Endoscopic Examination for Duodenal Ulcer Bleeding during Transcatheter Arterial Embolization: Analysis of Two Cases

Ryuzo DEGUCHI[*1], Toshiyuki SAKUMA[*1], Fusao OGASAWARA[*1], Shinji TAKASHIMIZU[*1], Jun KOIKE[*1], Tetsuya MINE[*1] and Yoshiro IWATA[*2]

[*1]*Department of Gastroenterology and* [*2]*Department of Radiology, Tokai University School of Medicine*

(Received July 28, 2009; Accepted November 13, 2009)

Endoscopy is usually effective in treating duodenal ulcer bleeding, but depending on the lesion site and overall patient condition, hemostasis may be difficult to achieve with endoscopy alone. We described two patients with duodenal ulcer bleeding in whom endoscopic hemostasis was difficult. Immediately after transcatheter arterial embolization, endoscopic examination was used to confirm hemostasis and completing of the angiographic procedures.

Key words: DU bleeding, TAE

INTRODUCTION

The use of *Helicobacter pylori* eradication therapy as a first-line treatment for peptic ulcer disease has dramatically reduced the incidence of ulcer recurrence [1]. However, bleeding remains a potentially serious complication in ulcer patients. Depending on the lesion site and overall patient condition, endoscopic hemostasis of duodenal ulcer (DU) bleeding is sometimes unsuccessful. In the past, surgical intervention was necessary, but with the development of transcatheter arterial embolization (TAE) using angiographic procedures, hemostasis can be achieved noninvasively without laparotomy [2].

One disadvantage of TAE in DU bleeding is that treatment is not performed under direct lesion visualization. Intermittent bleeding can make it difficult to determine the embolization site or to limit the extent of embolization when bleeding has stopped.

We recently performed endoscopy during TAE in two patients with DU bleeding in whom endoscopic hemostasis was difficult. The endoscopic findings were useful for assessing the extent of embolization, and determining the schedule for TAE and follow-up management of the lesions. Here, we present these cases and discuss the related literature.

Case 1

An 85-year-old woman with chronic renal failure of unknown etiology who was undergoing regular follow-up in our hospital was transported to our emergency room with a chief complaint of upper and lower GI bleeding. Her vital signs were: blood pressure, 92/31 mmHg; pulse, 75 regular beats per minute; body temperature, 37.6 °C; and respiratory rate, 20 per minute. Her laboratory data revealed a white blood cell count of 37000/mm^3, hemoglobin level of 4.2 g/dl, and platelet count of 17.4 × 10^4/mm^3. The serum albumin level was 2.6 g/dl. The blood urea nitrogen level was 206 mg/dl. The creatinine level was 5.3 mg/dl. The potassium level was 7.5 mEq/L. The levels of lactate dehydrogenase, liver enzymes, sodium, chloride and total bilirubin were normal. Based on the finding of hypotension and severe anemia on laboratory testing, urgent upper GI endoscopy was performed. A bleeding ulcer vessel trunk in the anterior duodenal bulb was clipped, active bleeding stopped, and endoscopy was completed (Forrest grade IB; Fig. 1). However, the following day, endoscopy revealed persistent active bleeding (Fig. 2). Despite injection with ethanol and hypertonic saline/epinephrine, the bleeding was not controlled, so angiography was performed.

On celiac-superior mesenteric angiography, no contrast extravasation or aneurysms were noted (Fig. 3). The posterior superior pancreaticoduodenal artery (PSPDA) was identified as the responsible lesion. A catheter was inserted, and gelfoam and coil embolization was started (Fig. 4a). When the distal segment was embolized, a branch to the duodenal bulb (not previously visualized) began to appear, with blushing of the bulb wall (Fig. 4b). To interrupt blood supply to the bulb, coil embolization was performed to the origin of the PSPDA. After embolization, gastroduodenal angiography revealed an absence of blood flow to the bulb (Fig. 4c). The catheter was then inserted into the anterior superior pancreaticoduodenal artery, with possible anastomosis to the PSPDA, and absence of blushing of the bulbar wall due to collateral circulation was confirmed. Common hepatic angiography also confirmed the absence of arterial branches to the bulb from the proper hepatic artery and right gastric artery (Fig. 4d). Intravascular hemostasis was completed. Next, in the angiography suite, endoscopy was performed. The DU bleeding before TAE was confirmed to be completely resolved (Fig. 5). The duodenal bulb mucosal surface appeared slightly pale. Embolization was considered

Ryuzo DEGUCHI, Department of Gastroenterology, Tokai University School of Medicine, 143 Shimokasuya,Isehara,Kanagawa 259-1193, Japan
Tel: +81-463-93-1121 Fax: +81-463-93-7134 E-mail: deguchi@is.icc.u-tokai.ac.jp

