



Acute pancreatitis associated with massive bleeding due to a duodenal ulcer

Daisuke Uchida¹ · Yoshinari Kawai² · Hironari Kato¹ · Hiroyuki Okada¹

Received: 3 December 2018 / Accepted: 22 January 2019 / Published online: 29 January 2019
© Japanese Society of Gastroenterology 2019

Abstract

A 74-year-old man presented to the emergency department with acute abdominal pain in addition to anemia and melena, which were suspected to be due to gastrointestinal bleeding. Computed tomography (CT) revealed a blood-filled duodenum and acute pancreatitis. We prioritized treatments for pancreatitis, as the vital signs were stable, and temporary hemostasis was achieved. Two days later, esophagogastroduodenoscopy revealed a duodenal ulcer with an exposed vessel, and endoscopic hemostasis was performed. We urge clinicians to consider the possibility of pancreatitis associated with massive bleeding due to a duodenal ulcer.

Keywords Pancreatitis · Duodenal ulcer · Gastrointestinal bleeding

Abbreviations

WBC	White blood cell
RBC	Red blood cell
Ht	Hematocrit
Hb	Hemoglobin
Plt	Platelet
TP	Total protein
Alb	Albumin
T-Bil	Total bilirubin
D-Bil	Direct bilirubin
AST	Aspartate transaminase
ALT	Alanine transaminase
ALP	Alkaline phosphatase
γGTP	Gamma guanosine triphosphate
Amy	Amylase
ChE	Cholinesterase
LDH	Lactate dehydrogenase
Cr	Creatinine
BUN	Blood urea nitrogen
Na	Sodium
K	Potassium
Cl	Chlorine

Ca	Calcium
BE	Base excess
CRP	C-reactive protein
PT	Prothrombin time
PT-INR	Prothrombin time-international normalized ratio

Introduction

Gastrointestinal bleeding sometimes causes severe situations and immediate interventions are needed [1]. The treatment strategy depends on the causes of bleeding. Peptic ulcers are the most common causes of gastrointestinal bleeding and require invasive treatment, such as endoscopic hemostasis, interventional radiology, and surgery. They can also cause secondary problems, such as aspiration pneumonia, peritonitis, and ischemic organ failure. Although acute pancreatitis is rarely associated with gastrointestinal ulcer bleeding, it hampers treatment once it develops. Pancreatitis can develop in association with gastrointestinal diseases for several reasons. One is inadequate blood flow due to endoscopic procedures or pancreatic divisum [2–4]. Another is physical duodenal pressure elevation associated with massive bleeding in the duodenum [5]. Both bleeding and pancreatitis are severe situations requiring prompt treatments and careful management. We herein report a case of severe pancreatitis associated with massive bleeding due to a duodenal ulcer.

✉ Daisuke Uchida
pt77172s@okayama-u.ac.jp

¹ Department of Gastroenterology, Okayama University Hospital, 2-5-1 Shikata-cho, Okayama 700-8558, Japan

² Department of Gastroenterology, Onomichi Municipal Hospital, Hiroshima, Japan

Case report

A 74-year-old man under medical treatment for liver cirrhosis (Child–Pugh score A: five points) presented to the

Table 1 Laboratory data on admission (day 1)

Blood count	
WBC	$4.9 \times 10^3/\mu\text{L}$
RBC	$4.3 \times 10^6/\mu\text{L}$
Ht	39%
Hb	12.8 g/dL
Plt	$17 \times 10^4/\mu\text{L}$
Chemistry	
TP	6.6 g/dL
Alb	4.1 g/dL
T-BIL	0.8 mg/dL
AST	24 IU/L
ALT	17 IU/L
ALP	156 IU/L
γ GTP	42 IU/L
Amy	139 U/L
ChE	149 IU/L
LDH	190 IU/L
Cr	1.14 mg/dL
BUN	10.3 mg/dL
Na	145 mEq/L
K	3.4 mEq/L
Cl	102 mEq/L
CRP	0.65 mg/dL
Coagulation	
PT	76%
PT-INR	1.14

