrograde cholangiopancreatography after EGD showed a dilated CBD with one filling defect of about 10 mm in size (Figure 3A).

Further diagnostic work-up

The gastric mucosal culture yielded *Enterococcus faecalis* and the biopsy showed that the gastric submucosa and mucosa were infiltrated by clusters of lymphocytes, neutrophils, and plasma cells (Figure 4). According to initial CT and endoscopic retrograde cholangiopancreatography, the etiology of the initial abdominal pain with fever was a CBD stone with cholangitis and spontaneous biloma. However, according to serial CT images and gastric mucosal culture, the persistent pain after biloma drainage was caused by PG. The etiology of PG was bile leakage after biloma drainage.

FINAL DIAGNOSIS

PG resulting from bile leakage after biloma drainage.

TREATMENT

The patient underwent conservative therapy for PG including parenteral nutrition, biloma drainage, an initial broad-spectrum antibiotic (cefepime 2 g, twice daily), and then a definitive antibiotic of ampicillin 2 g, 6 times a day for 10 d. Endoscopic retrograde biliary drainage was performed for internal biloma drainage (Figure 3B). Neither endoscopic sphincterotomy nor stone extraction was performed due to coagulopathy.

OUTCOME AND FOLLOW-UP

The abdominal pain was relieved after antibiotic treatment, and follow-up EGD 1 mo later showed a normal gastric mucosa with improved distensibility of the stomach (Figure 5). Follow-up abdominal CT 2 mo later showed that the biloma was almost resolved, and the biloma drainage was removed before discharge. The patient is currently being followed as an outpatient.

DISCUSSION

PG is an infrequent bacterial infection of the gastric submucosa. It was first described in 1862 by Cruveilhier[3] and an average of one case report *per* year has appeared over the last 60 years[4]. We searched for papers indexed in the PubMed database using the keyword "phlegmonous gastritis". In recent decades, from 2012 to 2022, 44 cases of PG have been reported in the English-language literature, which are summarized in Table 1[1,4-42]. Our literature review showed that PG affects all age groups (age range: 7 to 89 years old) and is highly common in the decades of 40s to 70s, with a male-to-female ratio of about 2:1. According to our findings of the literature review, half of the cases (22/44) were in an immunocompromised state, such as alcoholism, diabetes mellitus, acquired immunodeficiency syndrome, chronic hepatitis B, neutropenia after chemotherapy, and treatment with immunosuppressant drugs. Thus, an immunocompromised state appears to be a main risk factor for PG. Other risk factors for PG are prior endoscopic procedure (including for mucosal resection, submucosal dissection, hemostasis, ultrasonography with fine needle aspiration, and mucosal biopsy; 5/44), recent upper airway infection (3/44), malignancy (8/44), and prior gastrectomy or esophagectomy (3/44).

PG type is classified as diffuse or localized, according to the lesion range[4]. The diffuse type involves the complete stomach, and represents most cases[43]. In contrast, the localized type is most commonly restricted to the antrum, with rare cases involving the cardia or pylorus. Gastric wall abscess is a localized form of PG[43]. In our literature view, only 6 localized type PG cases were identified. The etiology of PG can be classified into primary, secondary, or idiopathic[17]. Primary PG represents a direct microbial invasion from gastric mucosa injury, which is caused by trauma, malignancy, peptic ulcer, or endoscopic interventions. Secondary PG represents a hematogenous/lymphogenous spread or direct influence of infection or inflammation in neighboring organs such as infection due to upper airway infection, pancreatitis, or cholecystitis. Idiopathic PG represents an unknown cause with absence