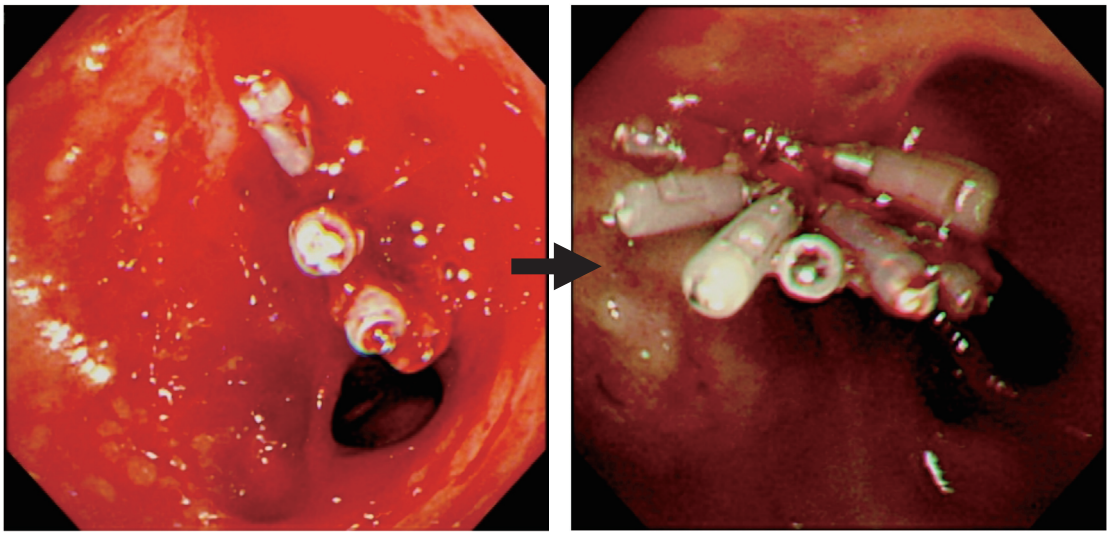

Fig. 1 Endoscopic images of the ulcer base with a vessel trunk visible in the anterior duodenal bulb (patient 1). Clipping of the vessel stopped active bleeding.

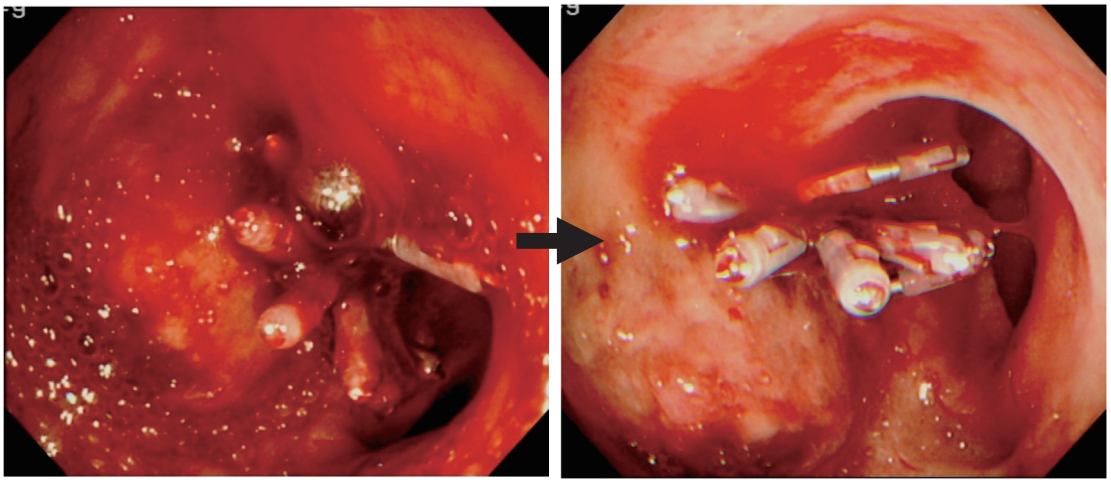

Fig. 2 The following day, endoscopy revealed persistent active bleeding. Despite re-clipping and chemical hemostasis, complete hemostasis was not achieved.

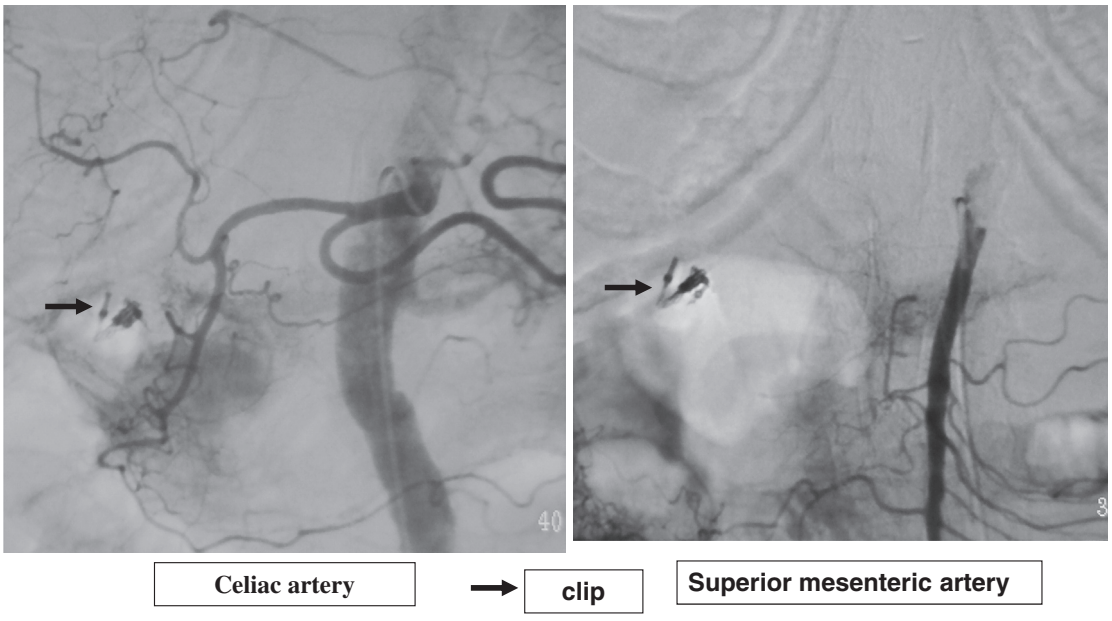

Fig. 3 On celiac-superior mesenteric angiography revealed withoutevidence of contrast extravasation around clips (arrow).