emergency department with acute abdominal pain with peritoneal signs and melena. A week before visiting, he was affected by gastroenteritis, which improved with antifatulent medication. At the visit, he had mild anemia (hemoglobin level: 12.8 mg/dL) according to a blood test (Table 1). Computed tomography (CT) revealed pancreatitis with air in the main pancreatic duct (Fig. 1a: yellow arrow), peripancreatic fluid collection (Fig. 1a: blue arrow), and hematoma in the bile duct (Fig. 1b: orange arrow) associated with a blood-filled duodenum (Fig. 1b: red arrow). After the insertion of a gastric tube, we prioritized conservative treatment for the pancreatitis, including the administration of proton pump inhibitors, protease inhibitors, and adequate fluid, and temporary hemostasis was achieved with the vital signs showing stability. His serum amylase level increased to 865 U/L the next day (Table 2), and we postponed esophagogastroduodenoscopy (EGD) to prevent aggravating the patient's pancreatitis.

However, 2 days after admission, his anemia progressed (hemoglobin level: 8.9 mg/dL) (Table 3), so esophagogastroduodenoscopy (EGD) was performed. A hemorrhaging duodenal ulcer with exposed vessels in the duodenal bulb (Fig. 2a) was detected, and hemostasis with endoscopic clipping was performed (Fig. 2b). Contrast-enhanced CT after EGD revealed peripancreatic fluid collection reaching beyond the lower pole of the left kidney (Fig. 3a), and pancreatic pleural effusion (Fig. 3b). Treatments for pancreatitis were continued, and the clinical symptoms improved gradually. However, the pleural effusion was intractable despite the improvement of pancreatitis, so he underwent pleural puncture 3 weeks after admission. This patient ultimately required a long duration of hospitalization (over 1 month) (Fig. 4).

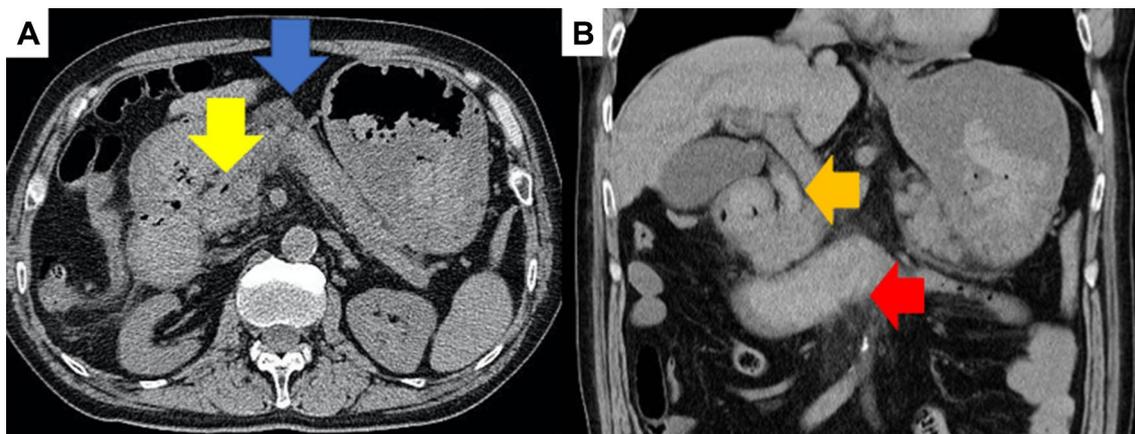


Fig. 1 **a** An axial view of an abdominal computed tomography (CT) scan. Air can be seen in the main pancreatic duct (yellow arrow), along with peripancreatic fluid collection (blue arrow). **b** A coronal

view of an abdominal CT scan. Hematoma can be seen in the bile duct (orange arrow), along with the blood-filled duodenum (red arrow)

