Duodenal Diverticular Bleeding Successfully Treated Using Transcatheter Arterial Embolization: a case report

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ABSTRACT

We present a case of an 82-year-old man with duodenal diverticular bleeding (DDB) who was successfully treated using transcatheter arterial embolization (TAE). Initially, endoscopy results indicated duodenal bleeding; however, the exact bleeding site could not be located. A follow-up computed tomography (CT) scan revealed contrast extravasation from a periampullary duodenal diverticulum. Repeated endoscopy confirmed the bleeding site. However, hemostasis failed because excessive bleeding obscured the field of vision. As his condition worsened, the patient was transferred to the radiology department for TAE. His condition gradually improved after TAE. No obvious complication was observed after discharge. This case demonstrates TAE as a feasible alternative for managing complex DDB cases.

The first case of duodenal diverticulum was reported in 1710 [1]. The advent of endoscopy and computed tomography (CT) led to the incidental identification of an increasing number of duodenal diverticula. Most duodenal diverticula were reported to be asymptomatic and rarely resulted in complications such as bleeding, perforation, or obstruction. Managing duodenal diverticulum bleeding (DDB) mostly entails endoscopic therapy or diverticulectomy. Only few studies have reported the use of transcatheter arterial embolization (TAE) for managing DDB.

We report the case of an elderly man with DDB who was successfully treated using TAE for hemostasis. We recommend TAE in DDB management, particularly in patients at high risks for surgery or who have had failed attempts at endoscopic hemostasis.

CASE PRESENTATION

An 82-year-old man initially presented to the psychiatry clinic at our hospital for symptoms of disturbed consciousness, which included self-talking and sleepwalking. He had an underlying history of hypertension that was under medical control. Severe pale conjunctiva was observed during physical examination. When he visited our clinic, his serum hemoglobin level was 3.5 g/dL. He was immediately transferred to our emergency department for further examination.

He denied experiencing any gastrointestinal disorder-related symptoms, such as abdominal pain, nausea, vomiting, or changes in bowel habits. His vital signs were relatively stable. Nevertheless, digital examination revealed tarry stool; moreover, the results of the fecal occult blood test were positive. The first endoscopic examination revealed a large ulcer at the A1 stage at the anterior wall of the pylorus and no active bleeding. The patient was hydrated, received blood transfusion and a high dose of esomeprazole, and was admitted to the gastroenterology ward of our hospital.

After admission, no incidence of tarry stool was observed. However, an episode of bloody stool occurred after 5 days. After blood transfusion and hydration, endoscopy was performed again. The second endoscopic examination revealed the previous gastric ulcer at the pylorus and some blood clots in the stomach and second portion of
the duodenum; however, no active bleeding site could be observed. Because the bleeding source could not be identified, we performed diagnostic angiography. Angiography of the superior mesentery artery (SMA), celiac trunk, gastroduodenal artery, and inferior mesentery artery did not reveal obvious contrast extravasation. Finally, abdominal and pelvic CT was performed, and the CT scans revealed active bleeding from a periampullary duodenal diverticulum (Fig. 1a-1b). The patient was then transferred to the intensive care unit because of unstable hemodynamics.

A third endoscopy was performed for hemostasis. However, the procedure was unsuccessful because of excessive bleeding despite administering several injections of diluted epinephrine and normal saline around the ampullary region, which obscured the field of vision. TAE was considered for DDB. The SMA angiogram indicated an active contrast extravasation from the inferior pancreaticoduodenal artery of the SMA (Fig. 2a-2b). We used 20% N-butyl-2-cyano-acrylate (NBCA), which was mixed with lipiodol at a 1:4 ratio, to embolize the target vessel until flow stasis. This was followed by adjunctive medical therapy involving the administration of a high dose of esomeprazole and octreotide in addition to blood transfusion. The patient’s condition gradually improved and tarry stool was no longer observed. The patient resumed oral intake soon after. His serum hemoglobin levels returned to normal by the time he was transferred back to the general ward. His condition was stable at discharge, and bleeding did not recur at follow-up.

**DISCUSSION**

Reviewed previous publications, the incidence of duodenal diverticulum ranges from 0.16% to 22% [2]. On the basis of its morphology, duodenal diverticulum can be categorized into an intraluminal or extraluminal type. In approximately 85%-90% of cases, duodenal diverticulum occurs in the second portion of the duodenum, particularly at the periampullary region, and is the most common location for this disorder [3, 4]. The pathogenesis of duodenal diverticula is not clearly understood. The most well-known theory is that the mucosa herniates into regions of mural weakness; in such regions, the vessel, biliary, or pancreatic duct enters the submucosa [5].