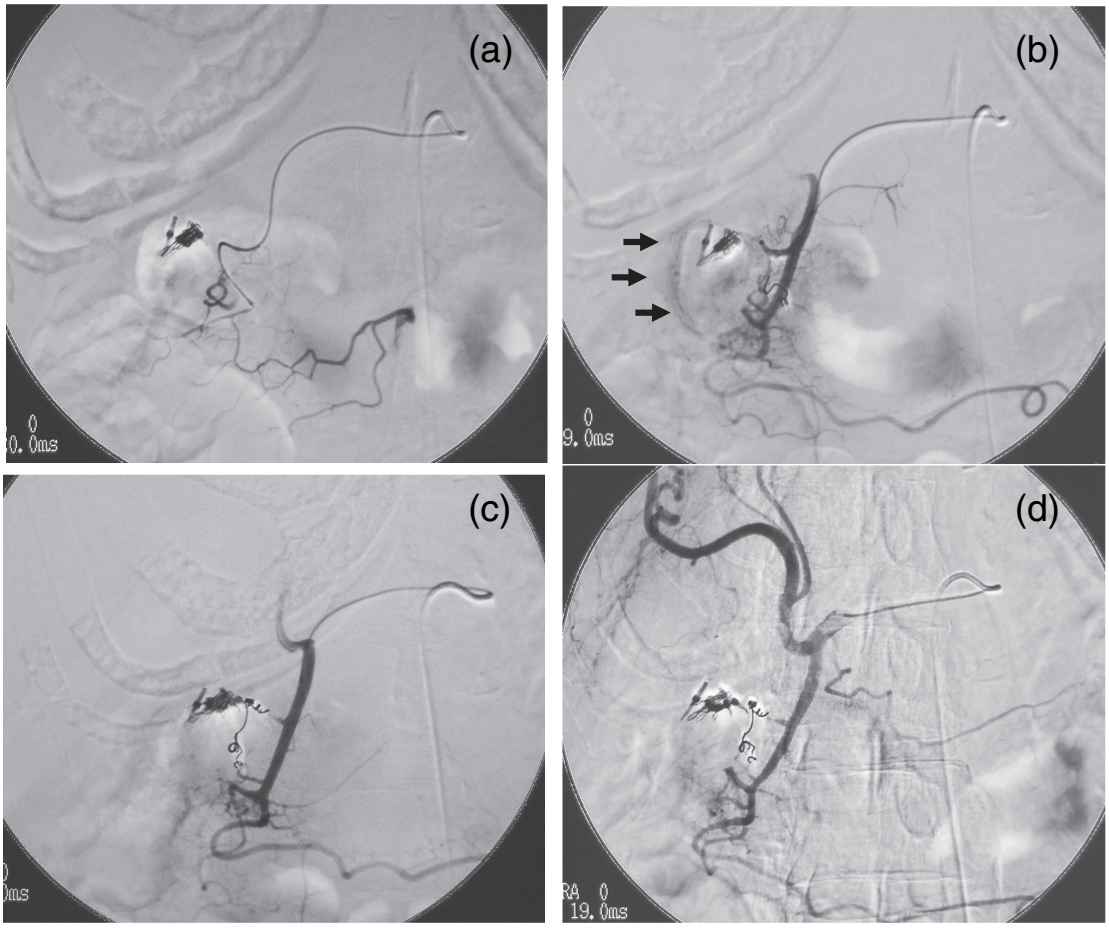

Fig. 4 Embolization procedure. (a) The posterior superior duodenal artery (PSPD) was identified as the responsible lesion. (b) After embolization of distal part of PSPD using microcoil, stain of lateral part of the bulb wall was revealed clearly (arrows). (c) Additional microcoil insertion to proximal part of PSPD. Angiography revealed absence of blood flow to the bulb. (d) Post embolization common hepatic angiography confirmed complete isolation of blood flow to bulb from celiac system.