Table 2 Laboratory data at day 2

Blood count	
WBC	$5.7 \times 10^3/\mu\text{L}$
RBC	$3.9 \times 10^6/\mu\text{L}$
Ht	37%
Hb	11.7 g/dL
Plt	$17 \times 10^4/\mu\text{L}$
Chemistry	
TP	5.9 g/dL
Alb	3.7 g/dL
T-BIL	0.9 mg/dL
AST	24 IU/L
ALT	18 IU/L
ALP	123 IU/L
γ GTP	35 IU/L
Amy	865 U/L
ChE	126 IU/L
LDH	162 IU/L
Cr	1.05 mg/dL
BUN	18 mg/dL
Na	142 mEq/L
K	3.4 mEq/L
Cl	107 mEq/L
Ca	8.2 mg/dL
BE	− 2.5 mmol/L
CRP	2.2 mg/dL

Table 3 Laboratory data at day 3

Blood count	
WBC	$11.9 \times 10^3/\mu\text{L}$
RBC	$2.9 \times 10^6/\mu\text{L}$
Ht	27%
Hb	8.9 g/dL
Plt	$8.3 \times 10^4/\mu\text{L}$
Chemistry	
TP	4.4 g/dL
Alb	2.8 g/dL
T-BIL	0.9 mg/dL
AST	245 IU/L
ALT	156 IU/L
ALP	325 IU/L
γ GTP	266 IU/L
Amy	2006 U/L
ChE	86 IU/L
LDH	422 IU/L
Cr	2.25 mg/dL
BUN	29.3 mg/dL
Na	140 mEq/L
K	3.8 mEq/L
Cl	106 mEq/L
Ca	7.6 mg/dL
BE	− 1.0 mmol/L
CRP	8.6 mg/dL

Discussion

Duodenal ulcer is a potentially severe problem that can require invasive treatment. Bleeding is the most frequently encountered situation and is typically treated with endoscopic hemostasis, interventional radiology, or surgery [6, 7]. The present patient was being treated for chronic hepatitis C with liver cirrhosis. The major cause of upper gastrointestinal bleeding in patients with liver cirrhosis is esophagogastric varices [8], and we initially suspected variceal bleeding. However, CT showed a massive hematoma in the duodenum and acute pancreatitis. Duodenal varices are one of causes of duodenal bleeding in patients with liver cirrhosis [9, 10]; however, this was considered unlikely based on the CT findings. Hematoma in the common bile duct indicated the potential for bleeding from biliary tract. Hemobilia is a major cause of upper gastrointestinal bleeding. There are some noniatrogenic causes of hemobilia, such as portal biliopathy, infection and malignancy [11], but these findings were not detected. Endoscopic retrograde cholangiopancreatography should be performed if hematoma in the bile duct is the main cause of pancreatitis; however, a blood test on admission did not reveal elevated hepatobiliary enzyme levels. In addition, the presence of air in the main pancreatic duct, which has been mixed with blood, indicated

regurgitation of blood from the duodenum across the papilla. We, therefore, prioritized the treatment of his pancreatitis because of his stable vital signs and the anticipated difficulty of an endoscopic procedure due to massive hematoma. At the day after admission, we planned to perform EGD, but blood test showed a sharp rise in the level of serum amylase. EGD was postponed; however, we were forced to perform EGD due to suspicion of rebleeding as a result. EGD revealed duodenal ulcer bleeding, and we presumed that his pancreatitis had been caused by the regurgitation of blood associated with the acute elevation of duodenal pressure, even though he had no history of sphincterotomy. CT showed no findings that would trigger obstructive pancreatitis (e.g., pancreatic stone, biliary stone, or a pancreatic tumor involving the intraductal papillary mucinous neoplasm). Additionally, there were no findings that would trigger hemobilia (e.g., portal biliopathy, biliary tumor, or a liver tumor). We, therefore, hypothesized that the condition was caused by the regurgitation of blood across the papilla.