Table 1 Reported cases of phlegmonous gastritis between 2012 and 2022

Ref.	Year of publication	Age in yr	Sex	Risk factors	Type	Symptom	Diagnosis	Microorganism	Treatment	Result
Saito *et al* [5]	2012	55	F	ALL	Diffuse	Septic shock	CT + EGD	Bacillus species	ATB	Discharge
Itonaga *et al*[6]	2012	70	F	EUS-FNA	Diffuse	EP	CT + EGD	*Streptococcus* spp.	ATB	Discharge
Fan *et al*[7]	2013	65	M	Splenectomy + esophagectomy	Diffuse	EP, fever	CT + EGD	*Staphylococcus aureus*	ATB	Discharge
Liu *et al*[8]	2013	84	M	Nil	Diffuse	EP	CT + EGD	Nil	Gastrectomy	Discharge
Yu *et al*[9]	2013	52	M	Liver cirrhosis, HBV, hepatectomy	Diffuse	EP, palpitation, dyspnea	EL + intraoperative EGD	*Klebsiella pneumoniae*	ATB	Discharge
Nair *et al* [10]	2013	72	M	Nil	Diffuse	EP, fever, N/V	CT + EGD	Nil	ATB	Discharge
Alonso *et al* [11]	2013	55	M	Nil	Localized	EP, fever, N/V, diarrhea	CT	*Streptococcus pyogenes*	ATB, endoscopic drainage	Discharge
Sahnan *et al* [12]	2013	56	F	GAVE s/p APC	Diffuse	EP, palpitation	EL	Streptococcus	Total gastrectomy	Death
Cortes-Barenque *et al*[13]	2014	35	M	Nil	Diffuse	EP, melena, hematemesis	CT + EGD	Group A streptococcus	ATB	Discharge
Rada-Palomino *et al*[14]	2014	62	M	HIV	Diffuse	EP, N/V, hematemesis, diarrhea	CT + EGD	*Streptococcus pyogenes*	ATB	Discharge
Min *et al* [15]	2014	51	F	Nil	Diffuse	EP, vomiting, palpitation	CT + EL	*Streptococcus pyogenes*	Total gastrectomy + ATB	Discharge
Morimoto *et al*[16]	2014	77	M	DM, GU	Diffuse	N/V, palpitation	CT	Group A streptococcus	ATB	Death
Nomura *et al*[17]	2015	80	F	SMA syndrome	Diffuse	EP, N/V	CT + EGD	*Enterococcus faecium*	Total gastrectomy + ATB	Discharge
Flor-de-Lima *et al* [18]	2015	7	M	Acute tonsillitis	Diffuse	EP, N/V	CT + EGD	*Streptococcus pneumoniae*	ATB	Discharge
Kato *et al* [19]	2015	64	M	Chronic pancreatitis, DM, subtotal gastrectomy	Diffuse	EP, N/V	CT + EGD	*Peptostreptococcus* spp.	ATB	Discharge
Matsumoto *et al*[20]	2015	74	M	MF, MM	Diffuse	EP, N/V	CT + EGD	*Bacillus thuringiensis*	ATB	Death
Kim *et al* [21]	2016	74	M	DM, alcoholic liver cirrhosis, HCC, GC	Diffuse	EP, N/V, palpitation	CT + EGD	Nil	ATB	Discharge
Kim *et al* [22]	2017	51	M	AS s/p infliximab	Diffuse	N/V	CT + EGD	Nil	ATB	Discharge
Hagiwara *et al*[23]	2018	65	M	ESCC	Localized	EP, fever	CT + EGD	*Streptococcus viridans*	ATB + total gastrectomy	Discharge
Ishioka *et al* [24]	2018	84	F	Dementia	Diffuse	Hematemesis	CT + EGD	*Proteus mirabilis*, α-Streptococcus	ATB	Discharge
Ishioka *et al* [24]	2018	44	M	DM	Diffuse	EP	CT + EGD	Staphylococci	ATB	Discharge
Ishioka *et al* [24]	2018	64	M	Brain tumor s/p chemotherapy	Diffuse	N/V, hematemesis	CT + EGD	Nil	ATB	Death