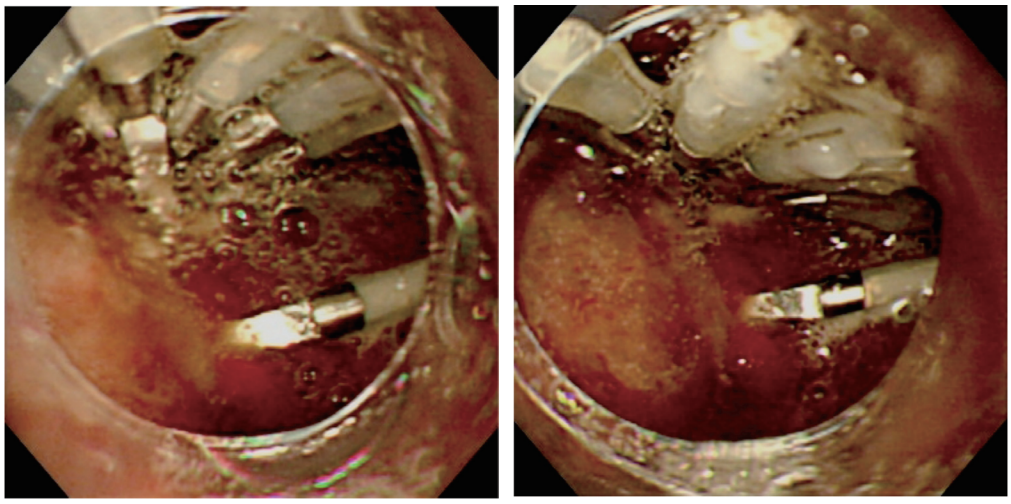

Fig. 5 DU bleeding after complete resolution of TAE.

effective, and angiography was completed.

The day after TAE, gastroscopy revealed multiple erosions/ulcers in the pylorus and antrum (Fig. 6). Duodenoscopy showed no bleeding, but there was a shallow ulcer proximal to the bleeding ulcer site (Fig. 7). One week after TAE, upper GI endoscopy showed that the erosions in the pylorus and antrum had disappeared, and the DU bleeding source had healed to a minor erosion (Fig. 8). The patient's hospital course was complicated by renal insufficiency, and following admission, she required transfusion of 18 units of packed red blood cells. She was discharged from the hospital 2 weeks after TAE.

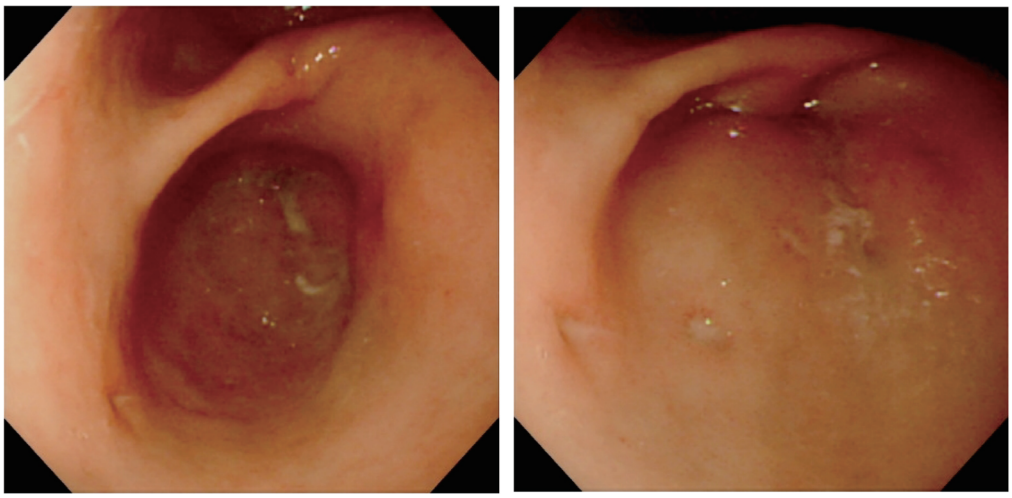

Fig. 6 Gastroscopic images at one day after TAE, showing multiple erosions/ulcers present in the pylorus and antrum.

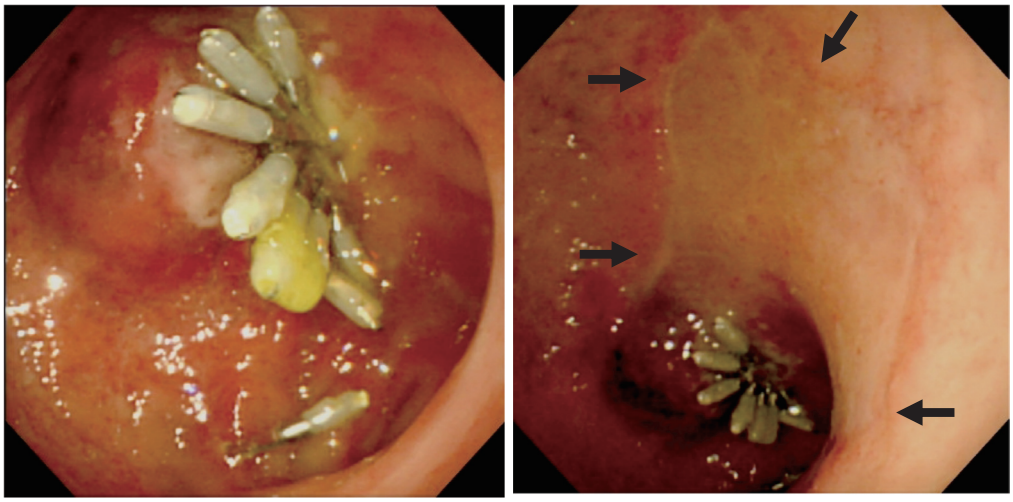

Fig. 7 In duodenoscopy one day after TAE, no bleeding was noted from the ulcer base in the anterior duodenal bulb, but proximally, there was a shallow ulcer (arrows).