Acute pancreatitis is a common disease that causes abdominal pain. Alcohol abuse and gallstones are responsible for the majority of cases, but we sometimes experience rare cases associated with other causes [12]. There are some reports of similar cases of acute pancreatitis associated with duodenal hematoma [5, 13, 14]. Chang et al. suggested

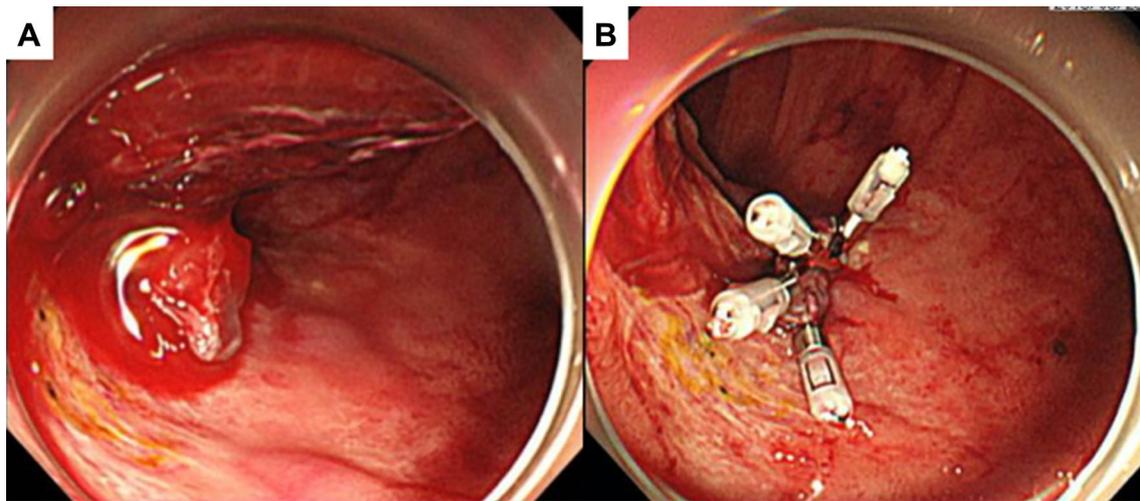


Fig. 2 **a** Esophagogastroduodenoscopy revealed a hemorrhaging duodenal ulcer with exposed vessels in the duodenal bulb. **b** Hemostasis with endoscopic clipping was performed