Upper gastrointestinal bleeding has several pathologic entities. Peptic ulcer, gastritis, duodenitis, and esophageal varices account for approximately 80% of cases of upper gastrointestinal bleeding [6]. DDB is a rare cause of upper gastrointestinal bleeding because most duodenal diverticula are asymptomatic. The first case of DDB was reported in 1951 [7]. DDB most commonly occurs at the third and fourth portions of the duodenum [8]. Although the etiology of DDB is not clearly understood, two mechanisms are observed. The first mechanism involves mucosal ulceration and blood vessel tear during sac distension. The second mechanism involves irritation by bowel contents [9]. The optimal therapeutic strategy for DDB has remained controversial because of the rarity of this condition. In most studies, patients were treated using endoscopic therapy or

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**Figure 1**

*Figure 1. a. Axial precontrast CT imaging shows a duodenal diverticulum in the second portion of the duodenum near the periampullary region and is approximately 4 cm in diameter (white arrow). b. Axial contrast-enhanced CT imaging shows contrast extravasation within the duodenal diverticulum, indicating active bleeding (white arrow).*
surgery involving diverticulectomy, and TAE was reported only in a few patients.

Advances in endoscopic technology and techniques have enabled clinicians to manage DDB by using endoscopic therapy. Endoscopy is a convenient tool because it includes both diagnostic and therapeutic properties. A retrospective single-center clinical review revealed that injection therapy was the most commonly used endoscopic method for managing DDB, followed by combination therapy, hemoclips, coagulation, and thrombin spray. An analysis of 23 cases indicated that the recurrent bleeding rate after endoscopic therapy was 13.04% [10]. Endoscopic therapy-associated complications for DDB have been rarely reported. A case of duodenal perforation after endoscopic hemostasis for DDB conducted using hemoclips was reported. The sharp tip of the clip and air inflation resulting in thinning of the duodenal mucosal wall may be the cause for the perforation [11]. The main disadvantage of endoscopic therapy is that massive bleeding in DDB obscures the field of vision, as observed in our case.

TAE is an alternative for DDB management in scenarios such as patients with unstable hemodynamics, poor endoscopic route to the bleeding source, or recurrent bleeding after the surgical procedure or endoscopic treatment. In 1970, angiography was first used to diagnose DDB [12]. Since then, angiography is the preferred choice for diagnosing and treating DDB. Detecting a bleeding site by using angiography is limited by the bleeding rate. The minimum bleeding rate must be over 0.5–1 mL/min for the contrast extravasation to be visible in angiography [13]. During TAE, the intervening radiologist must be familiar with the complex vascular anatomy around the duodenum to prevent complications such as ischemia or pancreatitis. Kwon et al reported a rare case of duodenal obstruction caused by peridiverticular fibrosis after TAE for DDB [14]. No absolute contraindications of TAE for gastrointestinal bleeding have been reported because TAE is often used as a lifesaving procedure. However, some related contraindications include renal insufficiency, coagulopathy, and contrast allergy. This information can be acquired from patient history and laboratory data.

Numerous embolic agents, such as coils, balloons, and particulate and liquid agents, can be used for controlling gastrointestinal bleeding, and most of these agents are permanent in nature. Each of these agents has its own advantage and disadvantage. Coils have the advantage that they can be introduced into a target vessel in an extremely precise manner, but permanent occlusion may impede their access to the target vessel. Gelatin sponge (Gelfoam®) has a temporary embolic feature, is inexpensive, and allows access to the target vessel if bleeding recurs. However, the recanalization speed cannot be predicted.
We used NBCA, a permanent liquid embolic agent, to manage DDB in the examined patient. In gastrointestinal bleeding, multiple feeding arterial branches are observed at the bleeding site. NBCA can pass through small vessels to achieve a more complete embolization of bleeders and prevent the backing bleeding. NBCA is usually mixed with lipiodol to first determine the viscosity of the mixture and then the extent of embolization [15]. Some clinical conditions may often be evaluated when using NBCA, such as the target vessel in which the catheter cannot enter because of the vascular anatomy, risk of coil migration, pseudoaneurysm, or vascular malformation. The common complications associated with NBCA for gastrointestinal bleeding are (1) ischemia of the bowel loop, (2) systemic or local reactions such as nausea, fever, or regional pain, and (3) catheter adherence to the vascular wall. Intestinal ischemia occurs after the NBCA embolization probably because of the regurgitation of NBCA, undesirable distal spread of NBCA, or NBCA adherence to the catheter, which later descends into the nontarget vessels during catheter removal [16]. Some skills or techniques may prevent intestinal ischemia. First, a slow injection of NBCA using fluoroscopy is administered until minimal regurgitation of NBCA [17]. Second, an appropriate concentration and volume of the NBCA–lipiodol mixture should be determined according to the experience of the interventional specialist. We used 20% NBCA (mixed with lipiodol in 1:4 ratio) to embolize the target vessel and avoid adherence of the microcatheter to the target vessel at the same time. Evaluating the complications and recurrent bleeding rate in TAE for managing DDB is a difficult task because of the rarity of this situation. Our patient did not exhibit any of the previously mentioned complications and was discharged in a stable condition.

In conclusion, most duodenal diverticula are asymptomatic but may occasionally entail complications such as hemorrhage or perforation. Interventional radiologists can choose the embolic agent for DDB according to the location of the target vessel and their own experience. Endoscopic hemostasis and TAE have both been proven to be generally safe and effective in DDB management. TAE is a suitable treatment alternative if massive bleeding obscures the field of vision or the patient is unsuitable for surgery.

REFERENCE