De Davide and Beaudoin [25]	2018	42	M	PA s/p infliximab	Diffuse	EP, N/V, fever	CT + EGD	Nil	ATB	Discharge
Yang *et al* [4]	2018	47	M	URI, alcoholism, GU	Diffuse	EP, N/V, fever	CT + EL	Group A strepto-coccus	Total gastrectomy + ATB	Discharge
Ramphal *et al*[26]	2018	45	M	Nil	Diffuse	EP, N/V, palpitation	CT + EL	Group A Strepto-coccus	Total gastrectomy + ATB	Discharge
Iqbal *et al* [1]	2018	56	F	AML	Diffuse	EP, fever	CT + EGD	*Citrobacter freundii, Enterococcus faecalis, Bacillus cereus*	ATB	Discharge
Matsuura *et al*[27]	2018	76	F	MDS, DM, GC s/p ESD	Diffuse	EP, fever	CT + EGD	*Klebsiella pneumoniae, Pseudomonas aeruginosa*	ATB	Discharge
Saeed *et al* [28]	2019	59	M	Morbid obesity s/p laparo-scopic sleeve gastrectomy	Localized	EP, N/V, fatigue, chills	CT	*Streptococcus sanguinis*	ATB + CT-guided drainage	Discharge
Shi *et al*[29]	2019	33	M	ALL s/p chemotherapy	Diffuse	EP, hematemesis	CT	*Stenotrophomonas maltophilia*	ATB	Discharge
Yasuda *et al* [30]	2020	74	F	Had eaten raw Ayu fish	Localized	EP, N/V, diarrhea	CT + EGD	*Aeromonas hydrophila*	ATB	Discharge
Campos-Murguía *et al*[31]	2019	37	F	MG, thymoma s/p resection	Diffuse	EP, N/V, melena	CT + EGD	*Streptococcus oralis*	ATB + total gastrectomy	Discharge
Kuriyama *et al*[32]	2020	70	F	Gastric DLBCL s/p chemotherapy	Diffuse	EP, N/V, fever	CT + EGD	*Pseudomonasaeruginosa*	ATB	Discharge
Yakami *et al*[33]	2021	32	M	Alcoholism	Diffuse	EP, N/V, fever	CT + EGD	Nil	ATB	Discharge
Yakami *et al*[33]	2021	33	M	Alcoholism	Localized	EP	CT + EGD	*Streptococcus viridans*	ATB	Discharge
Yakami *et al*[33]	2021	19	M	Nil	Localized	EP, N/V, fever	CT + EGD	*Pseudomonas aeruginosa, Strepto-coccus viridans*	ATB	Discharge
Taniguchi *et al*[34]	2021	21	M	URI	Diffuse	EP	CT + EGD	*Streptococcus constellatus/milleri*	ATB	Discharge
DeCino *et al*[35]	2021	47	M	DM	Diffuse	EP, N/V, fever	CT + EGD + EUS	Group A strepto-coccus	ATB	Discharge
Elisabeth *et al*[36]	2021	70	F	Nil	Diffuse	EP, N/V, fever, diarrhea	CT + EGD + EL	*Streptococcus pyogenes*	ATB	Discharge
Modares and Tabari [37]	2021	67	M	DM, s/p gastric mucosal biopsy	Diffuse	EP, N/V, fever	CT + EGD + EL	Group A Streptococci	ATB + total gastrectomy	Discharge
Takase *et al* [38]	2021	89	F	DM, CKD	Diffuse	EP, N/V	CT	Nil	ATB	Discharge
Saito *et al* [39]	2021	70	F	ALL s/p chemotherapy	Diffuse	Septic shock	CT + EGD	*Bacillus cereus*	ATB	Discharge
Wang *et al* [40]	2021	22	M	Eating contam-inated food	Diffuse	EP, N/V, fever, hematemesis	CT + EGD	*Enterococcus cecorum*	ATB	Discharge
Durdella *et al*[41]	2022	44	F	Nil	Localized	EP, N/V	CT	Nil	ATB	Discharge
Yu *et al*[42]	2022	72	F	Gastric adenoma s/p ESD	Localized	No	CT	Nil	Distal gastrectomy	Discharge

ATB: Antibiotic; ALL: Acute lymphoblastic leukemia; AML: Acute myeloid leukemia; APC: Argon plasma coagulation; AS: Ankylosing spondylitis; CKD:

Chronic kidney disease; CT: Computed tomography; DLBCL: Diffuse large B cell lymphoma; DM: Diabetes mellitus; EGD: Esophagogastroduodenoscopy; EL: Exploratory laparotomy; EP: Epigastric pain; ESCC: Esophageal squamous cell carcinoma; ESD: Endoscopic submucosal dissection; EUS: Endoscopic ultrasound; EUS-FNA: Endoscopic ultrasound fine-needle aspiration; F: Female; GAVE: Gastric antral vascular ectasia; GC: Gastric cancer; GU: Gastric ulcer; HBV: Hepatitis B virus; HCC: Hepatocellular carcinoma; HIV: Human immunodeficiency virus; M: Male; MDS: Myelodysplastic syndrome; MF: Myelofibrosis; MG: Myasthenia gravis; MM: Multiple myeloma; N/V: Nausea/vomiting; PA: Psoriatic arthritis; SMA: Superior mesenteric artery; s/p: Status post; URI: Upper respiratory infection.