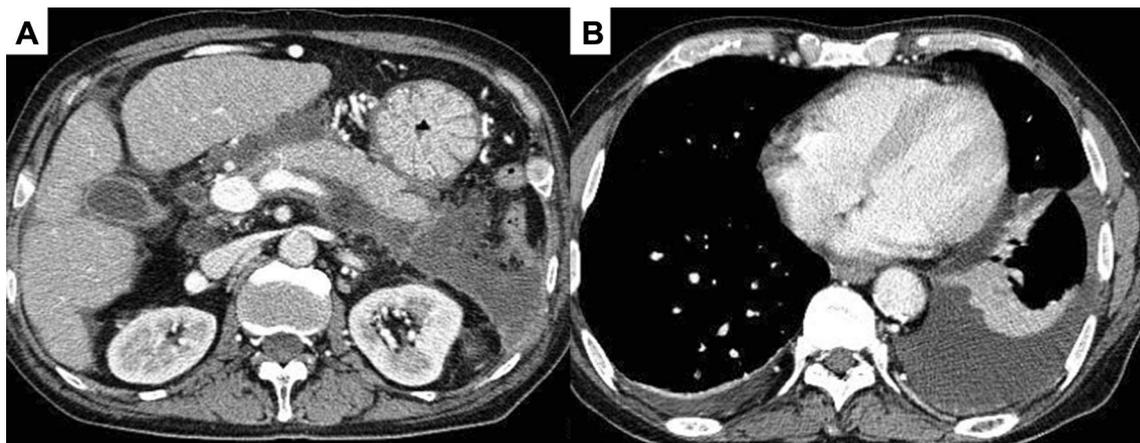


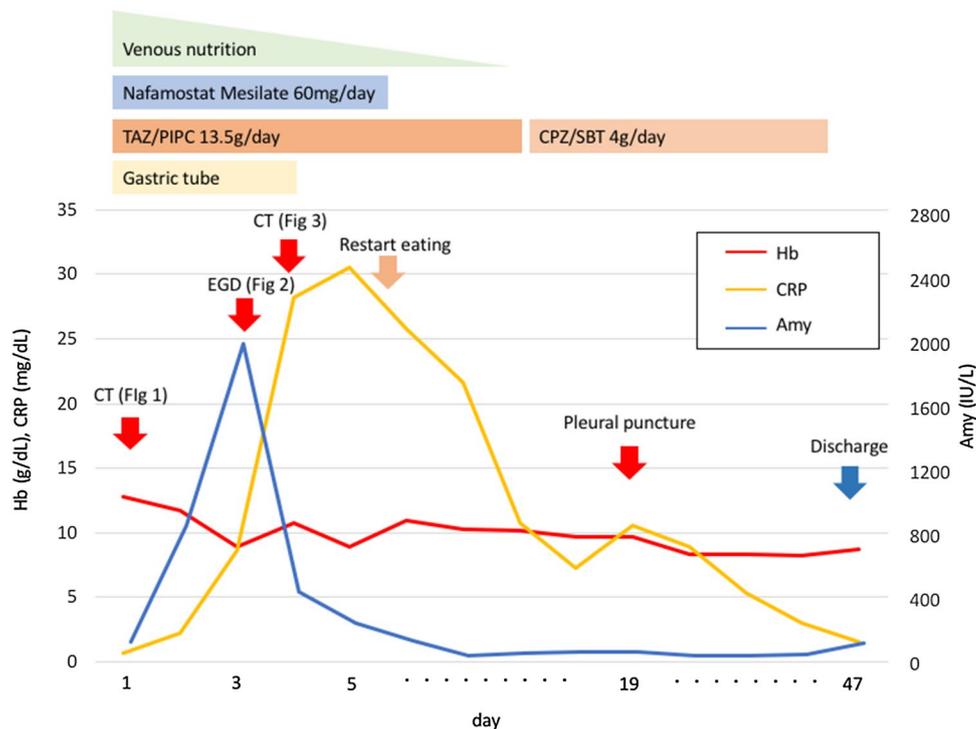
Fig. 3 **a** Contrast-enhanced computed tomography after esophagogastroduodenoscopy revealed peripancreatic fluid collection extending beyond the lower pole of the left kidney. **b** There was massive left-sided pleural effusion

two hypothetical mechanisms [5]. One is duodenal papilla obstruction due to hematoma. The other is vascular disruption associated with pancreatitis. In our case, the former mechanism was considered; however, the cause of this regional blood accumulation remains to be elucidated. Duodenal hematoma was responsible in the majority of cases with iatrogenic causes (e.g., endoscopic biopsy) in patients with coagulation disorders, including those with liver cirrhosis [3, 15]. However, they are described as “intramural” hematoma rather than “intraduodenal” hematoma. A previous report indicated intestinal edema associated with vomiting due to pain [5]. These situations resemble our own case in the sense that pancreatitis was caused by obstruction of the major papilla associated with bleeding. We also speculated that the condition in our case was influenced by

hypoperistalsis and intestinal edema, as the patient had viral enterogastritis before admission. Hypoperistalsis and massive bleeding into the duodenum during shots might have induced acute elevation of the duodenal pressure. We hoped to perform endoscopic retrograde cholangiopancreatography or magnetic resonance imaging to confirm our hypothesis, but the patient refused these examinations because his symptoms improved. This is a limitation of this report.

Patients with liver cirrhosis may develop peptic ulcers as often as non-cirrhotic patients, but ulcers in cirrhotic patients may be more severe than those in non-cirrhotic patients and more frequently require endoscopic treatment [16]. The present patient was unexpectedly complicated with pancreatitis, which can be a fatal disease, so a cautious strategy was required. We prioritized treatments for

Fig. 4 The clinical course after admission. *Hb* hemoglobin, *CRP* C-reactive protein, *Amy* Amylase



pancreatitis after the insertion of gastric tube for decompression; however, it is not clear whether this strategy was appropriate. The patient required emergency endoscopic hemostasis due to duodenal ulcer bleeding and his hospital stay was prolonged as a result. Persistent infection of pleural and abdominal fluid collection after pancreatitis also contributed to his long duration of hospitalization. Earlier endoscopic management might have achieved a better result; however, this approach would have been associated with potential risks, including the possibility of aggravating the patient's pancreatitis. In previous reports, some cases required surgical treatment [5].