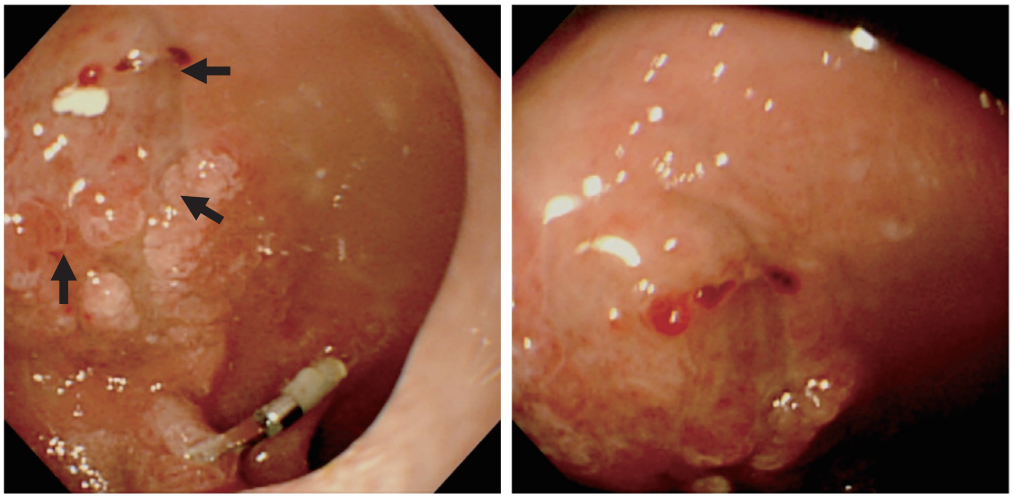

Fig. 8 Upper GI endoscopy one week after TAE. The DU that had been the source of bleeding healed to a minor erosion (arrows).

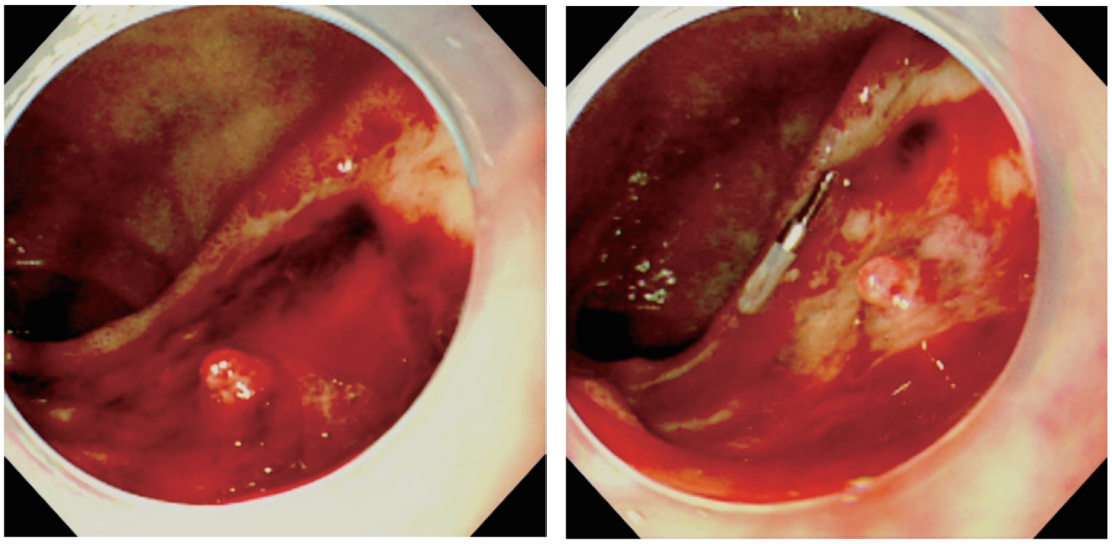

Fig. 9 In patient 2, the ulcer vessel was identified as the source of bleeding in the posterior duodenal bulb, but clipping did not achieve hemostasis. Despite injection of hypertonic saline, ethanol, and epinephrine, bleeding from the vessel stump continued.

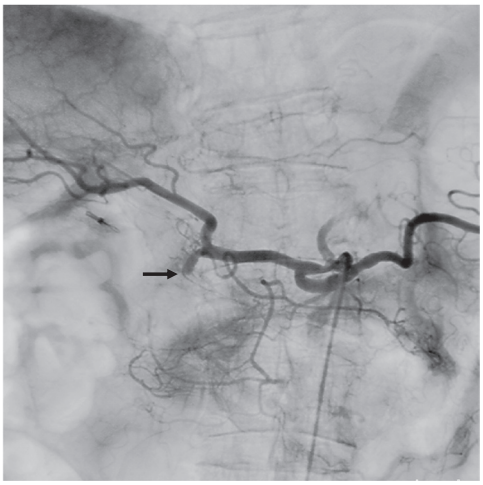

Fig. 10 Celiac angiography showing the blind-ended gastroduodenal artery.