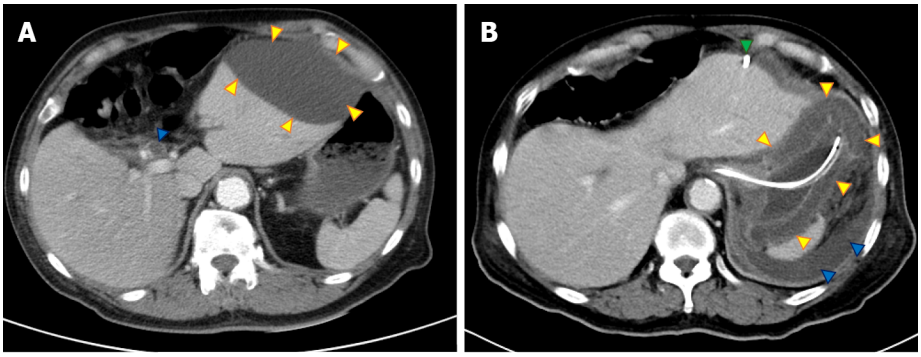

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Figure 1 Abdominal computed tomography. A: Initial abdominal computed tomography (CT) showed a dilated common bile duct (blue arrow) and left subphrenic abscess (yellow arrows); B: Repeat abdominal CT after biloma drainage (green arrow) showed bile leakage (blue arrows) and enlarged wall of the stomach (yellow arrows).

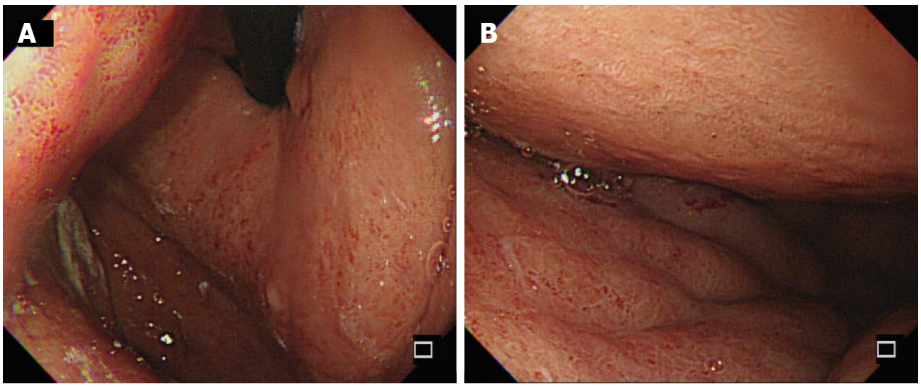

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Figure 2 Esophagogastroduodenoscopy. Esophagogastroduodenoscopy showed an edematous, hyperemic gastric mucosa with poor distensibility in the body of the stomach and the fundus of the stomach. A: The body of the stomach; B: The fundus of the stomach.

of a primary lesion. In our case, the PG was secondary.

The most common clinical presentation of PG is severe and acute epigastric pain accompanied by fever, vomiting, palpitation, melena, and hematemesis. The symptoms of PG mainly occur within 24 h, although they can develop over several days. It is important to differentiate PG from other acute abdomen etiologies such as acute pancreatitis, cholecystitis, and bowel perforation. Diagnostic modalities for PG include EGD, abdominal CT, and endoscopic ultrasonography (EUS). EGD findings show an edematous mucosa with fibrinopurulent exudates and superficial ulcerations, loss of rugae, and poor distensibility; however, these features are nonspecific to PG. Several differential diagnoses need to be considered, like acute gastric mucosal lesion, scirrhous gastric cancer, gastric syphilis, corrosive gastritis, malignant lymphoma of the stomach, gastrointestinal stromal tumor, and anisakiasis [16,35]. Each of these diseases is diagnosed according to the collective findings from EGD imaging examination along with patient data on clinical pattern, medical history, and culture test results. CT findings include obvious thickening of the gastric wall, and low-intensity areas within the gastric wall [17]. Of note, EUS has not been routinely recommended but is an excellent tool for detecting and tracking thickening of the gastric wall and degree of inflammation[16]. Standard forceps biopsy may not be diagnostic because it does not obtain sufficient submucosal tissue, which is the typically involved layer in PG.