We should consider the possibility of severe pancreatitis induced by duodenal ulcer bleeding. Treatment should be conducted systematically to avoid poor outcomes, especially in patients with severe underlying diseases.

Compliance with ethical standards

Conflict of interest The authors declare no conflicts of interest or financial relationships relevant to this article.

Human/animal rights All procedures were carefully conducted in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and in line with the Helsinki Declaration.

Informed consent Informed consent was obtained from the patient for being included in the study.

References

1. Abougergi MS, Travis AC, Saltzman JR. Impact of day of admission on mortality and other outcomes in upper GI hemorrhage: a nationwide analysis. *Gastrointest Endosc.* 2014;80:228–35.
2. Choi YH, Yoon SM, Kim EB, et al. A rare case of pancreas divisum accompanied by acute pancreatitis following endoscopic hemostasis for duodenal ulcer bleeding. *Korean J Gastroenterol.* 2017;69:248–52.
3. Dibra A, Kellici S, Celiku E, et al. Intramural duodenal hematoma after submucosal injection of epinephrine for a bleeding ulcer: case report and review. *G Chir.* 2015;36:29–31.
4. Tashima T, Ryozaawa S, Ohata K. Rare case of severe acute pancreatitis following over-the-scope clip-assisted duodenal endoscopic mucosal resection using a cap-fitted endoscope in a patient with pancreas divisum. *Dig Endosc.* 2018;30:679.
5. Shiozawa K, Watanabe M, Igarashi Y, et al. Acute pancreatitis secondary to intramural duodenal hematoma: case report and literature review. *World J Radiol.* 2010;2:283–8.
6. Laine L, Jensen DM. Management of patients with ulcer bleeding. *Am J Gastroenterol.* 2012;107:345–60.
7. Loffroy R, Rao P, Ota S, et al. Embolization of acute nonvariceal upper gastrointestinal hemorrhage resistant to endoscopic treatment: results and predictors of recurrent bleeding. *Cardiovasc Intervent Radiol.* 2010;33:1088–100.
8. Romcea AA, Tantau M, Seicean A, et al. The etiology of upper gastrointestinal bleeding in cirrhotic patients. *Clujul Med.* 2013;86:21–3.
9. Hashizume M, Tanoue K, Ohta M, et al. Vascular anatomy of duodenal varices: angiographic and histopathological assessments. *Am J Gastroenterol.* 1993;88:1942–5.
10. Copelan A, Chehab M, Dixit P, et al. Safety and efficacy of angiographic occlusion of duodenal varices as an alternative to TIPS: review of 32 cases. *Ann Hepatol.* 2015;14:369–79.

11. Berry R, Han J, Girotra M, et al. Hemobilia: perspective and role of the advanced endoscopist. *Gastroenterol Res Pract.* 2018;2018:3670739.
12. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology.* 2013;144:1252–61.
13. Silva JD, Veloso N, Godinho R, et al. Fatal acute pancreatitis following sclerosis of a bleeding duodenal ulcer complicated by an intramural duodenal hematoma. *Rev Esp Enferm Dig.* 2012;104:603–4.
14. Chang CM, Huang HH, How CK. Acute pancreatitis with an intramural duodenal hematoma. *Intern Med.* 2015;54:755–7.
15. Sugai K, Kajiwara E, Mochizuki Y, et al. Intramural duodenal hematoma after endoscopic therapy for a bleeding duodenal ulcer in a patient with liver cirrhosis. *Intern Med.* 2005;44:954–7.
16. Gonzalez-Gonzalez JA, Garcia-Compean D, Vazquez-Elizondo G, et al. Nonvariceal upper gastrointestinal bleeding in patients with liver cirrhosis. Clinical features, outcomes and predictors of in-hospital mortality. A prospective study. *Ann Hepatol.* 2011;10:287–95.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.