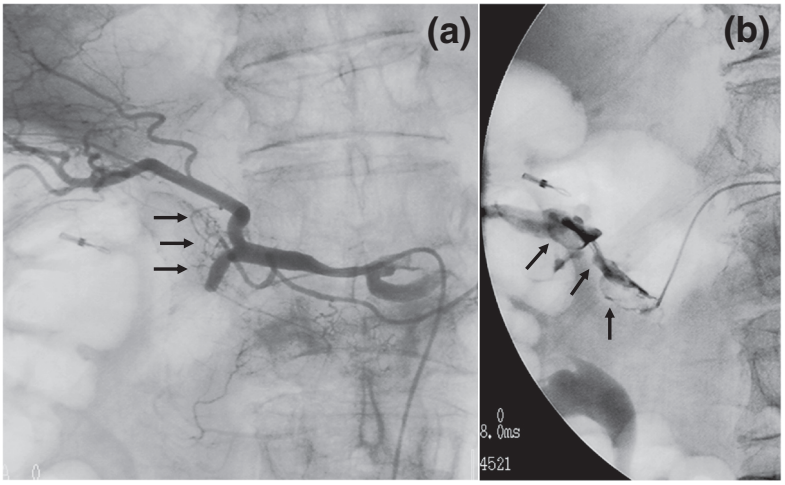

Fig. 11 (a) Visualized branch proximal to the blind end of the gastroduodenal artery. (b) During microcatheterization near the blind end, the catheter deviated into what appeared to be the duodenal lumen. Contrast medium from the catheter tip extravasated into the gut lumen.

Case 2

An 87-year-old man receiving non-steroidal anti-inflammatory drugs under the supervision of his personal physician for a lumbar compression fracture had tarry stools and was transported to our emergency department. His vital signs were: blood pressure, 98/62 mmHg; pulse rate, 115 regular beats per minute; body temperature, 36.7 °C; and respiratory rate, 16 per minute. His laboratory data revealed a white blood cell count of 16300/mm^3, hemoglobin level of 7.2 g/dl, and platelet count of 38.2 × 10^4/mm^3. The serum albumin level was 2.1 g/dl. The blood urea nitrogen level was 10 mg/dl. The creatinine level was 0.7 mg/dl. The potassium level was 3.3 mEq/L. The levels of lactate dehydrogenase, liver enzymes, sodium, chloride and total bilirubin were normal. Because of persistent tachycardia and severe anemia on laboratory testing, urgent upper GI endoscopy was performed. The ulcer vessel was identified as the source of bleeding in the posterior duodenal bulb, but clipping did not achieve hemostasis. In addition, injection of epinephrine-hypertonic saline and ethanol did not stop bleeding from the vessel stump (Forrest grade IB; Fig. 9), so angiography was performed.

Celiac angiography showed that the gastroduodenal artery was blind-ended (Fig. 10). A branch proximal to the blind end, but no aneurysm, was found, so the artery was considered occluded (Fig. 11a). During microcatheterization near the blind end, the catheter deviated to what appeared to be the duodenal lumen, and contrast medium from the catheter tip extravasated into the gut lumen (Fig. 11b). A 6-cm helical wound coil was released from the microcatheter with 3 cm in the gut lumen and 3 cm in the artery (Fig. 12). Then the gastroduodenal artery was filled with a coil. From the blind end of the gastroduodenal artery to the point where the it perforated the duodenum, the lumen was occluded due to endoscopic treatment, and at the start of angiography, no extravasation had been observed. Next, a coil was added in the right gastric artery to

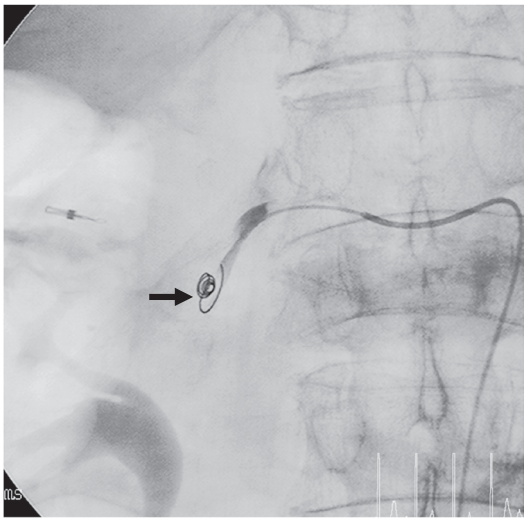

Fig. 12 A 6-cm helical embolization coil (arrow) was released from the microcatheter, with 3 cm in the gut lumen and 3 cm in the artery.

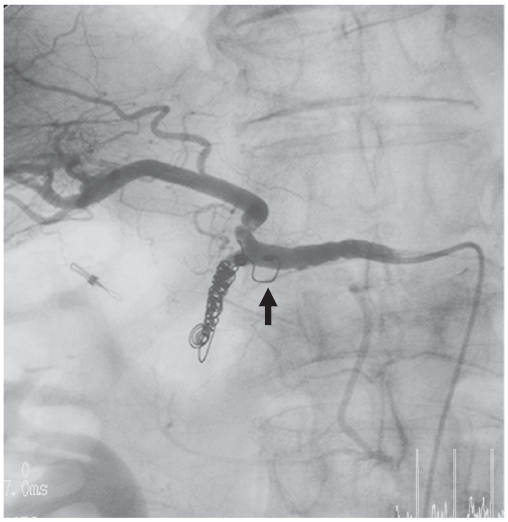

Fig. 13 A coil was added in the right gastric artery to stop blood flow from the pyloric branches.