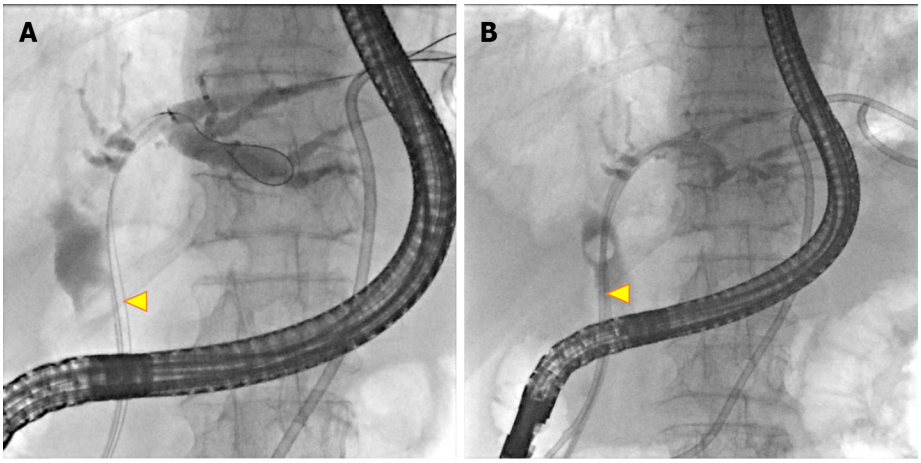

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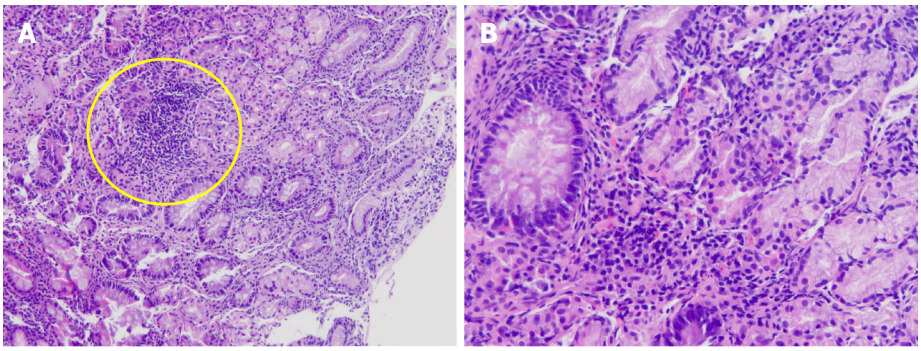
Figure 3 Endoscopic retrograde cholangiopancreatography. A: Dilated common bile duct (CBD) with one filling defect about 10 mm in size (yellow arrow); B: Endoscopic retrograde biliary drainage (yellow arrow) was performed for CBD stone-induced cholangitis.



DOI: 10.12998/wjcc.v10.i33.12430

Figure 4 Hemoxylin and eosin staining of gastric mucosal biopsy. A: At × 10 magnification, gastric submucosal and mucosal infiltration was observed (yellow circle); B: At × 20 magnification, clusters of lymphocytes, neutrophils, and plasma cells infiltrated into the submucosa.