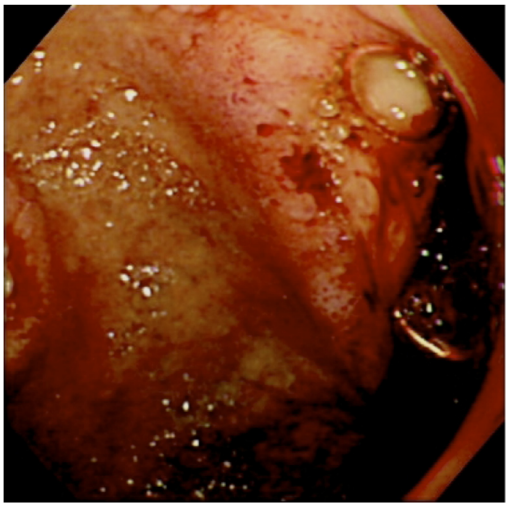

Fig. 14 Visualization of the exposed coil from the bleeding vessel trunk shows that active bleeding had stopped.

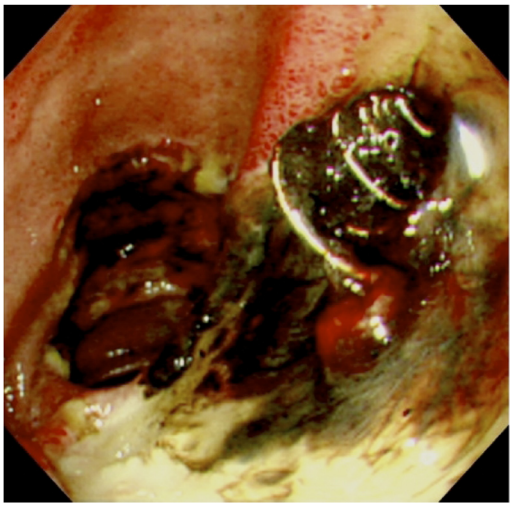

Fig. 15 Exposed coil visible from the vessel trunk of the ulcer base. There is no bleeding, but distal to the prior bleeding ulcer, a post-TAE ulcer is noted.

stop blood flow from the pyloric branches (Fig. 13).

After pancreaticoduodenal angiography (superior mesenteric artery branch) confirmed absence of an artery related to the bulb, we performed endoscopy in the angiography suite. The exposed coil from the vessel trunk bleeding source was visualized, and active bleeding had stopped (Fig. 14). The site where the microcatheter had deviated into the bulb lumen during TAE was indeed confirmed to have originated from the bleeding vessel. We decided to complete the embolization.

The day after TAE, endoscopy revealed the exposed coil from the vessel trunk of the ulcer base. There was no bleeding, but distal to the prior bleeding ulcer, a post-TAE ulcer was noted (Fig. 15). Endoscopy 6 weeks after TAE for hemostasis of DU bleeding showed the embolization coil and healing of the distal ulcer (Fig. 16). From the time of the urgent endoscopy, the patient required transfusion of 8 units of packed red blood cells. He was discharged from the hospital 2 weeks after TAE.

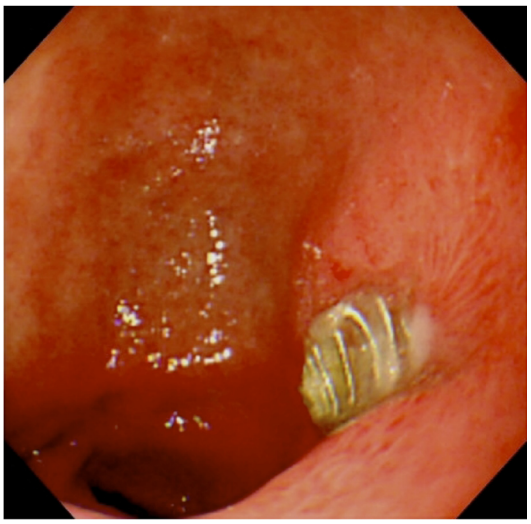

Fig. 16 Visualization of the coil following TAE shows that the bleeding ulcer had healed. The distal ulcer also healed.

Table 1 Problems in endovascular treatment of DU bleeding from an interventional radiology perspective

1. Problems related to anatomy

a. Many feeders exist in each part of the duodenal bulb, for example:

Superior duodenal artery – supplying the superior, anterior, and posterior walls Posterior duodenal artery – perfuses the inferoposterior wall

Pyloric branch – perfuses the pyloric side of bulb

Anterior and posterior superior pancreaticoduodenal artery – perfuses descending portion of bulb

b. Complexity of the arterial anastomosis of each feeder

2. Technical problems

a. Angiography may not always identify the point of bleeding, especially intermittent bleeding.

b. Catheter insertion is difficult for small feeders, and the bleeding point is often not accessible to the catheter tip.

c. Embolization at one site may not achieve hemostasis, because of remaining blood flow via the anastomosing branches from other arteries.

3. Post-embolization ischemia of gut and surrounding organ

a. Extensive embolization may be required in massive bleeding, regardless of ischemic risk.

b. There are no guidelines for avoiding duodenal ischemia in embolization treatment.

We reviewed endoscopic treatment of DU bleeding at our hospital between 2005 and 2008. Among a total of 34 patients, 32 (94.1%) had successful endoscopic hemostasis, 2 (5.9%) required angiography, and none (0%) required surgery. All patients were able to be discharged without requiring laparotomy.

DISCUSSION

Helicobacter pylori eradication therapy, now considered standard for treatment of peptic ulcer disease worldwide, has led to the decreased incidence of ulcer recurrence and the need for endoscopic hemostasis [3]. However, some patients like ours still present with bleeding ulcers, and endoscopic hemostasis is required.

Advances in endoscopic techniques have improved treatment outcomes, but patients with DU bleeding whose endoscopy fails to achieve endoscopic hemostasis and who require surgery (laparotomy) are not uncommon [4–5]. At our hospital from 2005 to 2008, all patients with DU bleeding who were successfully treated with noninvasive hemostasis required no laparotomy and were discharged from the hospital. This attests to increased technical skills of physicians performing endoscopy, but as described in this report, some patients still require hemostasis using angiographic procedures.

One disadvantage of TAE in DU bleeding is that treatment is not performed under direct lesion visualization. Intermittent bleeding and subtle differences of arterial anatomy in each patient can make it difficult to determine the embolization site or limit the extent of embolization when bleeding has stopped.

Table 1 lists some problems in endovascular treatment of DU bleeding from an interventional radiology perspective [6–9]. In many medical centers, patients with DU bleeding whose endoscopic hemostasis fails are automatically sent to surgery, as classically described in textbooks. The main reason for this is that not all hospitals have specialists who can perform endovascular procedures. In addition, some primary care physicians are not fully aware of the most up-to-date treatment available from specialists (e.g., endoscopic physicians and radiologists), and therefore some primary physicians may be unfamiliar with the optimal therapy. For example, with advances in angiography, including microcatheterization and microcoils, embolization has become highly effective in the last few years [10]. Nevertheless, transcatheter embolization carries a risk of complications including bowel ischemia, secondary duodenal stenosis and gastric/ hepatic infarction [11–12]. Our patients developed minor bowel ischemia, but they recovered without any additional treatment.

In our patients, endoscopy performed during TAE was very useful for precise embolization and for determining when to complete the procedure. Our review of the literature suggests that endoscopy is not routinely performed in the angiography suite. This may be due to segmentation of medical care, excessive respect for colleagues, or lack of familiarity among physicians of advances and limitations in other specialty disciplines.

Further, angiography can be performed in the time it takes to prepare for emergency surgery — about one hour. If TAE fails to achieve hemostasis, the patient can then be taken to surgery. This results in efficient and effective care for patients. Moreover, if TAE achieves hemostasis, the GI endoscopic physician better understands the embolization process, and is thus able to provide better patient follow-up.

REFERENCES

1) Sone Y, Kumada T, Toyoda H, Yokoyama M, Kato M, Asaka M. Eradicating Helicobacter pylori in peptic ulcer disease reduces medical costs in the community. Helicobacter. 13: 346–351, 2008.
2) Rahn NH 3rd, Tishler JM, Han SY, Russinovich NA. Diagnostic and interventional angiography in acute gastrointestinal hemorrhage. Radiology 143(2): 361–366, 1982.
3) Gisbert JP, Khorrami S, Carballo F, Calvet X, Gene E, Dominguez-Munoz E. Meta-analysis: Helicobacter pylori eradication therapy vs. antisecretory non-eradication therapy for the prevention of recurrent bleeding from peptic ulcer. Aliment Pharmacol Ther 19(6): 617–629, 2004.
4) Cheung FK, Lau JY. Management of massive peptic ulcer bleeding. Gastroenterol Clin North Am 38(2): 231–243, 2009.
5) Chiu PW, Ng EK, Wong SK, Teoh AY, Cheung FK, Yung MY, Sung JJ, Lau JY. Surgical salvage of bleeding peptic ulcers after failed therapeutic endoscopy. Dig Surg 26(3): 243–248, 2009.
6) Hiramatsu K. X-ray Anatomy of the Abdominal Vascular System. First Japanese edition: Igaku-shoin Ltd, 1982: 137–138.
7) Okazaki M. Emergent Transcatheter arterial embolotherapy for the patient with massive gastrointestinal hemorrhage. Jpn J Intervent Radiol 16: 138–142, 2001.
8) Furukawa A, Yamasaki M, Tanaka T, Nitta N, Takazakura

R, Sakoda M, Maeda K, Takahashi M, Murata K, Kanasaki S, Yokoyama K, Nagata T, Onaka T, Onaka Y, Sakamoto T. Gastrointestinal arterial hemorrhage; diagnosis and radiological intervention. Jpn J Intervent Radiol 18: 16–21, 2003.
9) Nakamura K. The role of interventional radiology in acute gastrointestinal bleeding. Jpn J Intervent Radiol 23: 141–152, 2008.
10) Aina R, Oliva VL, Therasse E, Perreault P, Bui BT, Dufresne MP, Soulez G. Arterial embolotherapy for upper gastrointestinal hemorrhage: outcome assessment. J Vasc Interv Radiol 12(2): 195–200, 2001.
11) Ljungdahl M, Eriksson LG, Nyman R, Gustavsson S. Arterial embolization in management of massive bleeding from gastric and duodenal ulcers. Eur J Surg 168: 384–390, 2002.
12) Gralnek IM, MSHS, Barkun AN, CM, M Sc, Bardou M. Management of bleeding from a peptic ulcer. N Eng J Med 359: 928–937, 2008.