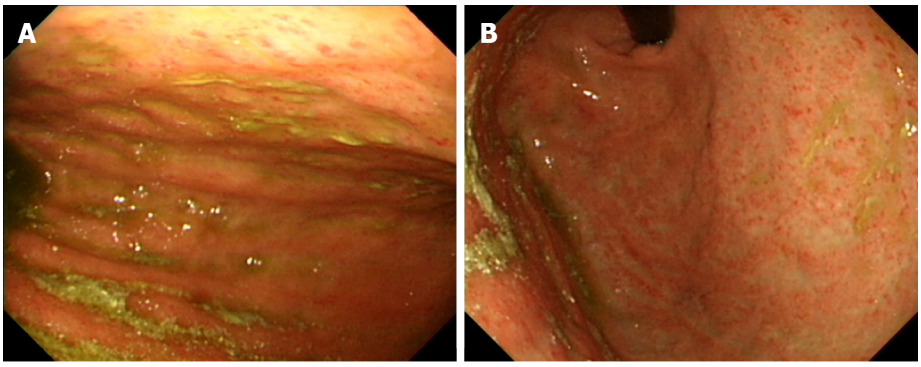


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Figure 5 Follow-up esophagogastroduodenoscopy. A normal gastric mucosa with improved distensibility of the stomach was observed in the body of the stomach and the fundus of the stomach. A: The body of the stomach; B: The fundus of the stomach.

In our review, the most common microorganism was *Streptococcus* spp. (56%, 19/34), followed by *Enterococcus* spp. (10%, 3/34). This result was in line with the 2014 bibliographic review by Rada-Palomino *et al*[14].

PG has a high mortality rate; the key to successful treatment is early diagnosis and therapy. Before invention with antibiotics, the mortality rate was 83%-92%[14]. Kim *et al*[2] reviewed 36 cases of PG between 1973 and 2003. The mortality rates for surgical intervention and conservative treatment with antibiotics were 20% and 50%, respectively. Recently, Rada-Palomino *et al*[14] reviewed 45 cases from

1980 to 2014 and found that the mortality rates for surgically and medically treated patients were 11% and 19%, respectively. In our review of cases reported from 2012 to 2022, the mortality rate was 12% (1/8) for surgery and 8% (3/36) for medical treatment. Through early diagnosis and appropriate antibiotic treatment, the mortality rate of PG has been gradually decreasing but is still high. Surgery should always be considered in refractory cases, which show clinical deterioration despite optimal medical management, and in the presence of complications, such as delayed perforation, abdominal compartment syndrome, bleeding, or stricture[9,15,17,22,37]. The surgery itself can be a partial or total gastrectomy, according to the range of inflammation[7]. In our case, conservation therapy with antibiotics alone was successful.

The recurrence rate of PG is low according to our literature review. Only one case, which was reported by Taniguchi *et al*[34], had recurrent PG at 5 d after discharge, and the causes considered most likely were a steroid treatment for allergy and a short-term course of antibiotics. To avoid recurrence, optimizing the process/timing of antibiotic cessation is important and should be determined by laboratory testing data and clinical pattern along with findings from follow-up imaging examinations (*e.g.*, CT or EUS).

A biloma is defined as a collection of bile located outside the bile duct. The main causes of such are iatrogenic or traumatic injuries[44], with cases of spontaneous biloma being relatively uncommon. The most frequent cause of spontaneous biloma is choledocholithiasis[45], with the underlying mechanism hypothesized as an increase in intraductal pressure due to stone obstruction. Unfortunately, the clinical presentations of biloma are non-specific, including abdominal pain, fever, nausea, vomiting, and jaundice[46].

Nowadays, treatment for spontaneous biloma is nonsurgical, including antibiotics and percutaneous drainage *via* pigtail catheter. ERCP is a feasible alternative with additional benefit because it can not only decompress the biliary tract by endoscopic sphincterotomy and stent placement but also identify the location and severity of an active bile leakage. Surgery is reserved for patients who fail endoscopic stone extraction or present a persistent active leak.

CONCLUSION

This case report describes a rare case of spontaneous biloma caused by choledocholithiasis followed by bile leakage-induced PG after the biloma drainage. PG itself is an uncommon diagnosis due to abdominal pain, but should be considered in patients with intraabdominal infection, especially from an infected organ adjacent to the stomach. The key to successful treatment is early diagnosis and initiation of therapy.

FOOTNOTES

Author contributions: Yang KC contributed to manuscript writing and editing, and data collection and analyses; Kuo HY and Kang JW supervised the study; Kang JW conceived the study; all authors have read and approved the final manuscript.

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